

function of conductivity is definitely depressed. The *P-R* interval is diminished in auriculo-ventricular nodal rhythm and in functional bundle-branch block (see Fig. 82).

The period of time occupied by the ventricular complex is approximately that of the ventricular systole. That occupied by the *QRS* group of deflections is of much importance. It should not exceed one-tenth second. If it does it indicates a delay in the conduction of the wave of excitation through the ventricular muscle. This occurs in bundle-branch block, arborization block, ventricular extrasystoles, in the ventricular variety of paroxysmal tachycardia, and, it may be, in extreme preponderance of either ventricle.

The *Q-T* or *R-T* duration varies considerably. With a normal cardiac rate, it probably averages between 0.32 to 0.35 second. It is diminished by an increased cardiac rate, and *vice versa*. When the cardiac rate is unusually frequent the duration of the diastolic interval is so shortened that the deflection *T* approaches more and more to the following *P*, and *P* and *T* may even coincide and are superimposed (Fig. 49).

In auricular hypertrophy, such as in mitral stenosis and congenital

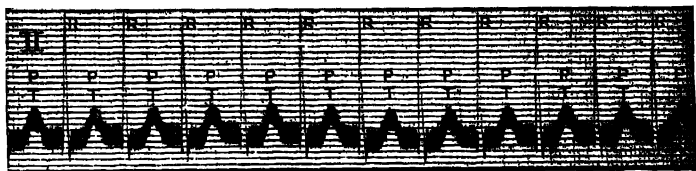


FIG. 49.—Electro-cardiogram in which *P* and *T* coincide and are superimposed.

pulmonary stenosis, especially the former, the *P* deflection, particularly in lead II, is of increased amplitude (Figs. 53 and 54), and not infrequently is also broad and has a flat top, and may be notched or bifurcate (Figs. 55 and 56). Some writers are of opinion that a mere increase in the amplitude of *P* is occasionally met with in normal hearts.

A large *Q* in lead III is often a feature of the *T3* type of infarction, either recent or old. It should be noted, however, that it may also occur with a horizontal or transverse position of the heart due to a high diaphragm, as, for example, in ascites, pregnancy, or obesity. In the differential diagnosis, whether there is also a *Q* of somewhat smaller amplitude in lead II and a characteristic alteration in the *R-T* (*S-T*) interval and in the form of the *T* deflections are of cardinal importance. In addition, a large *Q* due to upper displacement of the diaphragm is diminished or abolished by holding the breath after a deep inspiration. A *Q* in lead I is strongly suggestive of an old infarct of the wall of the left ventricle near the apex and the adjacent part of the interventricular septum.

Notching or splintering or slurring of the *QRS* complex of slight degree may be found in normal conditions, and in preponderance of either ventricle. These in pronounced degree and also notching which is irregular signify that the wave of excitation is spreading in the different parts of the ventricular musculature in an abnormal manner. They may be met with in bundle-

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branch block, arborization block, ventricular extra-systoles, and in the ventricular variety of paroxysmal tachycardia.

Diminution in the amplitude of *T* occurs during forced expiration, and it may be with advancing age. The *T* deflections may be flattened or inverted in the three leads in hypothyroidism (see Fig. 95). Diminution of *T* in lead II is frequently met with in marked left- or right-sided preponderance. Inversion of *T* in lead III alone (Fig. 48) is not infrequently found in health, and therefore its significance in a single examination is uncertain and it is of essential importance to know whether the deflection was formerly upright. Among pathological conditions, it is found in marked right-sided preponderance. Inversion of *T* in lead III and lead II may occur during forced expiration. Apart from this, it is probably pathological.

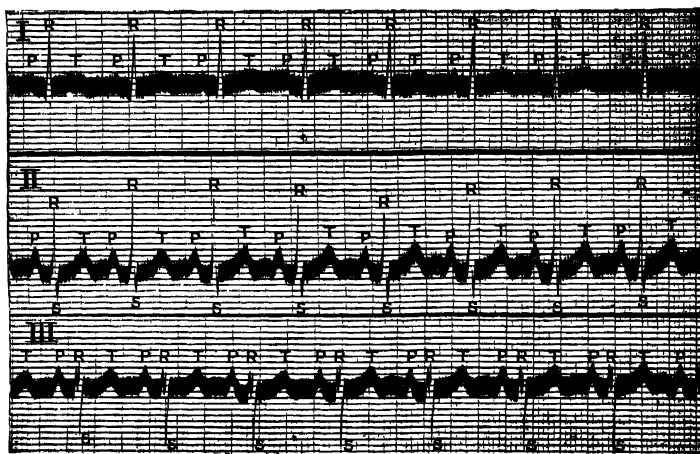


FIG. 50.—Electro-cardiogram showing predominant hypertrophy of the left ventricle.

Inversion of *T*₁ and *T*₂ may be regarded as pathological, suggesting coronary or myocardial disease. Inversion of *T*₁ is also to be met with in gross left-sided preponderance.

The changes in the *T* deflections which occur in bundle-branch block, coronary occlusion, and with full doses of digitalis are described elsewhere.

The characteristics of a normal fourth lead are as follows (Fig. 51). The first deflection, *P*, is upright, and is rather smaller than in the other standard leads but less so in lead IV *F*. The initial group of ventricular deflections is of larger amplitude than in leads I, II, and III, and is diphasic, the first wave, *R*, being positive and the second, *S*, negative, and they are approximately of equal size. The *S-T* interval, as in the other three leads, is usually isoelectric but it may, on the other hand, show a slight inclination to rise above or to fall below the zero line. The *T* deflection is positive, and is of increased amplitude.

The form of electro-cardiographic curves depends upon the point of origin of the impulse formation, the paths along which the wave of excitation travels, and the manner of conduction of the latter; any departure from the normal in respect of one of these will result in an alteration in the form of the electro-cardiogram.

A typical *P* signifies that the impulse arises in the sino-auricular node and that the wave of excitation spreads over the whole of the auricles along the normal paths. When the auricle contracts in response to impulses not generated at the sino-auricular node, almost always the *P* deflection is of abnormal form. The degree of this depends upon the distance of the site of origin from the normal, for example, if near the node *P* may be almost normal. Often the deflection is inverted. The various forms of abnormal *P* deflections which may be found in auricular or auriculo-ventricular nodal extra-systoles, auricular or auriculo-ventricular nodal paroxysmal tachycardia, auriculo-ventricular nodal rhythm, and auricular flutter are described later. A diphasic or inverted *P* in lead III alone may occur in left-sided preponderance and in the absence of any pathological condition.

Similarly, a typical ventricular complex signifies that the contraction of

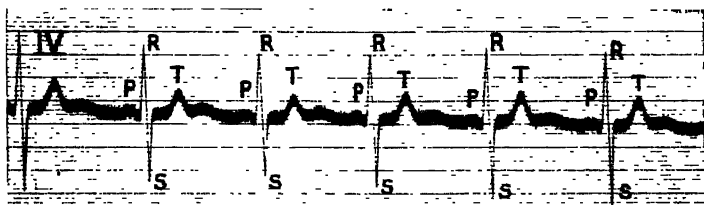


FIG. 51.—A normal electro-cardiogram of lead IV.

the ventricles is supra-ventricular in origin, *i.e.* they have contracted in response to an impulse which arises above the division of the auriculo-ventricular bundle, and that the wave of excitation proceeds along the normal paths. An atypical ventricular complex means that the contraction of the ventricles is ventricular in origin, *i.e.* the stimulus arises below the division of the auriculo-ventricular bundle. When the ventricular contraction is *aberrant* (see p. 847), the complex, while fundamentally typical, differs from the normal to some extent, this varying from a slight degree to even, rarely, that in which the complex resembles one of ventricular type.

CARDIAC HYPERTROPHY.—It has been previously pointed out that in cardiac hypertrophy, while both ventricles are more often affected than one alone, one ventricle is frequently involved to a greater degree than the other. This predominant hypertrophy or preponderance of either ventricle is revealed by the electro-cardiograph. If in cardiac hypertrophy an electro-cardiogram does not indicate either right- or left-sided preponderance, we may assume that the hypertrophy involves both ventricles approximately equally.

In left-sided preponderance, the amplitude of *R* is greater in lead I than in lead III, and the amplitude of *S* in lead III is greater than in lead I (Figs. 47 and 50). The deflections of greatest amplitude in leads I and III, there-

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fore, point away from each other. In right-sided preponderance, the amplitude of *S* is greater in lead I than in lead III, and that of *R* in lead III is greater than in lead I (Fig. 52), and so the deflections of greatest amplitude point towards each other.

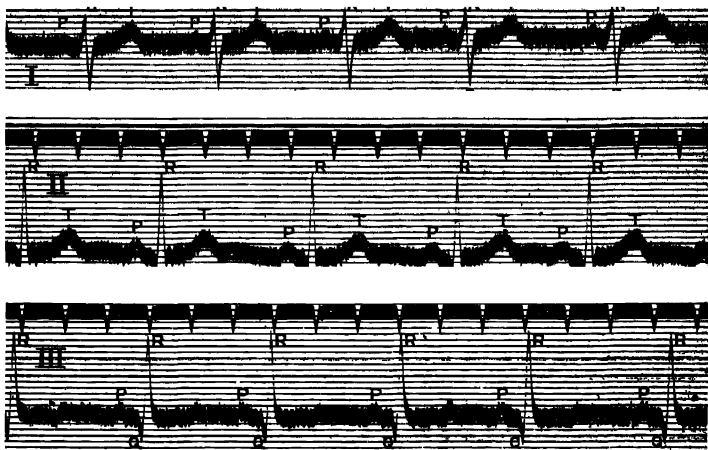
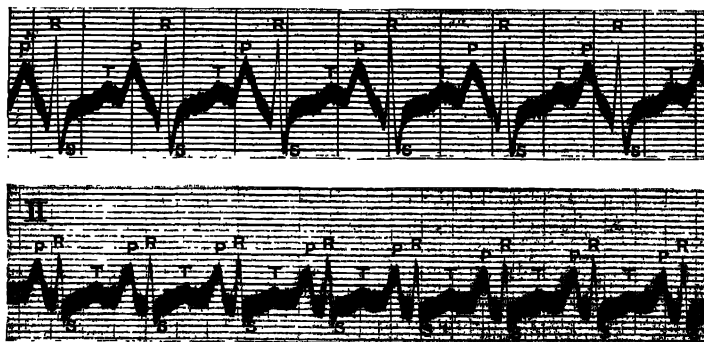


FIG. 52.—Electro-cardiogram showing predominant hypertrophy of the right ventricle.



FIGS. 53 and 54.—Electro-cardiograms from two different subjects showing increased amplitude of the deflection *P*.

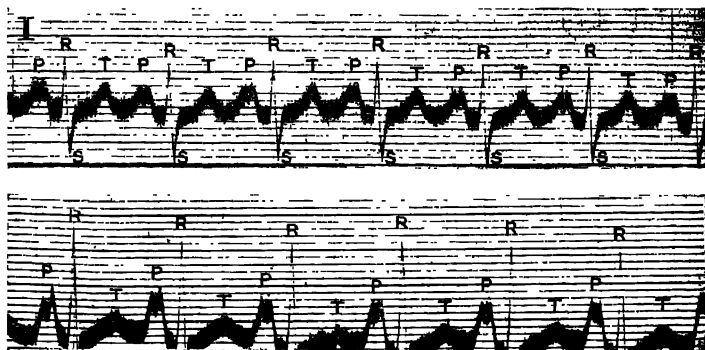
In marked preponderance of the left side, there is often inversion of *T* in lead I; and in that of the right side, inversion of *T* in lead III. Barnes and Whitton have suggested that these changes in the *T* deflection result from mechanical strain on one ventricle rather than actual disease of the myocardium other than hypertrophy. In extreme preponderance of either

ventricle, the period of time occupied by the *Q*, *R*, *S* group of deflections may be increased, exceeding one-tenth of a second.

In the diagnosis of preponderance of either ventricle it is necessary to exclude displacement of the heart, for the following reason. The electrical axis of the organ is influenced by the anatomical axis, so that displacement may give rise to electro-cardiographic curves of right- or left-sided preponderance. Thus, a horizontal or transverse position of the heart due to a high diaphragm tends to produce a curve of left ventricular preponderance; while a vertical position of the organ with a low diaphragm, as seen in asthenic subjects, tends to produce that of right ventricular preponderance.

It is also necessary to distinguish between predominant hypertrophy of the left or right ventricle and a lesion of the left or right main branch of the auriculo-ventricular bundle (new nomenclature) respectively. This is referred to on p. 1020.

CHRONIC VALVULAR DISEASE.—In aortic valvular disease there is usually



FIGS. 55 and 56.—Electro-cardiograms from two different cases of mitral stenosis. The deflection *P* is increased in amplitude, and is also broad, has a flat top, and is bifurcate.

left-sided preponderance. In mitral stenosis there is generally right-sided preponderance; and the *P* deflections may show the changes indicative of auricular hypertrophy described on p. 1003. The latter, when present, are of diagnostic value, especially when there is also right-sided preponderance. When auricular fibrillation supervenes, the curves will present features characteristic of that condition.

CONGENITAL HEART DISEASE.—The amplitude of the deflections is sometimes greater than in the acquired form of valvular disease. There is frequently right-sided preponderance, often of marked degree, especially in pulmonary stenosis.

In dextro-cardia all the deflections of a curve from lead I are inverted, while lead III is equivalent to the normal lead II, and lead II to the normal lead III (Fig. 57). This form of electro-cardiogram is pathognomonic of the condition. It is to be noted that if there is also a lesion giving rise to enlargement of the right ventricle, the electro-cardiogram will be that of left-sided preponderance with inversion of *P* in lead I. In uncomplicated wide patency of the interauricular septum there may be deviation of the electrical axis

to the right. In patency of the interventricular septum the electro-cardiogram is usually normal, except when the defect in the septum involves the

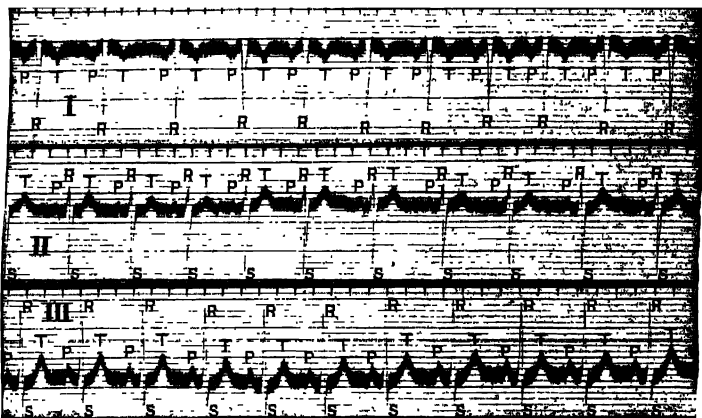
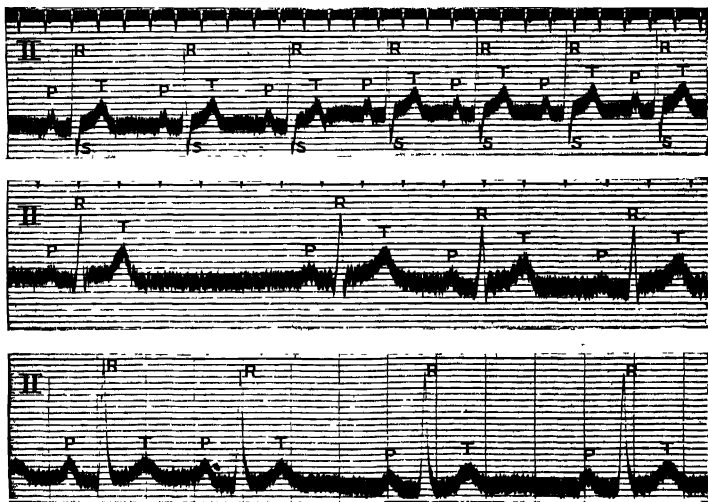


FIG. 57.—Electro-cardiogram from a case of transposition of the heart. All the deflections in lead II are inverted.



FIGS. 58, 59 and 60.—Electro-cardiograms from three different subjects showing sinus irregularity. The auricular and ventricular deflections are of normal form, but there is a variation in the length of the intervals between T and P.

auriculo-ventricular bundle and so gives rise to congenital heart-block. In congenital pulmonary stenosis the electro-cardiogram will reveal a marked right ventricular preponderance.

SINUS ARRHYTHMIA.—The electro-cardiogram of this rhythm may readily be identified (Figs. 58–60). There is merely a variation in the length of the diastolic periods, *i.e.* the intervals between *T* and *P* (see p. 885).

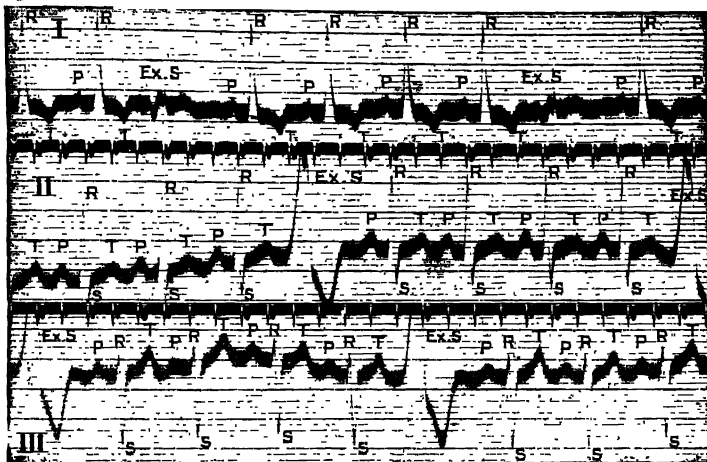


FIG. 61.—Electro-cardiogram showing ventricular extra-systoles, marked *Ex.S.* The corresponding *P* deflections are embedded in the diaphasic variations. There is also inversion of *T* in lead I, and left-sided preponderance.

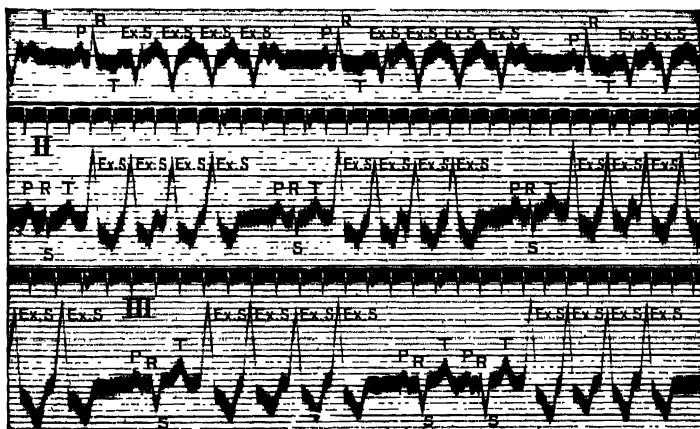


FIG. 62.—Electro-cardiogram showing rapid successions of ventricular extra-systoles. There is also inversion of *T* in lead I and left-sided preponderance.

EXTRA-SYSTOLES.—Extra-systoles may be readily recognised by means of the electro-cardiograph, and usually their site of origin.

In the ventricular variety (Figs. 61 and 62) the ventricular complex occurs

earlier than the anticipated time. As the ventricular contraction is ventricular in origin and therefore the wave of excitation travels along abnormal channels, the ventricular complex is wholly atypical: it is diphasic, and is of increased amplitude. It is of the same duration as that of the rhythmic contraction.

There are two main varieties of ventricular extra-systoles, and it is usually possible to differentiate them by means of the electro-cardiograph. In one, the ventricular complex consists of an upward, tall and pointed deflection, and then of a downward and broader deflection, in lead I; and of a downward, deep and pointed deflection, and then of an upward and broader deflection, in lead III. In the other variety, the ventricular complex consists of a downward, deep and pointed deflection, and then of an upward and broader deflection, in lead I; and of an upward, tall and pointed deflection, and then of a downward and broader deflection, in lead III. It was formerly thought, largely as the result of experiments in animals, that the first variety has its origin in the left ventricle or apical portion of the heart, and the second in the right ventricle or basal portion. Recent evidence has been adduced, based partly on direct observations on the exposed human heart, which strongly suggests that the opposite is the case. The direction of the deflections in lead II is usually the same as in lead III, but the reverse may be the case.

In the ventricular variety of extra-systoles, the auricle maintains its usual rhythm and contracts as the result of the normal stimulus from the sinus. It follows, therefore, that the corresponding *P* deflection appears at the anticipated time and is normal in form. But it is usually embedded in the ventricular complex, although it may be detected in this part of the electro-cardiographic curve in some cases, and occasionally is nearly separate.

If the ventricular extra-systole takes place after the normal auricular contraction, and the wave of contraction from the auricle has reached the ventricle and met that of the premature ventricular contraction in the ventricular wall, the ventricular complex of the premature contraction of the ventricle will present both typical and atypical features.

The site of origin of a ventricular extra-systole may occasionally be at one time in the basal or right portion of the ventricle, and at another in the apical or left portion, in the same subject.

A ventricular extra-systole is usually followed by a compensatory pause, which is complete. Sometimes, when the cardiac rate is slow, an interpolated extra-systole occurs.

In the auricular variety of extra-systole (Fig. 63) the *P* deflection takes place before the anticipated time. As the point of origin of the stimulus for contraction is at a site other than the sino-auricular node, almost always the *P* deflection is of abnormal form. The degree of difference depends upon the distance of the site of origin of the impulse from the sino-auricular node; and if near or at the node, *P* may be practically normal. Often the deflection is inverted (see lead II). The premature contraction of the auricle may take place so early as to coincide with the ventricular contraction of the preceding cycle, in which case *P* and the preceding *T* are superimposed. The *P-R* interval may be increased. The *P* deflection is usually followed by a premature ventricular complex. This is of typical form, since the ventricular contraction is of supra-ventricular origin and therefore the wave of excitation to the ventricle travels along the usual paths,

but it may be aberrant. Almost always aberrant ventricular beats are found only when there is diminished auriculo-ventricular conductivity. In some cases, the stimulus for contraction does not reach the ventricle at all, in which case the premature contraction of the auricle is not followed by a premature contraction of the ventricle—"blocked auricular extra-systole" (third extra-systole in lead I of Fig. 63).

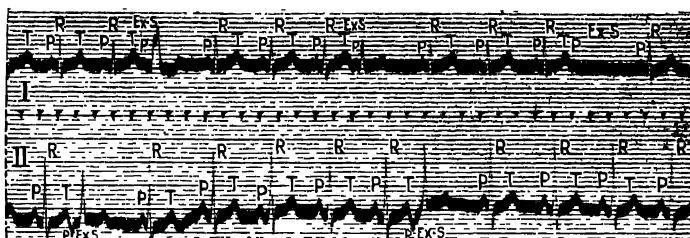


FIG. 63.—Electro-cardiogram showing auricular extra-systoles, marked *Ex.S.* The premature *P* deflections are of normal form in lead I, and of abnormal form—being inverted—in lead II. The premature ventricular complexes are of abnormal form. The third extra-systole in lead I is blocked.

In auricular extra-systole the compensatory pause is rarely complete.

In the auriculo-ventricular nodal variety of extra-systole (Fig. 64) there is prematurity of the *P* deflection and also of the ventricular complex. In cases in which the contraction of the auricle and ventricle is absolutely synchronous, the *P* deflection coincides with, and is embedded in, the *Q, R, S* complex (see lead II). When the contraction of the auricle begins before that of the ventricle, the *P* deflection precedes *R*, and the *P-R* interval is

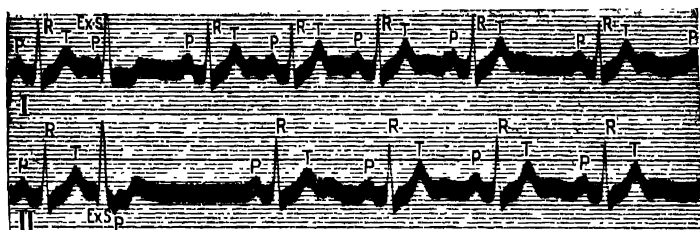


FIG. 64.—Electro-cardiogram showing auriculo-ventricular extra-systoles, marked *Ex.S.* In that of lead I the premature *P* deflection occurs before that of the ventricular complex, while that in lead II is embedded in the ventricular complex.

diminished (see lead I). When the auricular contraction commences after that of the ventricle, the *P* deflection follows the *R* deflection. When the *P* deflection is to be observed, as the auricular contraction is due to an impulse which arises at an abnormal point, it is almost always abnormal in form, often being inverted. As the ventricular contraction is supra-ventricular in origin, the ventricular complex is of typical form but may be aberrant.

In the auriculo-ventricular variety of extra-systole, the compensatory pause may, or may not, be complete.

Occasionally the beat immediately following an extra-systole arises from the same site as that of the premature contraction.

PAROXYSMAL TACHYCARDIA.—The first *P* deflection of the paroxysm is premature, and usually the paroxysm is followed by a long pause.

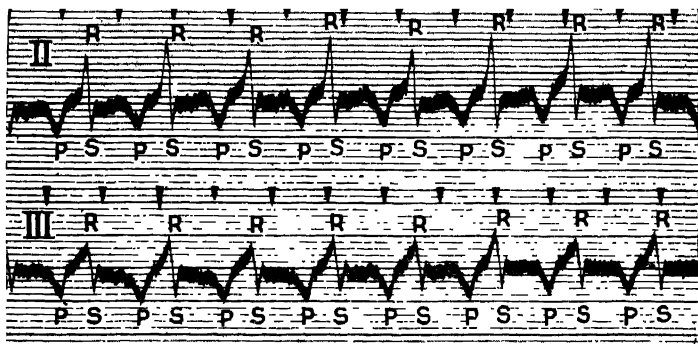


Fig. 65.—Electro-cardiogram showing the auricular variety of paroxysmal tachycardia.

There is no essential difference between the complexes of the individual beats during a paroxysm and those of single extra-systoles in the same individual.

In auricular paroxysmal tachycardia (Fig. 65) there is a rapid succession of *P* deflections occurring at regular intervals. As in the case of auricular extra-systoles, these are almost always of abnormal form, often being inverted. The contraction of the auricle may take place so early as to coincide with the ventricular contraction of the preceding cycle, in which

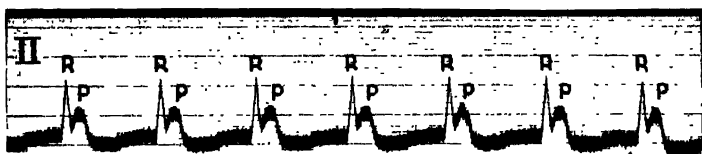


Fig. 66.—Electro-cardiogram showing the auriculo-ventricular nodal variety of paroxysmal tachycardia.

case *P* and the preceding *T* may be superimposed. The *P*–*R* interval may be increased. The *P* deflections are usually followed by ventricular complexes of the same form, or approximately so, as those preceding and following the paroxysm. There may be auriculo-ventricular block or bundle-branch block. In the latter case the ventricular complexes may resemble those of ventricular paroxysmal tachycardia. In this connection, in the differential diagnosis between the ventricular complexes of supra-ventricular and of ventricular origin, it is important to note whether any part of the *P* deflection

can be detected in the ventricular portion of the electro-cardiographic curve or not.

In auricular paroxysmal tachycardia, the rhythm is regular.

In auriculo-ventricular nodal paroxysmal tachycardia (Fig. 66) the electro-cardiogram is often difficult to determine. There is a rapid succession of auricular and also of ventricular complexes. In cases in which the contractions of the auricle and ventricle are absolutely synchronous, the *P* deflections coincide with and are embedded in the *Q*, *R*, *S* complexes. When the contractions of the auricle begin before those of the ventricle, the *P* deflections precede the *Rs*, and the *P-R* interval is diminished. When the auricular contractions commence after those of the ventricle, the *P* deflections follow the *Rs*. When the *P* deflections are to be observed, they are

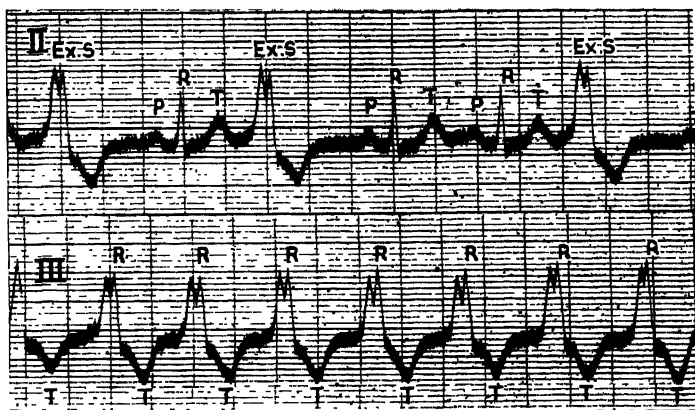


FIG. 67.—Electro-cardiogram of leads II and III. Lead II shows three isolated extra-systoles of ventricular origin. Lead III shows the ventricular variety of paroxysmal tachycardia, the complexes of which are of the same form as those of the isolated extra-systoles in lead II.

usually of atypical form, often being inverted. The ventricular complexes are of typical form but may be aberrant.

In auriculo-ventricular nodal paroxysmal tachycardia the rhythm may be regular or irregular.

In ventricular paroxysmal tachycardia (Figs. 67 and 68) there is a rapid succession of ventricular complexes. Usually each of these is of the same form as those of isolated extra-systoles arising in one ventricle (see p. 1010). In some cases, however, the point of origin of the beats is sometimes in one ventricle and at other times in the other, resulting in complexes of different form; and this may occur in the case of every other beat, so that complexes of different form alternate, which condition is sometimes called bi-directional ventricular tachycardia, and is of very serious significance. Sometimes the ventricular complexes are of indefinite form.

In the ventricular variety of paroxysmal tachycardia, the auricle generally maintains its usual rhythm, contracting as the result of an impulse arising

in the sino-auricular node. It follows, therefore, that the *P* deflections occur at the usual intervals and also are of normal form. They are generally embedded in the ventricular complexes, but in some cases they may be detected in this portion of the electro-cardiographic curve and occasionally they are nearly separate. In some cases, however, the ventricular rhythm gives rise to retrograde auricular beats, either with each ventricular contraction, or, less frequently, *i.e.* retrograde heart-block. Rarely there is auricular fibrillation, and more rarely auricular paroxysmal tachycardia, or auricular flutter.

In ventricular tachycardia, any existing irregularity of rhythm is usually so slight that it is detected only by means of graphic methods.

In the differential diagnosis of paroxysmal tachycardia by the electro-cardiograph, it is to be observed that, as the difference in the form of the complexes of the abnormal and the normal rhythms may be only slight it may be necessary to compare carefully the complexes of the paroxysm with those of the beats which either preceded or followed it—indeed, an analysis of either transition periods may be necessary.

AURICULO-VENTRICULAR NODAL RHYTHM.—In this condition, when the

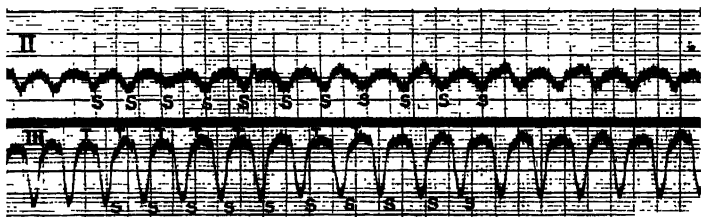


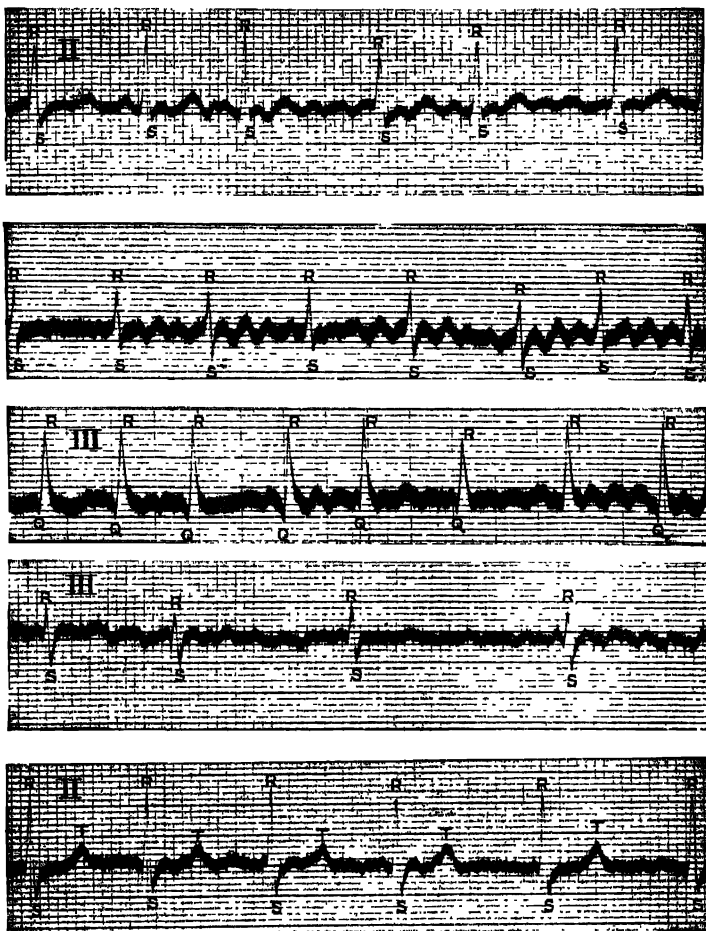
FIG. 68.—Electro-cardiogram of leads II and III showing the ventricular variety of paroxysmal tachycardia.

contractions of the auricle and ventricle are absolutely synchronous, the *P* deflections coincide with and are embedded in the *Q*, *R*, *S* complexes. When the contractions of the auricle begin before those of the ventricle, the *P* deflections precede the *Rs*, and the *P*–*R* interval is diminished. When the auricular contractions commence after those of the ventricle, the *P* deflections follow the *Rs*. When the *P* deflections are to be observed, they are usually of abnormal form, often being inverted. The ventricular complexes are of typical form but may be aberrant.

AURICULAR FIBRILLATION.—In auricular fibrillation the electro-cardiogram is characteristic (Figs. 69–73).

There is an absence of *P* deflections. There are, on the other hand, oscillations caused by the fibrillating auricle, called fibrillary waves, during ventricular diastole, at a rate ranging from 300 to 600, and usually about 450 times, per minute. They occur at irregular intervals, and their form and size vary, the latter being sometimes very minute and sometimes considerable. They are most evident in cases of slow cardiac action. They may coincide with the *T* deflections, in which case the outline of the latter is altered. Apart from the very rare cases in which complete auriculo-ventricular block is present, the ventricular rhythm is completely irregular, *i.e.* the *R* deflec-

tions occur at irregularly irregular intervals; their amplitude varies from cycle to cycle; and often there is no relationship between the length of a pause and the amplitude of the *R* deflection which follows it. When the



FIGS. 69-73 — Electro-cardiograms from cases of auricular fibrillation.

ventricular rate is slow or very rapid, this may be difficult to determine. As the ventricular beats are supra-ventricular in origin, the complexes are of typical form but they may be aberrant. There may be superadded ventricular extra-systoles, usually during the administration of one of the digitalis group of drugs (see p. 868).

In the differential diagnosis between the electro-cardiograms of auricular fibrillation and those of auricular flutter, in the former almost always the ventricular rhythm is completely irregular, while in the latter, it will be found that such is not the case, even when the rhythm is very irregular. Further, the deflections due to auricular systole in auricular flutter may be distinguished from the fibrillary waves in auricular fibrillation in that they are less frequent, rhythmic, of larger amplitude, and almost invariably of constant form and size.

AURICULAR FLUTTER.—In this condition it is of particular importance to analyse the three leads (Figs. 74–76). The *P* deflections may range from 180 to 360, the usual rate being about 300, per minute. In all the leads the deflections occur at regular intervals, and as soon as one terminates the next

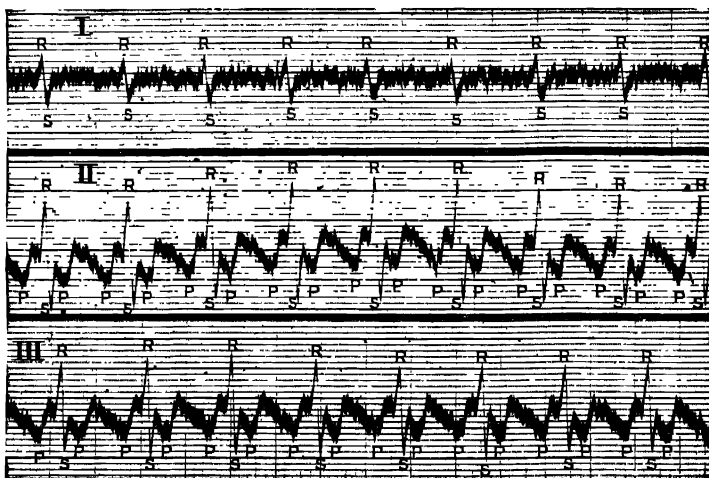


FIG. 74.— Electro-cardiogram from a case of auricular flutter, with 2 : 1 heart-block. The rate of the auricle is between 320 and 330 per minute.

one commences. In each lead of any given case they are almost invariably of constant form. In lead I the amplitude is comparatively small. As the impulse arises at an abnormal point, the deflections are atypical in form. The ascending limb is rather sharp, and the descending more gradual, and the summit may be dome-shaped. It is difficult to say at what part the deflection begins. As the ventricular complexes are supra-ventricular in origin, they are of typical form. They are superimposed upon the *P* deflections, modifying their outline. In some cases *T* may be detected. Excepting in the very rare cases of 1 : 1 rhythm, in which event there is an equal number of *P* deflections and ventricular complexes, there are two or more *P* deflections to each ventricular complex, according to the degree of auriculo-ventricular block (see p. 900). When the response of the ventricle to auricular contraction is at irregular intervals, the ventricular rhythm may be even very irregular.

It is necessary to differentiate the electro-cardiograms of auricular flutter from those of auricular fibrillation. This is dealt with on p. 1016.

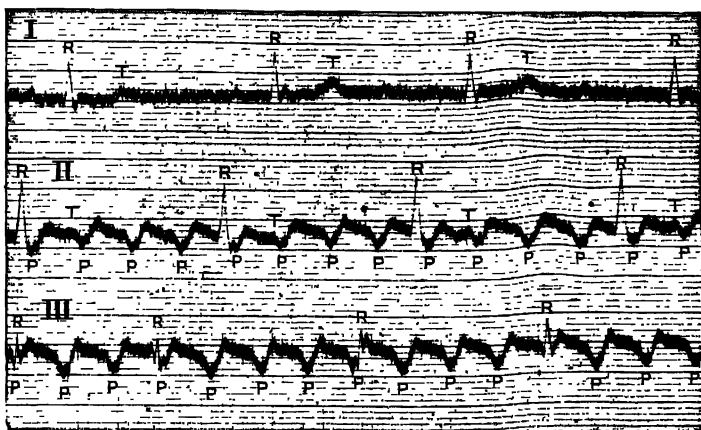


FIG. 75.—Electro-cardiogram from a case of auricular flutter, with 4 : 1 heart-block.

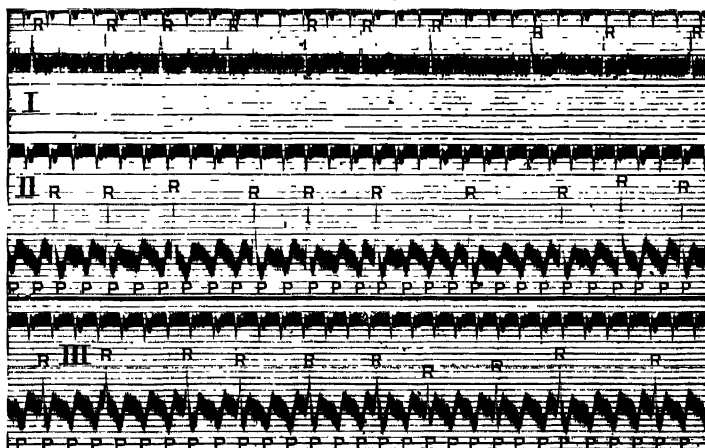


FIG. 76.—Electro-cardiogram from a case of auricular flutter. The auriculo-ventricular ratio is sometimes 2 : 1, at others 3 : 1, and at others again 4 : 1. The response of the ventricle to auricular contraction being at irregular intervals, there is irregularity of the ventricular rhythm.

VENTRICULAR FIBRILLATION.—The ventricular complexes are replaced by oscillations, occurring at very rapid and irregular intervals, and their form and size vary.

SINO-AURICULAR BLOCK.—In this condition there is an absence of both the auricular and ventricular complexes during an abnormally long pause. (See p. 904 and Fig. 77.)

AURICULO-VENTRICULAR BLOCK.—In the first grade (leads I and III of Fig. 78) there is merely an increase of the *P-R* interval, it exceeding 0.18

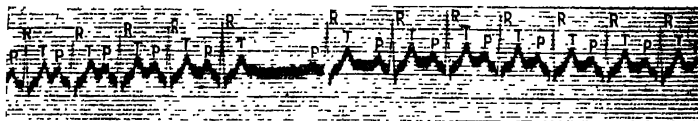


FIG. 77.—Electro-cardiogram showing an abnormally long pause due to sino-auricular block.

second. It may be increased to such a degree that *P* coincides with the preceding *T*.

In the second grade (lead II of Fig. 78) the *P* deflections are found at regular intervals and are of typical form. Sometimes the *P* deflections are not followed by ventricular complexes, the frequency of such depending

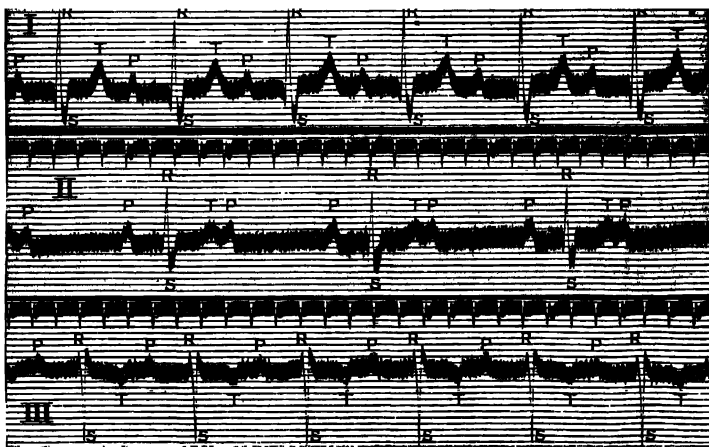


FIG. 78.—Electro-cardiogram showing partial heart-block. In each lead there is an increase of the *P-R* interval, and in lead II there is also continuous 2:1 rhythm, every other stimulus from the auricle failing to reach the ventricle. There is also inversion of *T* in lead II† and left-sided preponderance.

upon the degree of block, as described on p. 905. Unlike complete heart-block, on each occasion the ventricular complex is preceded by a *P* deflection. In the case of dropped beats, almost always there is a progressive increase of the *As-Vs* interval preceding and a progressive shortening of the interval following each dropped beat, so that the prolonged pause during a dropped beat is not equal to two regular pulse-beats. As the ventricular beats

are of supra-ventricular origin, the ventricular complexes are of typical form.

In complete heart-block (Fig. 79) the *P* deflections are found at regular intervals, and are of typical form. They are more frequent than the ventricular complexes. In addition, the time-relation between the *P* deflec-

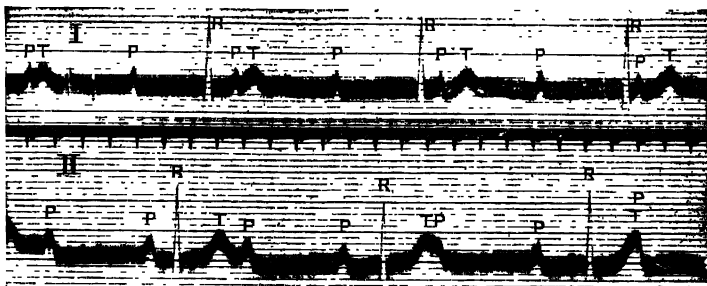


FIG. 79.—Electro-cardiogram of leads I and II showing complete heart-block, or dis-association of the auriculo-ventricular rhythm, the auricles and ventricles beating independently of each other.

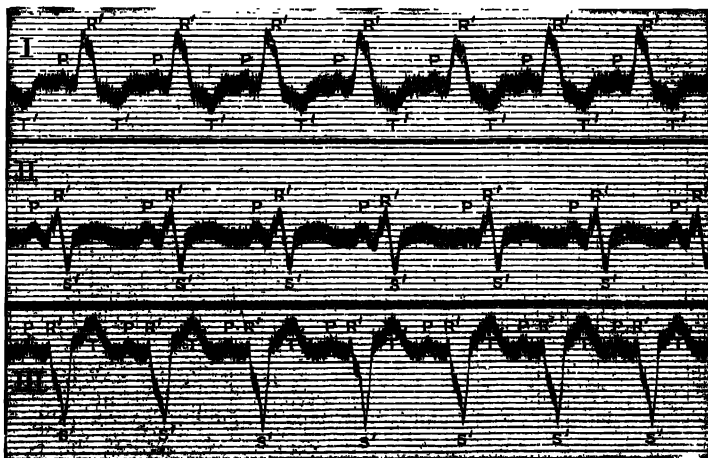


FIG. 80.— Electro-cardiogram showing a lesion of the left main branch of the auriculo-ventricular bundle (new nomenclature).

tions and the ventricular complexes is a constantly varying one, the former at one time preceding, at another following, and sometimes, again, coinciding with the latter. In the last case the *P* deflection is superimposed upon the ventricular complex. As the ventricular beats are of supra-ventricular origin, the ventricular complexes are of typical form. Sometimes there are superadded ventricular extra-systoles.

BUNDLE-BRANCH BLOCK.—A lesion of either of the two branches of the auriculo-ventricular bundle may be recognised by means of the electro-cardiograph (Figs. 80 and 81).

The ventricular complex is diphasic and of increased amplitude. The initial group of deflections (*Q*, *R*, *S*) is of increased duration, exceeding one-tenth second and comprising more than one-third of the whole complex, and usually exhibits pronounced or irregular notching. The terminal deflection (*T'*) points in the opposite direction to the initial group of deflections in leads I and III. In lead II, *Q*, *R*, *S* is usually of less amplitude and is often diphasic; *T'* may point in either direction. In the diphasic ventricular complexes of leads I and III there is usually no iso-electric period between *Q*, *R*, *S* and *T*. The *P*-*R* interval may be increased.

Two types of curves may be recognised: (1) The common type, in which

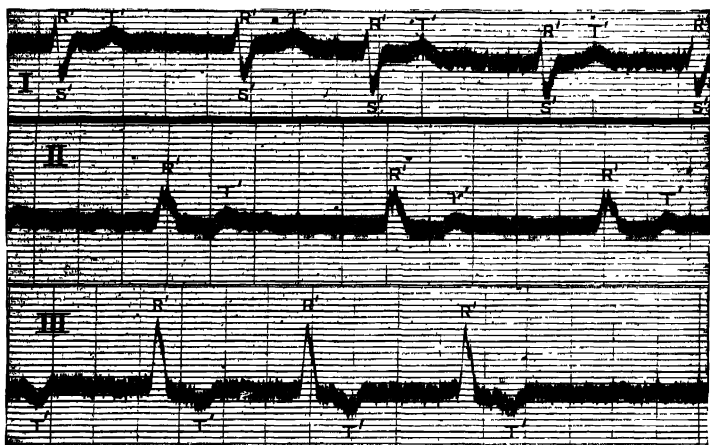


FIG. 81.—Electro-cardiogram showing a lesion of right main branch of the auriculo-ventricular bundle (new nomenclature). There is also auricular fibrillation.

there is a large *R'* in lead I and a large *S'* in lead III, *T'* pointing downwards in lead I and upwards in lead III. (2) The rare type, in which there is a large *S'* in lead I, and a large *R'* in lead III, *T'* pointing upwards in lead I and downwards in lead III. It was formerly supposed that the first type was indicative of right bundle-branch block, and that the second signified left bundle-branch block. It is now believed that the opposite is the case.

It is necessary to distinguish between a lesion of the left or right main branch (new nomenclature) and preponderance of the left or right ventricle respectively. The distinguishing features are that in bundle-branch block the initial group of deflections is of increased duration and usually exhibits pronounced or irregular notching, and *T* points in the opposite direction to the main initial deflection in leads I and III. As already noted, increased duration of the *Q*, *R*, *S* complex may be met with in extreme preponderance of either ventricle. But taking cases of preponderance as a whole, the

period of time is materially less. In addition, pronounced or irregular notching does not occur, and *T* is not of increased amplitude and, apart from exceptional cases, points in the same direction.

FUNCTIONAL BUNDLE-BRANCH BLOCK.—A condition, which is rare, in which there is increased duration of the *Q*, *R*, *S* group of deflections together

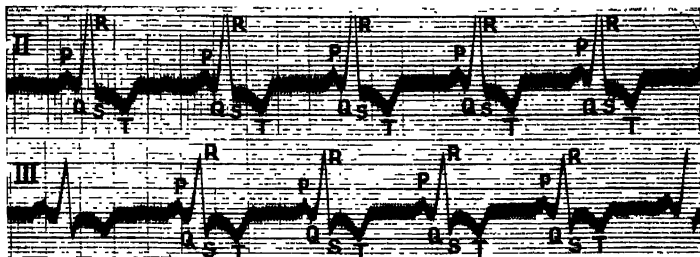


Fig. 82.—Electro-cardiogram showing functional bundle-branch block.

with a diminished *P*-*R* interval (Fig. 82) has been termed functional bundle-branch block.

ARBORIZATION OR INTRA-VENTRICULAR BLOCK.—The initial group of deflections is of increased duration and usually exhibits pronounced or irregular notching, and is of low voltage (Fig. 83). Some believe that occasionally the *T* deflection points in the opposite direction to the initial

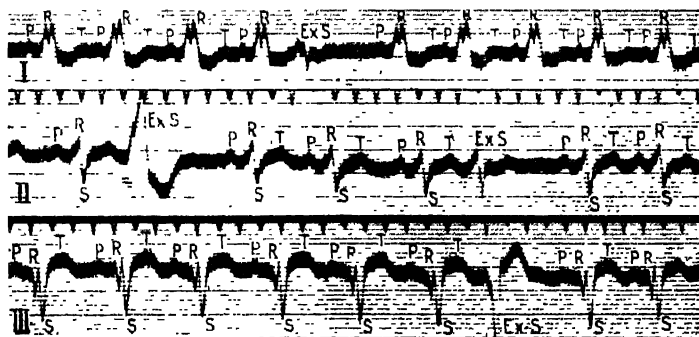


Fig. 83.—Electro-cardiogram showing arborization block. There is an extra-systole, marked Ex.S., in lead I, two extra-systoles in lead II, and one in lead III.

deflection in leads I and III. Even if such should be the case, its form is not so abnormal.

It should be pointed out that some writers are of opinion that the electro-cardiogram just described indicates a delay of the wave of excitation along the right or left main division of the auriculo-ventricular bundle, *i.e.* incomplete or partial bundle-branch block.

ALTERNATION OF THE HEART.—This may sometimes be recognised by means of the electro-cardiograph, by an alternation in the amplitude of the deflections due to the contraction of the ventricle (Fig. 84). Both the *R* and *T* waves may be affected, or one more than the other. It should be noted that alternation of the heart is sometimes shown in a sphygmogram without any corresponding evidence in an electro-cardiogram, while rarely the opposite holds good. It should be further noted that the alternation in a sphygmogram and electro-cardiogram does not always correspond; i.e. the smaller ventricular deflections correspond with the larger pulse-wave.

ANGINA PECTORIS.—Left-sided preponderance is usual. In a proportion of cases the ventricular complexes are abnormal. Most varieties of these

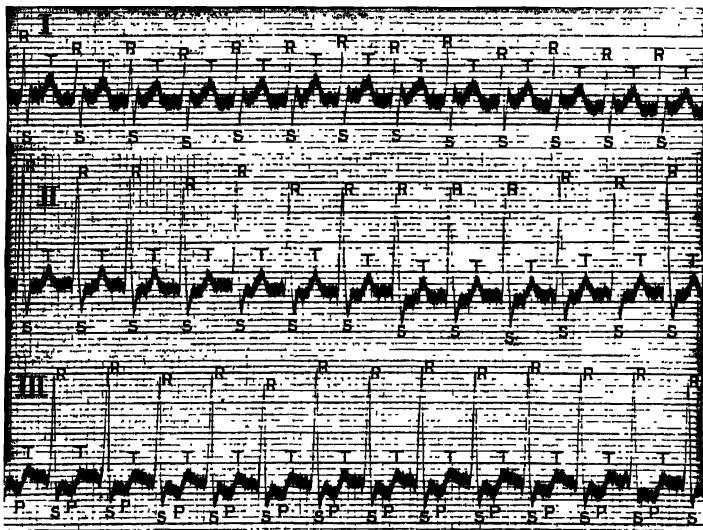


FIG. 84.—Electro-cardiogram from a case of paroxysmal tachycardia. There is alternation in the amplitude of the *R* deflections.

abnormalities may be met with, including flattening or inversion of *T* in lead I or II, or both; increased duration, and notching of the *Q*, *R*, *S* group of deflections; bundle-branch block; and a large *Q* deflection in lead III (see p. 1025). There may be extra-systoles and some degree of heart-block. Other abnormalities of rhythm are exceptional. The changes in the ventricular complexes may be of considerable value in doubtful cases. Negative findings, on the other hand, are of no importance.

Transient modifications of the electro-cardiograms similar to those of coronary occlusion with infarction have been observed in some cases of angina pectoris during the attacks. Such are of much diagnostic significance.

CORONARY OCCLUSION WITH INFARCTION OF THE HEART.—Electro-cardiograms of this disease are usually characteristic and of great diagnostic value. They are as follows:

Within a few hours there is usually a deviation of the *R-T* or the *S-T* segment. This portion of the curve commences from the *R* or *S* deflection either above or below the iso-electric level and proceeds in a more or less horizontal direction, resulting either in a plateau-shaped elevation or in a depression respectively. This alteration is generally most noticeable in

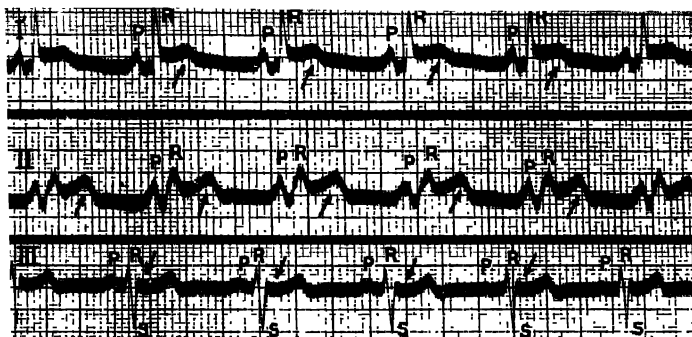


FIG. 85.—Electro-cardiogram from a case of the T^1 type of infarction of the heart taken early after the onset of symptoms. The *R-T* portion of the curve commences above the zero level in leads I and II, and slightly below the zero level in lead III, as indicated by arrows.

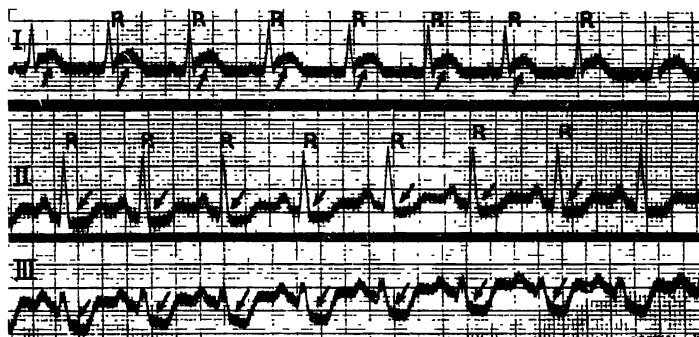


FIG. 86.—Electro-cardiogram from a case of the T^1 type of infarction of the heart taken early after the onset of symptoms. The *R-T* portion of the curve commences above the zero level in lead I, and below the zero level in lead III and rather so in lead II, as indicated by arrows.

leads I and III. In this event, the corresponding portions of the curves in these leads are divergent. Thus, if there is *R-T* elevation in lead I there is *R-T* depression in lead III (Figs. 85 and 86); and *vice versa* (Figs. 87 and 88). Sometimes the deviation of the *R-T* segment is best observed in leads III and II, or in leads I and II; or the alteration may be present in one lead only. The foregoing features are perhaps pathognomonic of the condition.

After a few days or more, the *R-T* portion of the curve gradually returns to the iso-electric level, and the *T* deflections gradually reappear. The latter assume a direction opposite to that to which the *R-T* segments were

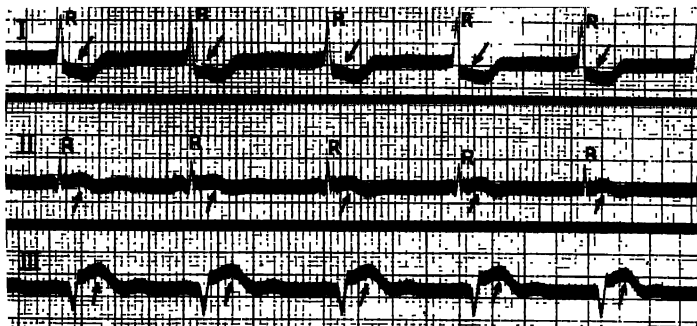


FIG. 87.—Electro-cardiogram from a case of the T^3 type of infarction of the heart taken early after the onset of symptoms. The *R-T* portion of the curve commences above the zero level in lead III and slightly so in lead II, and below the zero level in lead I, as indicated by arrows.

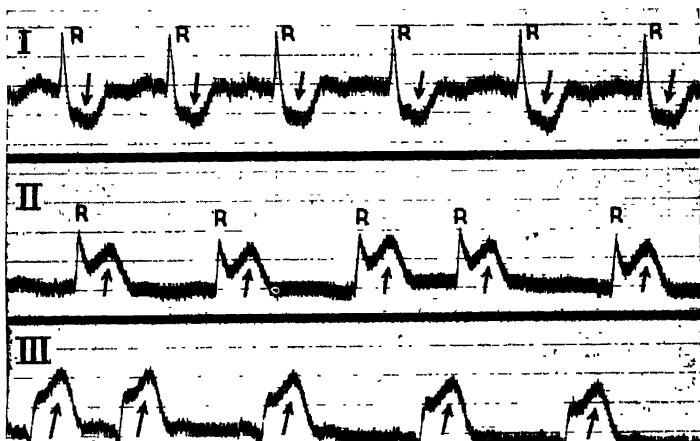


FIG. 88.—Electro-cardiogram from a case of the T^3 type of infarction of the heart taken a few hours after the onset of symptoms. The *R-T* portion of the curve commences below the zero level in lead I and above the zero level in leads II and III, as indicated by arrows.

previously deviated. Thus, inversion of the *T* deflections follows elevation of the *R-T* segments, and upright *T* waves follow depression of the *R-T* segments. (Figs. 89-91.) The *T* deflections are usually sharply defined, and their amplitude is often large. The *R-T* intervals preceding the

altered *T* waves frequently exhibit convexity or concavity. The foregoing features are not so characteristic as are those of the first stage, but, taken together with the clinical features, they afford strong corroborative evidence of the disease.

In both stages, sometimes there is diminished amplitude and increased duration of the initial group of ventricular deflections (*Q*, *R*, *S*), and the latter is generally associated with notching or splintering.

There are various types of curves, the two commonest being: (1) Lead I shows *R-T* elevation and, later, inversion of the *T* deflections; and in lead III, the *R-T* segments are depressed, with the subsequent development of upright *T* waves. (Figs. 85, 86 and 89). (2) Lead III shows *R-T* elevations and, later, inversion of the *T* deflections; and in lead I, the *R-T* segments are depressed, with the subsequent development of upright *T* waves (Figs. 87, 88, 90 and 91).

The first type, which is the more common, is associated with occlusion

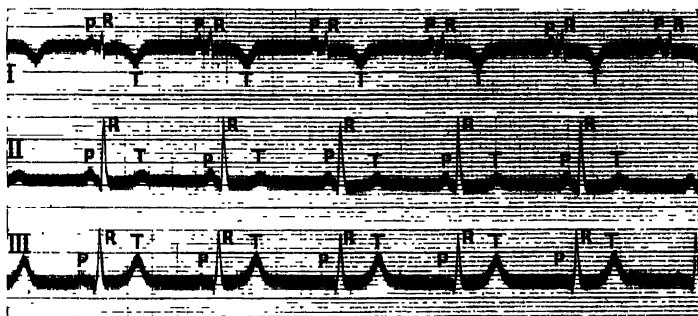


FIG. 89. — Electro-cardiogram from a case of the T^1 type of infarction of the heart. The *T* deflections in lead I are inverted, and are rather sharply defined and of increased amplitude. Those in lead III are upright, and are sharply defined and of increased amplitude.

of the descending branch of the left coronary artery and infarction of the wall of the left ventricle near the apex, especially the anterior part, and the adjacent part of the interventricular septum; and is termed the T^1 type. The second is associated with occlusion either of the right coronary artery or the circumflex branch of the left and infarction of the posterior wall of the left ventricle near the base; and is designated the T^2 type.

In both types of curves the *T* deflections in lead II are often slightly inverted or flattened.

Some changes in the *T* deflections towards the normal usually supervene within a few weeks or months, and ultimately the *T* deflections in all leads may become normal, but sometimes those in one lead remain inverted and sharply defined, and even these changes are very suspicious.

A large *Q* deflection in lead III (see Figs. 90 and 91) is often a noticeable feature of infarction of the posterior wall of the left ventricle, either recent or old. It is to be noted, however, that it may also occur in other conditions (see p. 1003).

Lead IV is particularly valuable in occlusion of the descending branch of the left coronary artery, *i.e.* the T^1 type. This lead alone may be affected, while in some cases the changes are more evident, or occur earlier and last longer than in the three other leads. They resemble those of lead I of this type. Thus, there is elevation of the $R-T$ intervals, and subsequently in-

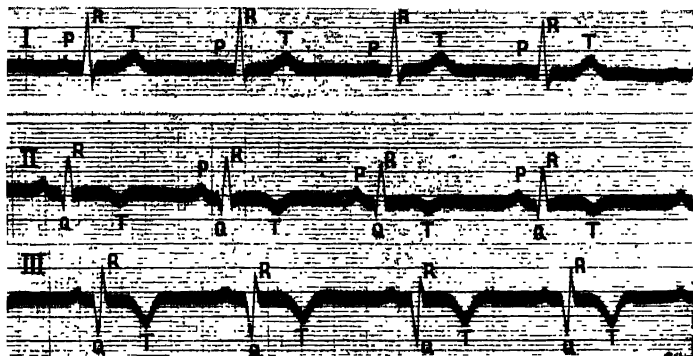


FIG. 90.—Electro-cardiogram from a case of the T^3 type of infarction of the heart. The T deflections in lead II are inverted. Those in lead III are inverted, and are sharply defined and of increased amplitude. There is a large Q in lead III.

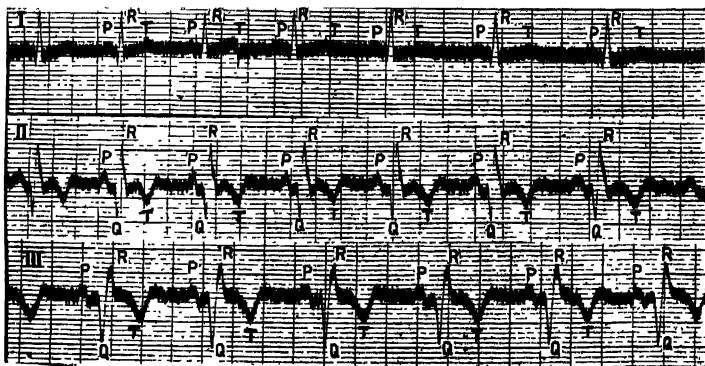


FIG. 91.—Electro-cardiogram from a case of the T^3 type of infarction of the heart. The T deflections in leads II and III are inverted, and are sharply defined and of increased amplitude. There is a large Q in leads III and II.

verted T waves. (Figs. 92 and 93.) Lead IV is of less value in infarction of the posterior wall of the left ventricle, *i.e.* the T^3 type. There may be no changes, or if present they are less marked. If there are changes, they generally resemble lead I of this type. Thus, there is depression of the $R-T$ intervals and, later, upright T deflections. The latter are of increased amplitude, and may be huge. (Fig. 94.)

The foregoing changes in the electro-cardiograms of coronary occlusion with infarction of the heart are not constant. But they are frequent, and when they do occur are of great diagnostic value. It is necessary to point out that it is the successive changes in the curves which are especially

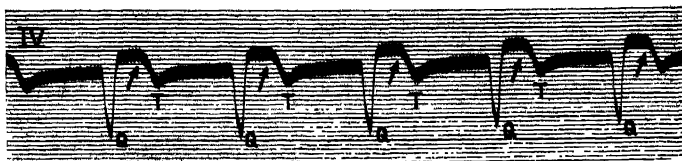


FIG. 92.—Electro-cardiogram of the fourth lead from a case of the T^1 type of infarction of the heart. There is R - T elevation, as indicated by arrows, and inversion of T . There is also a large Q .

important. For this reason, serial records taken over a period of time are of much greater value than a single one, for in cases in which the latter does not reveal the characteristic changes, later curves may do so. It is unusual to find an absence of these changes during the whole of the first two weeks

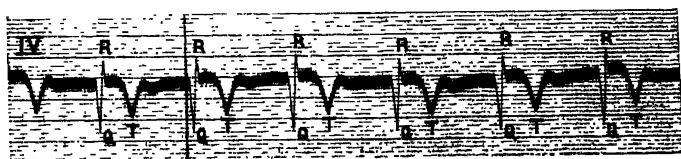


FIG. 93.—Electro-cardiogram of the fourth lead from a case of the T^1 type of infarction of the heart. There is slight R - T elevation, and the T deflections are inverted, sharply defined and of increased amplitude. There is also a large Q .

after the onset of symptoms, though transient changes may have disappeared in later electro-cardiograms.

In conclusion, it is to be noted that a similar deviation in the R - T (or S - T) interval has also been recorded in rheumatic carditis, pericardial

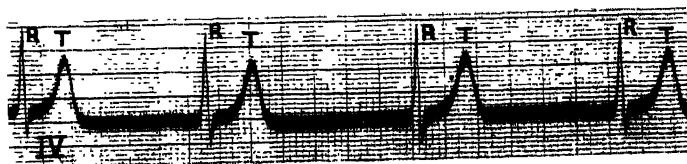


FIG. 94.—Electro-cardiogram of the fourth lead from a case of the T^3 type of infarction of the heart. The T deflection is positive, sharply defined and huge.

effusion, uræmia, and pneumonia. The clinical features of these conditions, however, do not resemble those of coronary occlusion.

It has been pointed out that transient changes in the electro-cardiograms similar to those of coronary occlusion have been observed during attacks of angina pectoris.

Low Voltage.—The term low voltage is used when no part of the *Q*, *R*, *S* complex exceeds five millimetres in the three leads. It is almost always of

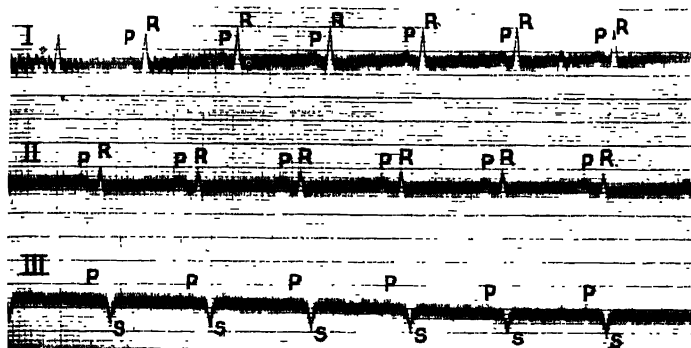


FIG. 95.—Electro-cardiogram from a case of hypothyroidism. The *T* deflections are absent in the three leads; and there is also diminished amplitude of the *Q*, *R*, *S* complex.

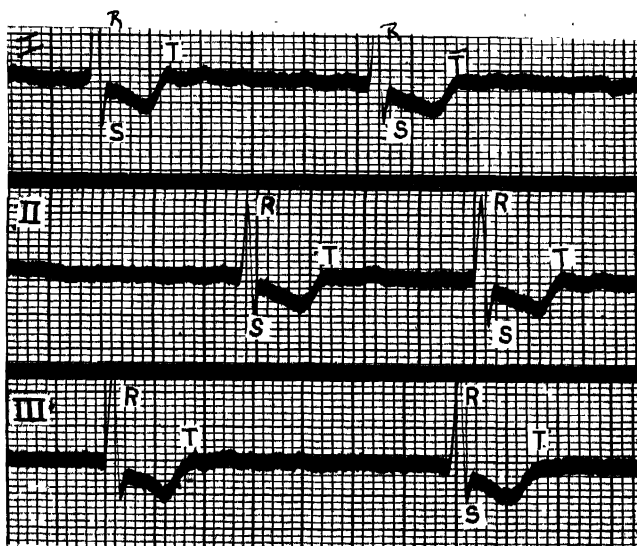


FIG. 96. — Electro-cardiogram from a case of auricular fibrillation fully under the influence of digitalis.

pathological significance, and may be met with in marked chronic myocardial disease, severe congestive failure from any cause, coronary occlusion, pericardial effusion, and hypothyroidism. In the last named there are also

changes in the other deflections (see below). As stated on p. 1021, low voltage is one of the various features found in arborization or intraventricular block.

HYPOTHYROIDISM.—The *T* deflections are flattened or inverted in the three leads; and frequently there is also diminished amplitude of the *Q*, *R*, *S* complex. See Fig. 95.

THE EFFECT OF DIGITALIS.—Electro-cardiograms of patients fully under the influence of digitalis may show depression of the *R-T* interval with flattening or inversion of the *T* deflections (see Fig. 96). This should be distinguished from the *R-T* deviation due to coronary occlusion. In the former the corresponding portions of the curves always point in the same direction; while in the latter usually they point away from each other in leads I and III.

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DISEASES OF THE BLOOD VESSELS

The term arterio sclerosis, which means no more than arterial hardening, has led to much confusion as it has been employed by various authors in different senses to describe different pathological conditions of the arteries. Thus, it has been used to comprise the following conditions:

(a) Diffuse hyperplastic sclerosis, characterised by intimal hyperplasia and hypertrophy of the media;

(b) Degeneration of the middle coat of the artery, known as Mönckeberg's medial sclerosis, in which lime salts are deposited more or less symmetrically in rings around the artery; and

(c) Arterial degeneration of the intima, known as atheroma, or, because of the tendency to calcification, as atherosclerosis.

Or it is confined to the first and second. Or it is restricted to the first. Or it is employed to include the three mentioned above and, in addition, arterial inflammation, which when chronic is most often the result of syphilis but may be caused by other infections. Or, finally, it is employed by a few writers to include even other pathological conditions, such as infiltration. It is, therefore, clear that the term arteriosclerosis should not be used except in a very general sense.

It is proposed to adopt the following classification of diseases of the arteries:

- (i) Arterial inflammation
 - (a) Acute arteritis
 - (b) Chronic arteritis
 - (c) Thrombo-angiitis obliterans
 - (d) Polyarteritis nodosa or periarteritis nodosa
- (ii) Diffuse hyperplastic sclerosis
- (iii) Mönckeberg's medial sclerosis
- (iv) Fatty Degeneration of the Media
- (v) Atheroma (Atherosclerosis)
- (vi) Other Degenerations allied to atheroma
- (vii) Arterial infiltration.

1030 DISEASES OF THE CIRCULATORY SYSTEM

And the following will be dealt with separately : (1) The syndrome of intermittent claudication, caused by several of the foregoing pathological conditions ; (2) Aneurysms, whether (a) saccular, most frequently caused by syphilis ; (b) aneurysmal dilatation, most often due to atherosclerosis ; or (c) the less common forms of aneurysm.

ARTERIAL INFLAMMATION

The arteries may be infected from their intima, either by micro-organisms settling on the surface or by the arrest of an infective embolus within the lumen. They may also be infected by micro-organisms reaching the media or adventitia through the vasa vasorum or by direct inward spread of inflammation from the surrounding tissues.

ACUTE ARTERITIS

Acute arteritis was formerly described as a common event in many diseases, the staining of the intima being mistaken for inflammation. Acute inflammation of the arteries is, however, a rare disease, and is usually met with as a complication in the acute infections. The intima of the aorta may be infected in cases of septicæmia and pyæmia, and most commonly in cases of progressive septic endocarditis, when the organisms usually found are streptococci. Vegetations may be seen upon the intima and the inflammation rapidly involves the subjacent coats. Occasionally the aorta may be infected in a septicæmia or pyæmia, through embolism of the vasa vasorum, or the ascending aorta may be infected by spread through the vessel wall from a pericarditis. The wall of the aorta may rupture, or an aneurysm be formed, but this is rare. Acute multiple arteritis is most frequently seen as a sequel of typhoid fever, but cases have been observed after small-pox, scarlet fever, influenza, and pneumonia. In many cases the organisms of the disease have been found in the vessel wall.

Symptoms.—The symptoms depend upon the vessels affected. In the case of the femoral artery, there may be severe pain in the course of the vessel with sometimes redness and swelling in the part affected. The pulse below is obliterated. The limb becomes pale and cold, and then livid. Gangrene may or may not follow ; it depends upon the rapidity with which the vessel is blocked. In some cases where the onset is severe and the symptoms suggest that gangrene will follow, the circulation improves and colour returns to the limb. In other cases, several of the arteries may be infected at the same time, with high fever and symptoms of an acute infection. Some cases of cerebral thrombosis in which a young or middle-aged patient makes a good recovery and lives for twenty or thirty years without any other vascular catastrophes are probably of this type.

Prognosis and Treatment.—In acute arteritis treatment, other than rest and general and local measures for the relief of pain, is of little avail. The condition is a very severe one. If the artery involved is a large one, every effort should be made to avoid infectious gangrene, and in some cases a surprising return of circulation may be observed.

CHRONIC ARTERITIS

Ætiology.—Acquired syphilis is by far the most common cause of chronic arteritis, and less frequently congenital syphilis. Tuberculous endarteritis is not uncommon in the small pulmonary arteries and in the arteries of the brain in tuberculous meningitis. Endarteritis obliterans may also be caused by infection with pyogenic organisms of a subacute or chronic type. Moreover, changes in the adventitia of the small arteries are also found in polio-encephalo-myelitis and in encephalitis lethargica.

Pathology.—Chronic arteritis is a focal affection, and is found in muscular and elastic arteries of all calibres. It is common in the aorta and large elastic arteries, and it also frequently attacks the small arteries. The large muscular arteries, however, are but rarely affected by syphilis. Chronic inflammation of the arteries has been divided into—(1) Endarteritis, where the intima is affected; (2) mesarteritis; and (3) periarteritis, where the external coat is involved. In the great majority of cases of inflammation of the smaller arteries all coats are involved. The muscular and elastic fibres tend to be destroyed and this may result in direct rupture. The changes in the intima are very conspicuous. Its layers become very much thickened by inflammatory infiltration and proliferation. In the early stages round cells are seen, and later spindle-shaped fibroblasts, definite granulation being thus formed, while in cases of syphilis plasma cells and eosinophil leucocytes are often present. The result of this thickening in such small arteries is to narrow the vessel, and the condition is often termed endarteritis obliterans. The lumen may finally become completely blocked, leading in the brain to cerebral softening, and in other tissues to fibrosis. The adventitia is also greatly thickened in chronic syphilitic arteritis and consists of inflamed tissue infiltrated by lymphocytes, plasma cells, and occasional eosinophil leucocytes.

Syphilis of the aorta or *syphilitic mesaortitis* is a focal inflammation, but it may implicate almost the whole length of the aorta. The inflammation extends from the adventitia. The vasa vasorum proliferate and in most cases pass into the intima. About these vessels is a zone of granulation tissue, usually consisting only of plasma cells, lymphocytes, eosinophil leucocytes, and fibroblasts, but occasionally there are gummata with giant cells. Endarteritis of the vasa vasorum is found only in the more intense reactions. The elastic fibres of the media are completely destroyed in the areas of granulation tissue; occasionally the media is necrosed between areas of granulation tissue. The *Sp. pallida* has been demonstrated in the lesions. The intima of the aorta is usually thickened over the areas of inflammation, and this thickening has been in the past confused with the degeneration of the intima that we know as atheroma. The inflammatory thickening of the intima due to syphilis can, in its earlier stages, be distinguished by the naked eye from atheromatous thickenings by its sharper demarcation, pearly colour, rubber-like consistency, crenated outline, pitted surface, and freedom from fatty degeneration. The weakening of the vessel wall on account of the replacement of the middle coat frequently results in dilatations, varying in size from minute stellate patches to large aneurysms. The scarring and pitting are due to fibrous tissue replacing the inflamed media and are characteristic. In the later stages atheroma usually occurs

in the thickened intima over the areas of inflammation; also syphilitic mesaortitis and primary atheroma may co-exist, especially in later life.

The *coronary* circulation is often found affected in these cases, with the result that there are attacks of angina pectoris. Owing to the mesaortitis of the ascending aorta, the orifices of the coronary arteries become narrowed or blocked, but the disease does not spread far down the arteries, in this way providing a striking contrast with atheroma. It may cause extensive necrosis or fibrous patches in the myocardium, and the heart is not uncommonly enlarged. Frequently the aortic ring has expanded, or the aortic valves have become involved by the syphilitic inflammation, so that aortic regurgitation is a common sequel.

The *arteries of the brain* are frequently involved by syphilis, which causes endarteritis obliterans and consequent cerebral softening. This change is also present in syphilitic meningitis, where the syphilitic inflammatory reaction is more intense. The kidneys are but rarely affected, though occasionally gummata are found. The eyes are not infrequently attacked and a condition of syphilitic choroido-retinitis may be seen on ophthalmoscopic examination. In other organs, such as the liver and testicle, gummatous necrosis, followed by fibrous changes, is found. In rarer instances the trachea and lungs are affected.

Symptoms.—The symptoms that result from syphilitic vascular disease depend upon the organ affected.

In the aorta, aneurysm is a common result from syphilis. Aortic regurgitation, with its usual effect on the heart, is even more common. The symptoms of necrosis or fibrosis of the myocardium are severe anginal pains, often resulting in sudden death. Occasionally the heart muscle may give way, with the usual signs of chronic cardiac failure. If the brain is affected by arterial thrombosis, the symptoms depend upon the area affected; hemiplegia, aphasia, and hemianopia may all result. Albuminuria is quite uncommon in syphilitic vascular disease. Iritis may be present, and more commonly choroiditis, in which white patches surrounded by pigmented areas give a striking appearance to the fundus oculi. The optic disc is often white and atrophied. In syphilitic meningitis, oedema of the optic papilla is often present. The radial artery is practically always normal to the touch in cases of syphilitic vascular disease.

Course.—The course of syphilitic arterial disease is very variable. The symptoms resulting from endarteritis and subsequent thrombosis in medium-sized arteries such as the cerebral, are generally present in the acute secondary stage, from 2 to 5 years from the date of infection, but the vascular disease may exist with exacerbations all through life, and the important and characteristic cardiovascular changes in the first part of the aorta generally follow 15 to 25 years after the original infection.

Prognosis.—The prognosis in cerebral syphilitic vascular disease is on the whole good, if the condition be taken early enough and treated properly. It has to be remembered, however, that vascular syphilis is often complicated by parenchymatous syphilis, where the spirochaetes are not only found in the walls of the vessels, but also in the cerebro-spinal tissues themselves, and this complication certainly increases the gravity of the condition. In the later cardiovascular syphilis the prognosis is bad unless the diagnosis is made early before any serious cardiac symptoms have developed.

Treatment.—Prophylaxis is of the greatest importance in syphilitic vascular disease. With regard to treatment, in the acute stages of syphilitic vascular disease it is generally thought that mercury is the best drug to use. Mercurial inunctions or soluble intramuscular mercurial injections should be given for 2 or 3 weeks before any intravenous injections are prescribed. Moreover, iodides should be given by the mouth, to absorb as far as possible the inflammatory products in the intima of the vessels. The indiscriminate use of arsphenamine and neoarsphenamine in the acute stages of syphilitic vascular disease has been followed by most disastrous results, their administration causing further swelling of the intima and further blocking and thrombosis of the small vessels. After mercury and iodides have been given for 3 or 4 weeks, neoarsphenamine, should be administered cautiously in small doses and gradually increased.

THROMBO-ANGITIS OBLITERANS

This name was suggested by Buerger in 1908 for a disease characterised by acute inflammation of the deep arteries and veins, and sometimes by a migratory inflammation of the superficial veins in the extremities. Thrombosis develops and the vessels become occluded.

Ætiology.—The malady is found most commonly in middle-aged male Hebrews. The cause is unknown, but the pathological changes suggest that it is due to an infection. Syphilis is usually not present, the Wassermann reaction being negative. Excessive tobacco smoking has been suggested as a predisposing factor.

Pathology.—The deep vessels of the arms and legs, especially the latter, are occluded by thrombosis in various stages of organisation; their walls are traversed by vessels, and show a little inflammatory infiltration. In about a quarter of the cases this is associated with a migratory phlebitis in the superficial veins of the limbs. The condition is characterised by extensive progressive thrombosis, with organisation, with little inflammation of the vascular coats.

Symptoms.—The onset is gradual with pains in the feet and toes. The patient is unable to walk for more than a few minutes without severe cramp-like pain in the legs (intermittent claudication, see page 1041). The thrombi in the superficial veins, when they occur, are also very tender. Redness of the extremity, especially when in a dependent position, is often noted, while blanching occurs when the limb is raised. Diminution or loss of pulsation in the arteries, such as the radial or dorsalis pedis, is often present. In the later stages, the cramp-like pain becomes intense, and the disability in walking often leads to marked mental depression. Trophic changes appear in the skin, with gangrene, and fissures and ulcers may occur.

Diagnosis.—(1) Raynaud's disease more often attacks females; the upper extremities are most affected; and X-rays show marked atrophy of the bones of the hands. This is not present in Buerger's disease. (2) In erythromelalgia the limbs become red and flushed, but the arteries pulsate forcibly; gangrene does not occur. (3) In gangrene due to Mönckeberg's sclerosis, the calcified arteries may be well seen by means of the X-rays.

Prognosis.—The course of the disease varies. Some cases progress

rapidly, while others last for years. Gangrene may require high amputation of the limb.

Treatment.—As the cause is not known, there is no specific treatment. With regard to drugs, iodides and glyceryl trinitrate are used. Gentle massage is useful, and diathermy sometimes helps. Ultra-violet therapy has been tried. Buerger recommends *passive postural exercises*, and employs the following method: "The affected limb is elevated, with the patient lying in bed, to from 60 to 90 degrees above the horizontal, being allowed to rest upon a support for from thirty seconds to three minutes, the period of time being the minimum amount of time necessary to produce blanching or ischæmia. As soon as blanching is established, the patient allows the foot to hang down over the edge of the bed for from two to five minutes, until reactionary hyperæmia or rubor sets in, the total period of time being about one minute longer than that necessary to establish a good, red colour. The limb is then placed in the horizontal position for about three to five minutes, during which time an electric heating pad or a hot-water bag is applied, care being taken to prevent the occurrence of a burn. The placing of the limb in these three successive positions constitutes a cycle, the duration of which is usually from six to ten minutes. These cycles are repeated over a period of about one hour, some 6 to 7 cycles constituting a séance" (Leo Buerger, *The Circulatory Disturbances of the Extremities*). Others claim that the maintenance of a high venous pressure and stimulation of the heart are the most effective lines of treatment. Pain is relieved by rest in the recumbent position, and heat applied to the painful limb by electric pads or electric light baths is also useful. Ulcers of the legs should be treated surgically.

POLYARTERITIS NODOSA OR PERIARTERITIS NODOSA

Polyarteritis nodosa is a rare complaint characterised by prolonged fever and the occurrence of nodular swellings, and in some cases aneurysms of the medium-sized arteries. It may affect arteries in almost any part of the body, producing a very diverse symptomatology.

Ætiology.—Young adults are most commonly affected, and males more often than females. The cause is unknown. The Wassermann reaction is negative, but the pathological changes and course of the disease suggest an infectious agent.

Pathology.—The medium-sized arteries are usually affected, especially those of the heart, kidneys, and intestines. A remarkably focal acute inflammation extends through all the coats of the artery with hyaline or fibrinoid necrosis of the arterial wall, and within the lumen are often found thrombi, which may become organised. Aneurysmal dilatation is usually present. There may be many polymorphonuclear leucocytes and some mononuclear cells, lymphocytes, and plasma cells; eosinophil cells are a characteristic but inconstant finding. White or yellowish-white nodules, from the size of a pin's head to that of a pea, can be seen on the arteries. Owing to the alteration in the lumen of the vessels, necrosis and infarcts occur in the organs supplied.

Symptoms.—The disease may start in almost any way, even with bronchial catarrh or with epigastric pain. There is tachycardia and irregular fever, with marked prostration. Some cases may start as an acute illness, simulating nephritis or rheumatic carditis; in others long continued malaise is the main

presenting symptom. Acute abdominal pain may be caused by disease of the mesenteric arteries—indeed, even perforation of the intestine and peritonitis have followed. If the arteries of the heart are involved, evidence of myocardial disease will be present; and when the kidneys are affected, blood and casts appear in the urine. Occasionally bronchial asthma, cough and hæmoptysis have been noted. Later, in a small proportion of cases, nodular swellings, varying in size up to a pea, may be felt in the subcutaneous tissues of the abdomen, thorax, and limbs. Examination of blood shows the anæmia and a moderate leucocytosis. Blood cultures are sterile.

Diagnosis.—This is extremely difficult, owing to the variable symptoms displayed. In most cases, a pyrexial infection, of unknown origin, has its nature revealed only post-mortem. Rarely, however, where a node was felt in the subcutaneous tissues and excised during life, the diagnosis has been made before death. The diagnosis should now be made more often if it is suspected in any obscure and varied illness in which there is some evidence of arterial involvement.

Prognosis.—It has been said that in most cases death occurs within a few weeks to a few months after the onset of symptoms. Now, however, when more cases are recognised, it is found that many recover and that the disease may run an intermittent course for many years.

Treatment.—This is the same as that of any acute infection. Arsenic, mercury, and quinine should all be tried, though the results up to now have not been encouraging. In the main the treatment should be symptomatic as the parts involved are so varied.

DIFFUSE HYPERPLASTIC SCLEROSIS

The diffuse hyperplastic sclerosis of Jores and Evans, formerly known as arterio-capillary fibrosis of Gull and Sutton, is characterized by intimal hyperplasia, especially of the smaller arteries and arterioles, and hypertrophy of the media, particularly of the medium-sized arteries.

Ætiology.—Diffuse hyperplastic sclerosis is common in late middle age, and is by no means a scule change. In the great majority the causes are those of hyperpiesia (see p. 1065). The affection may also be due to gout and lead poisoning, and chronic nephritis. In rare cases associated with chronic interstitial nephritis, it may occur in young children. It is more common in males than in females. The inherited constitution is of great importance as shown by the high familial incidence in certain cases. Syphilis has no part in the causation of this condition, though, of course, it may be present in syphilitic cases.

Pathology.—The condition is widespread, frequently involving the whole arterial system. It affects the smaller arteries and the arterioles, and the medium-sized arteries. The kidneys and spleen are most commonly affected, especially the first; the next most frequently, the brain; while the pancreas, liver, suprarenal glands, stomach and intestines are less often implicated. The characteristic lesion consists of a cellular proliferation of the intimal cells and increase of hyaline material. At a later stage there is fatty degeneration in the arterioles alone. The thickening of the intima may lead to obliteration of the lumen of the vessels. There is hypertrophy of the media of the medium-sized arteries, *e.g.* the radials, with little or no

change in the intima. In the case of the smaller arteries and arterioles, a patchy ischæmic fibrosis of certain organs, *i.e.* the kidneys and brain, may take place. Hæmorrhages may also occur.

There may be co-existent atheroma, *e.g.* of the cerebral and coronary arteries, and, it may be, of the aorta, due to age or, in the opinion of some, the result of the hypertension.

Symptoms.—In diffuse hyperplastic sclerosis the vessel wall may be felt to be uniformly thickened—the so-called “whip-cord” artery. The degree of hardening of the arteries is found on palpation to vary at different times. The artery feels hardest when the vessel is most contracted and consequently smallest. As the muscular arteries are those chiefly affected, the radial, brachial, and temporal arteries are involved. The systolic blood-pressure in cases of simple hyperpiesia may be 160–240 mm. or more; it may reach even 300 mm. in cases associated with chronic interstitial nephritis. Symptoms of cardiac hypertrophy are common in the early stages, but in favourable cases they may not appear for years. In some cases cardiac hypertrophy is followed by dilatation, with its attendant symptoms. Headache, of a throbbing and bursting character, and generally in the occipital region, is an early symptom of arterial hypertrophy, and giddiness and fullness in the head are frequently complained of. Transient paralysis may occasionally be met with, and this has been attributed to spasm of the hypertrophied arteries. Later on, cerebral hæmorrhage may occur, with the production of hemiplegia. Albuminuria and casts in the urine may be found, while profuse renal hæmorrhage may occur. Uræmic symptoms do not occur frequently and then not till late unless the condition is secondary to chronic nephritis. Gastro-intestinal symptoms are often present. The patient may first complain of dyspepsia. A chronic diarrhoea without obvious cause in an elderly man should lead to a careful examination of the arteries and kidneys. Bronchitis and emphysema may mask a cardiovascular hypertrophy, and the enlargement of the heart may be overlooked unless there is a careful examination of the blood-pressure. The changes in the fundi are numerous and characteristic, and have been discussed in the section on hypertension (see pp. 1066, 1067). Intermittent claudication is not uncommon in these cases.

Prognosis.—The prognosis depends very largely on the degree of renal involvement. In hyperpiesia the condition may last for many years and only be terminated by hæmorrhage into the brain, the ischæmic fibrosis of the kidney, which accompanies it, being of no clinical importance. If, however, there is evidence that the cardiovascular hypertrophy and high blood-pressure are complicated by true nephritis, the condition is a grave one and uræmia may ensue; and when well-marked albuminuric retinitis is present death usually occurs within six months, though very rare cases have been recorded where a certain amount of ocular change has persisted for years.

Treatment.—The first indication is to remove the cause of the condition as far as possible. As a rule the most important thing is the regulation of the patient's life, the removal of anxiety and the reduction of his work and activity to a reasonable amount. The reduction of alcohol and tobacco is often needed, and it is the average daily consumption that counts in this direction. Any gouty tendencies should be corrected by the administration of alkalis and intestinal antiseptics, such as sodium benzoate. All sources of focal sepsis, whether in tonsils, teeth, nasal sinuses, or genito-urinary tract,

should be carefully sought for and, if found, removed. Moderation in food and drink, with regular exercise and care in promoting diaphoresis, is essential. Turkish and vapour baths may be given cautiously. The administration of a purgative pill once a week, and the use of a mild saline purgative each morning are indicated ; while in acute crises the abstraction of one pint of blood from the arm has often saved life.

MÖNCKEBERG'S MEDIAL SCLEROSIS

This form of degeneration is accompanied by a deposition of lime salts in the middle coat.

Ætiology and Pathology.—The cause is undoubtedly a senile degeneration of the elastic tissue and the muscle of the large muscular arteries, and a deposition of masses of lime salts in the dying tissue. It has no relation to syphilis. This degeneration has been caused experimentally in animals by a great variety of toxins. In man the causation is obscure, but it is common in diabetes and in old people. The lime salt is deposited, more or less symmetrically, within the media in plaques, which encircle part or all of the lumen. The affection is very common in the arteries of the leg below the bifurcation of the femoral ; occasionally the radial and ulnar arteries are affected ; rarely the aorta.

Symptoms.—The symptoms are coldness and œdema of the legs, as the result of defective circulation through them, and finally, and not uncommonly, gangrene results, this form of degeneration being usually present in senile and diabetic cases. The arteries feel like pipe stems, and sometimes crackle when rolled beneath the finger. They can be well seen by means of the X-rays.

Prognosis.—This depends partly upon the amount of gangrene present and partly upon the associated conditions.

Treatment.—It is clear that if amputation has to be undertaken a local amputation is of little value, and the limb should be amputated above the knee, as the arterial degeneration almost always extends to the bifurcation of the femoral artery.

FATTY DEGENERATION OF THE MEDIA

Fatty degeneration of the media occurs commonly in all arteries. *It tends to occur in cases of high blood pressure, and is consequently a common and important secondary complication of diffuse hyperplastic sclerosis.* It is probably the result of toxins or lack of nourishment, and is frequently present in cases of severe anæmia. It also occurs in old age, and is associated with cardiovascular hypertrophy. The muscle fibres become lost, being replaced by fibrous tissue. The vessel wall usually becomes weakened and is liable to rupture.

ATHEROMA

Synonyms.—Atherosclerosis ; Degeneration of the Arterial Intima.

Definition.—Atheroma is a variety of arterial degeneration which affects

and is almost confined to the intima. It is characterised by the accumulation of debris, which is at first fatty and later becomes impregnated with lime salts. The Greek word was used by Galen to signify a swelling full of gruel-like material.

Ætiology.—There is no doubt that atheroma is found more frequently and is more widespread with advancing age. Long life is a question of the blood vessels, and it has been well said that a man is only as old as his arteries. The quality of the arterial tissue that has been inherited may be poor, and a tendency to the development of atheroma at about the same age is often seen in all the members of certain families, thus showing the influence of heredity in the production of the condition. Atheroma also results from the amount of wear and tear to which the vessels have been subjected. It appears as a secondary change in cardiovascular hypertrophy, so that it tends to be more frequent in patients with hypertension than in other subjects of the same age. The affection is much more common in men than in women, and in subjects who have hard and prolonged mental or physical work. Moreover, it is much more common in the aorta than in the pulmonary artery, and when it does occur here it is nearly always associated with high pulmonary pressure, *e.g.* in mitral stenosis and in pulmonary fibrosis. The severity of the disease increases with the length of time during which the high blood-pressure has existed. Over-eating and stress and strain of modern life are probably factors in the ætiology of the condition. It has been said that the most important cause of atheroma is chronic poisoning. Acute degeneration of the media has been found after typhoid fever in young people, and has been caused experimentally by the injection of bacterial toxins. Chronic lead poisoning and gout are also ætiological factors. On the other hand, syphilis has no connection with atheroma, though the condition of the aorta known as syphilitic mesaortitis was for a long time confused with the chronic intimal degeneration we now know as atheroma. Disease of the kidneys probably has no direct relation with atheroma, though the high blood-pressure of chronic interstitial renal fibrosis and of secondary contracted kidney is an important factor in producing atheroma in the large elastic arteries.

Pathology.—Atheroma occurs in the large elastic and muscular arteries. The condition is usually most marked in the aorta. The coronary, cerebral, retinal, radial, brachial, and temporal arteries are frequently affected. The peripheral arteries may be normal to the feel in cases where there is advanced atheroma of the coronary or cerebral arteries. In the slighter degrees, minute yellow flecks or patches on the aorta may be observed by the naked eye. In the later stages, yellow plaques or buttons are conspicuous, and under the microscope masses of large fatty crystals, with a covering layer of fibrous tissue, are to be noted. Atheromatous plaques may ulcerate and the contents be discharged into the aorta, and thrombi are often deposited on the surface of these atheromatous ulcers. As the atheroma may be associated with degeneration of the media, a general dilatation of the aorta is very common. On the other hand, circumscribed aneurysm due to severe medial degeneration very rarely occurs. The aortic valves are frequently affected by atheromatous degeneration, and aortic stenosis or aortic regurgitation may result. A yellow atheromatous patch is commonly seen on the anterior flap of the mitral valve. Atheroma often causes great narrowing of the

lumen of the vessels, and eventually a thrombosis may form and complete occlusion result; this is the most dangerous result of atheroma; it frequently occurs in the large divisions of the coronary arteries, especially in the anterior interventricular branch of the left, and is not uncommon in the vessels of the brain. With regard to the heart, atheroma has a most profound influence, owing to the fact that it is one of the commonest causes of fibrosis of the myocardium; but the chief danger is a sudden blockage of one of the coronary arteries, generally the anterior ventricular branch of the left coronary. In these cases, if death does not follow immediately, a sudden softening of the heart muscle (*myomalacia cordis*) may occur, and an aneurysm of the heart may result, and in certain rare cases actual rupture of the heart wall has followed—a broken heart. In the brain, atheroma results in cerebral thrombosis, and is the commonest cause of this condition in old people, and in middle-aged people who have not had syphilis. Very commonly, however, especially in those cases of atheroma where the blood-pressure is raised, hæmorrhage may occur. In most cases of atheroma the kidneys are not involved, though occasionally atheromatous plaques may be found on the branches of the renal artery. Should, however, one of these plaques be large enough to cause much narrowing of an interlobar artery, a wedge-shaped red area of fibrosis in the distribution of the artery will occur. The renal changes, however, are relatively unimportant, and they rarely lead to symptoms during life.

Symptoms.—The blood pressure is only raised if the atheroma happens to complicate cardiovascular hypertrophy. In the aorta a diffuse dilatation, with pulsation in the supra-manubrial notch, may be present. The radial, brachial, and temporal arteries are often irregularly thickened and tortuous and can often be seen pulsating beneath the skin, but this dilatation has none of the important effects that so often follow a saccular aneurysm. Atheroma of the coronary arteries frequently gives rise to cardiac failure, and more often to angina pectoris; and sudden death is not uncommon, owing to a sudden thrombosis of a large branch. In the brain, hemiplegia usually results from hæmorrhage and more rarely from thrombosis. Ocular symptoms are rare in atheroma of the retinal arteries, which may be seen with the ophthalmoscope to be irregularly swollen and tortuous, but swelling of the optic disc and retinitis are not present.

Prognosis.—The course and prognosis are extremely uncertain. Circulation through the diseased vessels may proceed fairly satisfactorily for a long time, but thrombosis may occur with alarming suddenness, and with the direst results, if a cerebral or cardiac artery is affected.

Treatment.—The treatment of atheroma is unsatisfactory. Prophylaxis exists in the removal of the cause when possible. Great attention should be paid to diet, and repletion should be studiously avoided. Alcohol and tobacco should be taken with greatest moderation, and attention should be paid to regular exercise, and the action of the skin should be assisted by warm baths. It is doubtful if drugs are of value in the treatment of atheroma; but small doses of potassium iodide seem to be of some use in absorbing the degenerative products and assisting in the circulation of the blood through the obstructed areas.

OTHER DEGENERATIONS ALLIED TO ATHEROMA

There are closely allied intimal degenerations, in which this accumulation of fatty debris is absent.

In *fibrotic degeneration*, muscle fibres and to a less extent elastic fibres disappear after little or no fatty degeneration, and the intima becomes fibrotic. This may be found in any artery, but is very common in the smaller arteries, for instance, the interlobular and afferent arteries of the kidneys, where atheroma is very rare.

Hyaline degeneration affects the ultimate arterioles in the kidney and other organs; the muscle and elastic fibres disappear rapidly, and the intima becomes swollen and hyaline, and usually fatty. It is common with high blood pressure, but is otherwise rare.

Both these forms of intimal degeneration are of importance, because intimal fibrosis is much less focal than atheroma, and both forms, in affecting arteries of small calibre, lead in the same way as atheroma to a narrowing of the lumen sufficient to cause ischæmic destruction of the tissues. They may therefore be considered together with atheroma in their clinical effects.

ARTERIAL INFILTRATION

The commonest form of infiltration is amyloid infiltration.

Ætiology.—Amyloid or lardaceous disease occurs in cases of long supuration due to pyogenic organisms, and is frequently associated with the secondary pyogenic infections that occur in tuberculosis of the bones and joints, in chronic syphilitic ulceration, and in actinomycosis.

Pathology.—The amyloid substance is extracellular and is deposited beneath the endothelium of capillaries, the reticulum of adenoid tissue and the pulp of the spleen, and in the smaller arteries and veins, especially in their middle coats. The affected organs are firm to the touch and have a waxy appearance. The amyloid substance can be demonstrated macroscopically by pouring tincture of iodine on the affected organ, the waxy material being stained a deep mahogany colour. Microscopically, an iodine staining may be used, or a methyl-violet stain, which colours the amyloid substance pink and the parenchymatous cells blue. In the kidney, the small arterioles in the glomeruli, those around the convoluted tubules and those in the medulla, are first attacked. In the intestine, the arterioles in the villi stand out clearly. There are two forms of amyloid infiltration of the spleen: the diffuse waxy spleen, where the venous sinuses are outlined and the central artery of the Malpighian capsule is affected; and the sago spleen, where the Malpighian capsule is greatly enlarged by the amyloid infiltration—its central artery is untouched, but its branches into the capsule are greatly swollen by the waxy material. In all these organs the parenchymatous cells are unaffected directly by the amyloid infiltration, but in the later stages necrose, owing to interference with their nourishment.

Symptoms.—The patient is pale, but often has a waxy complexion with a bright colour in the cheeks. Chronic, profuse and painless diarrhoea is common. The urine contains a large quantity of albumin, and is usually fair in

amount and of low specific gravity. The liver and spleen are enlarged, and ascites and oedema of the legs are often present.

Prognosis.—If the chronic suppuration can be cured, the condition may sometimes disappear; but in the majority of cases this is not possible, as the septic condition is engrafted on to a chronic tuberculous or other granulomatous condition, which is almost always impossible to eradicate.

Treatment.—This consists in trying to remove the cause.

INTERMITTENT CLAUDICATION

So far the various conditions discussed have been classified according to the underlying pathology. Intermittent claudication is only a symptom, but it provides such a characteristic syndrome that it is best taken separately here. The term “intermittent limp” or claudication is applied to a condition in which severe pain, in one or both legs, comes on after walking for a certain distance.

Ætiology and Pathology.—In the large majority of cases this syndrome occurs in elderly men, who have well-marked calcification of the middle coat of the arteries of the lower limbs (Mönckeberg's degeneration, *q.v.*). In rarer cases it may be present in atheroma or diffuse hyperplastic sclerosis or thrombo-angiitis obliterans (*q.v.*). It may be associated with high blood pressure, gout, diabetes, syphilis, or excessive indulgence in tobacco.

The symptoms are due to the arteries of the leg being unable to supply the muscles with the increased flow of blood that the limb requires during walking. It is one of the earliest signs of partial impairment of the arterial flow.

Symptoms.—The characteristic pain comes on after walking a certain distance and is brought on more quickly by faster walking. It may be accompanied by cramp in the calves, and also by numbness or tingling sensations. It causes the patient to limp and finally to stop. After resting for a minute or two, he is able to continue walking, but the symptoms again recur after he has walked for a further period. Sometimes the pain may only be described as tiredness or may only be felt in the feet and unless a careful examination is made the pain may be attributed to flat feet. Often the patient may suffer from angina pectoris, the causation of the two conditions being very similar, or his anginal pain may seem to disappear as his intermittent claudication appears, really because he is no longer able to walk quickly enough to provoke the anginal pain. In nearly all cases of long standing there is absence of pulsation in the dorsalis pedis artery, or in the posterior tibial of the affected limb, which often shows signs of circulatory disturbance, being swollen, congested, and mottled, while the toes may be white and cold; but in early cases the arteries may still carry enough blood when the patient is resting so that it is more difficult to confirm the diagnosis. The amount of calcification in the arteries, which is often very extensive, may be determined by X-ray examination. In many cases dry gangrene of the limb has supervened.

Prognosis.—The prognosis is bad, but the attacks may persist for years before more serious results, such as gangrene, appear.

Treatment.—Exercise must be limited, and the patient warned to move

slowly and avoid hurrying in his walks. The production of intermittent venous occlusion may give helpful results. Diathermy has been used to relieve the pain. Heart muscle extracts and preparations of the pancreas given hypodermically have been tried, but without much success. Dry gangrene may require amputation of the limb, and when threatened may be relieved by removing the sympathetic nerves round the femoral artery.

ANEURYSM

Definition.—The word aneurysm is derived from the Greek to widen or dilate, and may be said to include any dilatation of an artery.

Aneurysms are generally divided into—

1. **TRUE ANEURYSMS.** in which the walls of the dilatation are formed by the coats of the artery. These may again be divided into—

(a) *Diffuse aneurysm.*—These are general dilatations of an artery. The dilatation is generally not great, and is of little clinical significance except that it indicates medial degeneration. The artery is sometimes tortuous in addition. The so-called cirroid aneurysm is a very extreme example of this.

(b) *Circumscribed aneurysm.*—These are limited to a segment of an artery or to a part of its circumference.

(c) *Dissecting aneurysm.*—These are caused by the splitting of the coats of the artery, the blood having passed through the lumen into the wall of the artery, separating one coat from another.

(d) *Arterio-venous aneurysm.*—In these there is a communication between an artery and a vein; there are two varieties in this group—(1) aneurysmal varix, and (2) varicose aneurysm.

2. **FALSE ANEURYSMS** are those following a wound or rupture of an artery, with the formation of a diffuse or circumscribed hæmatoma, and are bounded by tissues external to the wall of the artery.

When a true aneurysm ruptures and gives rise to a false aneurysm the resulting structure is known as a mixed aneurysm.

Ætiology and Pathology.—The two main factors in the causation of aneurysm are—(1) loss of the muscular and elastic fibres in the wall of the artery; (2) strain. The latter is generally brought about by repeated and prolonged muscular effort, and high arterial pressure may sometimes be an additional factor. The importance of strain as a causal factor is borne out by the fact that aneurysm is much more frequent in men than women—about five to one—and occurs more frequently in the fourth decade of life than at any other period. It also occurs much more frequently in hard manual workers, such as dock labourers, soldiers and sailors. *By far the most common cause producing weakening of the large elastic arteries is syphilitic inflammation. In persons dying of aneurysm, examination of the aorta in the neighbourhood of the aneurysm will generally reveal mesaortitis* (see page 1031). In the first part of the aorta this is nearly always so, but as one gets farther away from the aortic valves an increasing number of aneurysms are due to atheroma and non-syphilitic degeneration of the media; in the abdominal aorta less than half are syphilitic. Small aneurysms may also result from erosion of the walls of the arteries in cases of septic endocarditis—the so-called **mycotic aneurysm**. Frequently these are multiple; they occur most often

in the cerebral arteries or in the peripheral arteries. Extensive growth of streptococci and septic granulations may be seen in the neighbourhood of the dilatations.

Aneurysm may also be the result of congenital defects in the media of the vessel, which is very commonly seen about the circle of Willis, at the junction of the anterior communicating artery with the anterior cerebral. The aneurysms vary from about the size of a pin's head to that of a pea, and not infrequently their rupture gives rise to a diffuse subarachnoid hæmorrhage, the origin of which is often overlooked unless careful search is made for the aneurysm. Congenital aneurysm has also been described in the aorta, at the point of insertion of the ductus Botalli, and in cases of coarctation of the aorta, in which condition there is great narrowing of the aorta just below the origin of the left subclavian artery.

Loss of support by surrounding tissues also appears to lead to the production of aneurysm, *e.g.* at the base of a gastric ulcer a small aneurysm often projects as a nodule and is liable to rupture. Peptic erosion may also be a cause of weakening the walls of such arteries. In the cavities of the lungs, occurring as the result of pulmonary tuberculosis, it is quite common to find an aneurysm on the walls of the arteries lying in such cavities.

It is very doubtful if external trauma alone is ever the cause of true aneurysm, but injury to the artery by penetrating wounds by knives or bullets may certainly cause it.

Slight medial degeneration leads to diffuse aneurysm, and severe medial degeneration may cause circumscribed aneurysm. It is the usual cause of circumscribed aneurysms of muscular arteries, such as the popliteal, but is a very rare cause of circumscribed aneurysm of the aorta. Atheroma itself does not lead to aneurysm, but it may be complicated by medial degeneration. Continued high blood-pressure is an important contributory factor in the formation of a diffuse dilatation of the aorta, but this has few clinical resemblances to saccular or even to fusiform aneurysm.

One of the most striking appearances in an aneurysm is the coagulation of blood in the sac itself. This does not occur in diffuse dilatation of the aorta, but in those cases of sacculated aneurysm where the wall has become roughened. The sac becomes lined with fibrinous deposits, and occasionally an aneurysm may be cured by the deposition of successive layers of fibrin, so that the sac becomes almost completely filled. On the other hand, thrombus in aneurysms may form emboli and so lead to infarcts. Again, in many fatal cases of aneurysm, rupture and hæmorrhage have taken place, the deposition of fibrin having failed to prevent the blood reaching the surface. Rupture may take place externally, or into any of the hollow viscera or the serous cavities. Rupture into the pleura is common, as also is rupture into the trachea or into the œsophagus. In these cases, death is usually sudden, though oozing may have taken place some time before the final rupture. When the hæmorrhage forces itself into the connective tissues or muscles, it takes place much more slowly, and in the cases of the limbs may allow time for treatment. An aneurysm often exercises pressure on the other organs and structures in its neighbourhood. The heart is displaced away from the aneurysm; and the blood vessels are often narrowed so that circulation through them is impeded. The trachea, the bronchus, the œsophagus, and the nerves passing near the aneurysm also suffer. When the aneurysm meets

bony tissues, absorption of the bone takes place and the vertebræ are frequently eroded in this way, the bone being absorbed more rapidly than the intervertebral cartilage. When the aneurysm presses against the anterior surface of the chest, the ribs and sternum are pushed forward and finally are absorbed and perforated.

I. ANEURYSM OF THE THORACIC AORTA AND ITS BRANCHES

These are two types—(1) The diffuse aneurysm, or general dilatation, which occurs in medial degeneration of the aorta ; and (2) the circumscribed, usually saccular, aneurysm, almost always the result of syphilitic inflammation.

GENERAL DILATATION OF THE AORTA (DIFFUSE ANEURYSM)

Symptoms.—In general dilatation of the aorta the enlargement is extensive, but never reaches a very great size. The symptoms are generally due to atheroma and the medial degeneration that accompanies it. Very often there is interference with the coronary circulation, and this leads to diminution of the circulation of blood through the heart and consequently to cardiac pain. The aorta may sometimes be felt pulsating in the supra-manubrial notch, and the X-ray photograph will show general dilatation of the aortic arch. When the aortic ring is not stretched, the dilatation of the aorta beyond it may lead to the formation of a systolic murmur. Occasionally these patients die suddenly, on account of thrombosis of an atheromatous branch of a coronary artery. Provided there is no aortic regurgitation or coexisting coronary disease, patients with a dilated aorta may live for many years without serious discomfort.

CIRCUMSCRIBED ANEURYSM

These are generally saccular but may be fusiform.

Symptoms.—**ANEURYSM OF THE ASCENDING PART OF THE ARCH OF THE AORTA.**—An aneurysm of the ascending part of the arch tends to grow forward and outwards, and to produce a pulsating tumour that is palpable and audible at the level of the second or third interspace ; hence it is often called the *aneurysm of physical signs*. It often erodes the ribs and sternum. The tumour is tender and is often the seat of pain, which may be constant but is increased by exertion. A soft systolic murmur may be heard over it. If syphilitic aortitis spreads to the aortic valve or if the part adjacent to the aortic valves is affected, the aortic ring may be dilated and aortic regurgitation will take place. There will then be hypertrophy and dilatation of the left ventricle, with an aortic diastolic murmur, and the symptoms of aortic regurgitation will be added.

The pressure effects produced by an aneurysm of the ascending aorta are as follows : The heart is displaced downwards and to the left. The superior vena cava is pressed upon ; this may result in cyanosis of the head and neck, and œdema of the arms, and enlarged veins may occasionally be seen coursing over the front of the thorax. Very rarely the aneurysm may rupture into the superior vena cava, in which case the symptoms noted may come on quite suddenly and are very marked. There is often a systolic thrill, and on auscultation a continuous murmur, which is increased during

systole, is of great diagnostic value. The aneurysm may press upon the right bronchus, causing a chronic cough, due to irritation of the bronchus and stasis of its contents, and deficient or absent breath sounds over the upper lobe of the right lung. It may occasionally press upon the pulmonary artery and in rare cases, actually open into it, causing great dilatation of the right ventricle and auricle. Such aneurysms may present themselves to the left of the sternum rather than in the usual place, the right. Aneurysm of the ascending aorta has been known to perforate into the right ventricle and much more frequently into the pericardial sac. When the aneurysm comes forward it may irritate the pleura; and in some cases a loud pleuritic rub, audible over the aortic area, may be one of the early signs. Not uncommonly this form may rupture into the pleura or sometimes externally.

ANEURYSM OF THE TRANSVERSE ARCH OF THE AORTA.—As the arch of the aorta passes from right to left it also passes from before backwards, and consequently aneurysms arising from the transverse and descending parts of the aortic arch are situated more deeply in the chest than those arising from the ascending portion. Aneurysms of the transverse and descending parts of the aortic arch have been called by Broadbent *aneurysms of symptoms*, because their presence has often to be inferred by the pressure symptoms that they produce, while a pulsating tumour is only present in the very late stages and may not appear at all.

1. *Pain.*—Pain is one of the commonest and earliest symptoms of aneurysm. It may often occur behind the sternum or across the back and pass down the left arm and be very severe: it then lasts longer than true anginal pain and is more liable to occur at night; this form of pain occurs when the ascending aorta is distended. When, however, the transverse arch is affected the pain is sometimes felt on the left side of the neck and even in the occipital region; it is probable that this pain in the neck is a reflected pain caused by abnormal afferent impulses reaching the cervical spinal cord as a result of the distension of the transverse arch. A boring, persistent pain in the chest is probably the result of direct pressure of the aneurysm. Sometimes there may be true angina pectoris owing to involvement of the mouths of the coronary arteries.

2. *Respiratory Symptoms.*—Dyspnoea is common in aneurysm, and apart from associated heart disease is usually caused by pressure upon a bronchus. There is often stridor, which in this case is heard both in inspiration and in expiration. Hæmorrhage also occurs as a result of leaking of an aneurysm through the bronchus. It may at first be slight, but often a huge gush of blood supervenes, causing death. It is said that reflex irritation of the vagi will occasionally cause bilateral adductor spasm of the vocal cords and marked dyspnoea. In this case the stridor is only heard with inspiration and disappears if a little chloroform is inhaled. Patients with aneurysm often have a ringing, rough, brassy cough. If the pressure on the bronchus has been gradual, secondary changes occur in the lung, and compression of the left bronchus in the early stages may produce over-distension of the left lung with diminished or absent breath sounds, so as to lead to a suspicion of pneumo-thorax; later on bronchitis occurs with dilatation of the bronchus with expectoration of purulent phlegm. Pressure on the trachea may occasionally be observed in cases of aneurysm of the aortic arch, the larynx being drawn downwards and backwards with each cardiac pulsation. A

physical sign described by Surgeon-Major Oliver is known as *tracheal tugging*. The patient should be placed in the erect position, and directed to close his mouth and elevate his chin. The cricoid cartilage should be grasped between the finger and thumb and gentle steady upward pressure be made upon it. If there is an aneurysm, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand. This sign may occur in aneurysm before other symptoms are evident, but is not very reliable.

3. *Pressure on nerves.*—(a) The left recurrent laryngeal nerve.—This nerve courses round the arch of the aorta and passes up behind it, and is consequently often involved in aneurysm of the transverse arch. The abductor fibres of the recurrent laryngeal nerves succumb to the effect of pressure before the abductor fibres and consequently the vocal cord is at first in the position of adduction. During respiration, the right vocal cord moves up to and meets the adducted left vocal cord and respiration is unaffected. During phonation, the right vocal cord again comes up to and meets the left vocal cord, and the voice may be quite normal. It is therefore clear that a laryngoscopical examination may reveal the early stage of pressure on the left vocal cord before there has been any alteration in the voice. As the pressure increases, the adductor muscles are affected and the left vocal cord remains motionless in the cadaveric position half-way between full inspiration and full expiration. The voice is now hoarse, and it not uncommonly happens that hoarseness is the first symptom for which the patient presents himself. (b) The sympathetic nerve.—Pressure upon the sympathetic nerve causes first of all irritation and later on a paralysis of the cervical sympathetic fibres. When the sympathetic is irritated, the pupil on the same side is dilated, and there may be sweating and flushing of the same side of the face and ear. When the cervical sympathetic is paralysed, the pupil is smaller than on the opposite side, the eyeball may be sunken into the orbit, and there may be a slight degree of ptosis. Unequal pupils in aneurysm are, however, frequently found without any of the other symptoms of sympathetic irritation or paralysis, and in these cases Doctors Wall and Walker suggest that the difference in the pupils is due to the pressure of the aneurysm upon the arteries passing to the neck. They have shown that in a condition of low blood pressure the pupils are dilated, whilst in a condition of high blood pressure they are contracted, and that pressure upon one common carotid will cause dilatation of the pupil on the same side. They point out that under these conditions the dilated pupil is constantly on the same side as the smaller temporal pulse, and they consider that the dilated pupil and the small pulse are due to the same cause, namely, pressure upon the arteries supplying that side of the neck. Unequal pupils also occur as the result of syphilitic disease of the nervous system. We may get bilateral pin-point pupils, or the pupils may be unequal and irregular in outline. In both cases the reaction to light is lost, while the reaction to accommodation remains. (c) Pressure upon the intercostal nerves occasionally results from an aneurysm that presses backwards and erodes the vertebræ and posterior portions of the ribs. In these cases the pain is very severe along the affected nerves. In the distribution of the nerve itself the skin may be anæsthetic—the so-called *anæsthesia dolorosa*. (d) In some cases the aneurysm presses upon the brachial plexus, shooting pain occurring in the head and neck and down the right arm.

4. *Pressure upon the branches springing from the aorta.*—This is not uncommon, and may result in the absence of one radial or temporal pulse, or inequality between the two pulses. If the innominate artery is pressed upon, the right radial and temporal arteries may be small and imperceptible, while in an aneurysm of the transverse arch of the aorta, the left subclavian artery is compressed, in which case the left radial pulse may be affected. A sphygmographic tracing is often of value in demonstrating the difference between the two pulses, and a forced expiration, by increasing the intrathoracic pressure, will often accentuate the difference between the two sides. When the pulses are markedly unequal, the blood-pressure is also diminished on the side of the feebler pulse; a difference of pressure of over 30 mm. between the two sides is in favour of aneurysm provided it is not due to coarctation of the aorta.

5. *Pressure on the œsophagus.*—This may result in slight difficulty in swallowing but the dysphagia is very rarely important. Occasionally the aneurysm may ulcerate into the œsophagus so that death takes place from a sudden rupture.

ANEURYSM OF THE DESCENDING PORTION OF THE ARCH.—In these cases the sac frequently projects backwards and erodes the vertebræ from the third to sixth thoracic, causing great pain and occasionally compression of the spinal cord, resulting in paraplegia. Dysphagia is more common, and sometimes a tumour appears in the region between the scapula and the spine, and may attain a very large size.

ANEURYSM OF THE DESCENDING THORACIC AORTA may occur close to the diaphragm. Aneurysm of this form is frequently overlooked, pain in the back being the most prominent early symptom.

Diagnosis.—*Inspection.*—This is most essential. Abnormal pulsation should be looked for in the thorax, and can often be seen when the patient is seen obliquely in a good light. Posterior pulsation is generally observed to the left of the spine. Enlarged veins over the chest, suffusion of the face, and alteration in the pupil may be noted. The apex-beat is often displaced from its normal position, especially when the sac is large, this being due to pressure of the aneurysm on the thorax; the heart itself is seldom hypertrophied, unless there is a leakage through the aortic valves.

Palpation.—Palpation may reveal the area and degree of the abnormal pulsation. There may only be a diffuse impulse, but if the sac has perforated the chest wall, a forcible heaving and expansile impulse may be felt. Occasionally a diastolic shock is to be noted. This has been thought by some to be due to the forcible closure of the aortic valves producing an effect within the aneurysmal sac; another explanation is that the contraction of the heart draws in the ribs during systole at the point where they are adherent to the aneurysm and the diastolic shock is produced by the elastic recoil of the ribs and costal cartilages. Occasionally a systolic thrill may be felt.

Percussion.—A dull area may in some cases be made out in the second right interspace in cases of aneurysm of the ascending aorta. Much more rarely an aneurysm of the arch may press forwards and to the left, and produce dullness below the left clavicle. Pressure upon a bronchus may at one period result in hyper-resonance from lung distension, and later, owing to absorption of air in the lung, the percussion note may become dull.

Auscultation.—There may be no murmur, even in a large aneurysm, but a systolic murmur is not uncommon. When both systolic and diastolic murmurs are heard, aortic regurgitation is present in addition to the aneurysm. Accentuation of the aortic second sound is a most constant auscultatory sign of aneurysm, but it occurs with syphilitic aortitis before an aneurysm has developed.

Reference has already been made to alteration in the radial and temporal pulses. Exceptionally, in a large aneurysm of the descending aorta there may be absence of pulsation in the abdominal aorta and peripheral arteries of the legs, the dilatation of the thoracic aorta being sufficient to convert the intermittent into a continuous stream.

Examination by radioscopy is most important in every case where aneurysm is suspected and may give valuable information. The chest should be examined from the anterior, the posterior and the right oblique positions. The pulsation of the tumour and its relation to the aorta may actually be seen, but photographs should always be taken, both in the anterior-posterior and oblique positions, as much information is obtained from the density of the shadow cast by the aneurysm. If the latter is very dense, it can be reasonably inferred that deposition of the laminated clot has taken place within the sac.

Complications and Sequelæ.—The main complication causing death is rupture, which may take place either externally, into the pericardium, into the pleura, into the œsophagus, into the bronchus, or into the lung tissue itself. Pressure on the trachea, causing stridor and respiratory obstruction, is a very distressing complication. Bronchitis may occur during the course of the illness, and may be recovered from more than once. Broncho-pneumonia and gangrene of the lung not infrequently occur when there is pressure upon a bronchus, and empyema may occasionally result. Tuberculosis of the lung may coexist with aneurysm, but death from hæmoptysis, the result of perforation of a deep-seated aneurysm into the bronchus, has often been mistaken for the profuse hæmorrhage of tuberculosis. Cardiac failure is responsible for a large number of deaths. This may be the result of interference with the circulation through the coronary arteries, and in other cases it is due to the aortic regurgitation. Cerebral embolism sometimes occurs in cases of aortic aneurysm, a portion of the clot within the artery becoming displaced and passed up to the brain.

Course.—Most cases live from 2 to 5 years from the time when the first symptoms have appeared. Occasionally life may be prolonged for several years by treatment, provided the diagnosis is made early. Spontaneous cure may be obtained by deposition of laminated clots within the cavity of the aneurysm, but this is comparatively rare.

Diagnosis.—Intrathoracic aneurysm is sometimes difficult to diagnose from intrathoracic neoplasm. In both there may be an externally projecting tumour, but in aneurysm the pulsation may be seen to be expansile. The diastolic shock indicates an aneurysm. Systolic murmurs may occur in both conditions, but the ringing aortic second sound is of great importance, and is rarely heard in tumours. Tracheal tugging is in favour of aneurysm, while progressive wasting and enlargement of glands in the neck are in favour of neoplasm. Aneurysm, as a rule, occurs in apparently healthy men between 45 and 60 years of age, whereas malignant growth in the chest is associated

with emaciation and pallor. In aneurysm there is a greater likelihood of the pupils and pulses being unequal, while in neoplasm oedema of the upper extremities and chest wall is not uncommon. In all cases an X-ray examination should be made, and will nearly always clear up the diagnosis. Clinical evidence of infection by syphilis and a positive Wassermann reaction are obviously important.

A violently pulsating thoracic aorta, either in association with aortic regurgitation or with violent throbbing of the heart, may lead to the unfounded suspicion of an aneurysm.

In cases where an empyema is pointing on the left side in the region of the heart, the tumour may pulsate. The throbbing is usually diffuse and widespread, and there is a coexistence of a pleural effusion. Exploration with a fine needle will usually settle the diagnosis. It must, however, be remembered that occasionally an empyema may be the result of extension of septic trouble from a bronchus which has become compressed by an aneurysm.

Prognosis.—In aneurysm this is always difficult. Complete cure is very unlikely, although pain and other unpleasant symptoms and physical signs may give way to treatment. The presence of aortic regurgitation is unfavourable, while an aneurysm progresses much more slowly in people of a placid disposition and those who lead a quiet life. Even in cases where treatment has apparently been most successful and pain and dyspnoea have been apparently relieved, sudden death from rupture may occur.

Treatment.—The recognition that the main cause of aneurysm is the weakening of the wall by syphilitic mesaortitis has brought anti-syphilitic remedies into the forefront of modern treatment of aneurysm of the thoracic aorta. Mercurial inunctions and injections have been but little tried, but in many cases a good deal of benefit has been observed. The iodides have been given in aneurysm for many years—long before syphilis had been recognised as a cause of the condition. The most striking effect of iodide is the relief of pain, and this may be obtained by even small doses, such as 5 grains three times a day. In all cases, however, large doses, such as 20 grains three times a day, should be given a trial. Neoarsphenamine has been given intravenously in many cases with great success; it should always be tried when the diagnosis is made early enough, but should never be used if congestive failure or paroxysmal nocturnal dyspnoea have developed.

Efforts to produce clotting within the sac should be tried in early cases of aneurysm. Tufnell's method—that of a complete rest and restricted diet—is only of historic interest. The patient had to lie in a quiet and secluded room for several months. Few patients put up with such a rigid diet and rest. Secondly, the administration of lime-salts, such as the chloride or lactate of calcium, should also be tried, in an endeavour to promote clotting within the sac. The gelatin treatment has been abandoned.

Many patients with intrathoracic aneurysm do better if, after a preliminary rest with the treatment appropriate to their stage, they are allowed to follow their general vocations, provided their work be not too strenuous for body or mind. Nearly always it will be necessary for their activity to be curtailed. Patients should be cautioned to take things as easily as possible, to avoid alcohol, to eat with great moderation, and to avoid any sudden exertion. At least 10 hours should be spent in bed. A certain amount of tobacco may be smoked.

Special symptoms may have to be treated. For severe pain, cyanosis and dyspnœa, venesection will often give marked relief. Amyl nitrite and iodide of potassium are of great service in relieving the anginal pain of aneurysm. Severe paroxysmal dyspnœa is nearly always due to direct pressure on the trachea; both inspiratory and expiratory stridor are present. The inhalation of chloroform does not give relief, and tracheotomy is useless. In some very rare cases there may be a bilateral abductor spasm of the vocal cords, due to irritation of the vagi, and relief may be obtained by chloroform. Intubation of the larynx is preferable to tracheotomy, which should never be performed to relieve the dyspnœa of aneurysm.

Surgical Treatment.—This may be considered under four heads—(1) ligature of the vessels arising from the arch of the aorta; (2) the passage of wire into the sac with or without galvanism; (3) needling the sac; and (4) ligature of the neck of the sac. There are few cases where the outlook under medical treatment is so bad that the certain risks and uncertain benefits of surgical treatment can be advised with wisdom.

Ligature of vessels has been of little service in the case of aneurysm of the aorta. In aneurysm of the innominate artery, combined simultaneous ligature of the right common carotid and subclavian arteries may be tried, but even this may be insufficient to prevent the flow of blood through the sac. Moore's method of introducing silver or zinc wire into the sac through a cannula has been used, but the best results have been in cases of abdominal rather than intrathoracic aneurysm. Puncture of the aneurysm and scratching its wall with the point of a needle, as advocated by Sir William M'Ewen, has sometimes been partially successful. Ligature of the neck of the sac may be undertaken when it appears to be small, but suitable cases are very rare.

ANEURYSM OF THE INNOMINATE ARTERY.—This is not uncommon. It forms a pulsating tumour, which can sometimes be felt above the right clavicle, and nearly always produces marked diminution in the right radial and temporal arteries. In this form of aneurysm, paralysis of the right recurrent laryngeal nerve occurs not infrequently, the right vocal cord being paralysed instead of the left.

ANEURYSM OF THE CAROTID AND SUBCLAVIAN ARTERIES is mainly of surgical interest. It has been thought to be most frequent in the common carotid, especially in women, but in many of these cases there is really a kinked carotid artery, due to hypertension and athero-sclerosis, that simulates an aneurysm. Subclavian aneurysm is nearly as frequent as carotid aneurysm. Syphilis is found in nearly all cases near the aorta but trauma becomes more important towards the periphery.

II. ANEURYSM OF THE ABDOMINAL AORTA AND ITS BRANCHES

Aneurysm may occur in any part of the abdominal aorta, but it is much less common than aneurysm of the thoracic aorta. A forcible dynamic pulsation of the vessel is often mistaken for aneurysm and no case should be diagnosed as aneurysm unless a tumour can be grasped between the fingers. Often in true aneurysm there is evidence of syphilis and the Wassermann reaction is positive. A systolic thrill can sometimes be felt, and a systolic murmur is, as a rule, audible. The complications in abdominal aneurysm are

many. Death may result from complete obliteration of the lumen by clots, or by erosion of the vertebrae and compression of the spinal cord, resulting in paraplegia. Occasionally the superior mesenteric artery may become blocked by a clot and acute intestinal obstruction result. The commonest complication is rupture, which generally takes place into the retro-peritoneal tissues, with the formation of a large rapidly-growing tumour in the flank. More rarely death takes place from rupture into the peritoneum or duodenum.

Treatment.—The treatment of abdominal aneurysm is the same as that of thoracic aneurysm. In cases where medical treatment is unsuccessful after a fair trial, surgical measures should be undertaken and are more likely to be successful than in thoracic aneurysm.

ANEURYSM OF THE SPLENIC ARTERY is very rare. A tumour can be felt near the spleen and it may perforate into the colon. If the diagnosis can be made, removal of the aneurysm and of the spleen should be undertaken.

ANEURYSM OF THE MESENTERIC ARTERY, which is also rare, generally results in plugging of the vessel or its branches, with the result that acute intestinal obstruction takes place and death occurs from this cause.

ANEURYSM OF THE HEPATIC ARTERY is very rare.

ANEURYSM OF THE RENAL ARTERY has occasionally been noted, and in some cases successfully removed.

ANEURYSM OF THE BRACHIAL ARTERY used to be common, but is now rarely seen.

ANEURYSM OF THE FEMORAL ARTERY is much more common, and is most often traumatic.

ANEURYSM OF THE POPLITEAL ARTERY is one of the most common of the peripheral aneurysms. It has been suggested that this is due to the fact of the exposure to stress and strain to which the popliteal region is subjected during violent lifting efforts.

In all cases of peripheral aneurysm not due to trauma, a syphilitic basis should be investigated; but the aneurysms of the muscular arteries of medium size are almost invariably due to medial degeneration, and a syphilitic aneurysm would be most unusual. The treatment is mainly surgical. Distal or peripheral ligation and excision have all been tried, and more recently Matas has suggested treatment by *Reconstructive Endoaneurysmorrhaphy*. After rendering the limb exsanguine, he freely opens the arterial sac and by a process of suturing reconstructs a channel between the afferent and efferent artery of the sac. This is theoretically the best treatment, but in practice it is often found impossible.

When the symptoms warrant surgical treatment, proximal ligation is probably the method most frequently used for a large artery and excision of the sac for a smaller one. Proximal ligation as close to the aneurysm as possible has been the classical operation since the time of John Hunter. It should be combined with distal ligation, as this does not increase the risk of gangrene and diminishes the risk of spreading infection or of an embolus becoming detached.

Until the War of 1914–1918 great care was taken to avoid injury to the vein, but it was found that proximal and distal ligation of the vein as well as of the artery reduced the risk of gangrene. This should therefore be the routine surgical treatment.

III. DISSECTING ANEURYSM

This may originate in an atheromatous ulcer. Very often the dissection of the coat is small, especially when the blood-pressure is not high. When, however, there is a very high blood-pressure and much degeneration of the media, an extensive dissecting aneurysm may occur. The degeneration of the media may cause a small split in the intima and the dissection separates the intima from the media, so that in some cases there may be a double tube instead of a single aorta. Extensive dissection frequently causes sudden death, but in other cases the patient may live on, and the association of a rapidly beating heart and a feeble pulse in the lower limbs has been suggested as a clinical sign by which the condition may be recognised. The immediate picture is very similar to that of a coronary thrombosis. If the dissection spreads up the common carotids, symptoms from interference with the cerebral circulation may follow and this soon after an attack, suggesting coronary thrombosis may reveal the diagnosis. Occasionally the patient makes a complete recovery and lives for years with the circulation through the new channel made by the dissection.

In many cases of extensive dissecting aneurysm of the aorta, due to medial degeneration, vascular hypertrophy is well marked, and consequently the blood pressure was high during life. Less commonly dilatation, rupture and the dissecting aneurysm of the aorta are apparently due to congenital weakness, histological evidence of degeneration or inflammation being absent.

IV. CIRROID ANEURYSM

Cirroid aneurysm is a condition in which an artery is dilated and tortuous. Occasionally it is due to medial degeneration of muscular arteries, particularly the splenic and temporal, but this form is of little clinical importance. More often it is due to defective development of the walls of arteries and their branches, and this form has been called serpentine angioma. The arteries, their branches, the capillaries, and even the efferent veins dilate progressively, causing destruction of the intervening soft tissues and erosion of bone. The superficial temporal, posterior auricular, and occipital arteries are most commonly affected. It also occurs in the brain, pancreas, orbit, and limbs. It is most common between puberty and 30 years of age. Although the condition is generally congenital it may develop greatly after some local injury such as a blow.

Pathology.—The arteries are dilated, thinned and very tortuous, and the disease tends to spread towards the capillaries and also along the arteries that feed the aneurysm. The skin over the aneurysm is often atrophied and may become ulcerated, leading to very dangerous hæmorrhage.

Symptoms.—There is an ill-defined pulsating tumour on the scalp, in which the tortuous vessels may be felt. In rare cases the tumour may be slow in its growth, but this is generally rapid and the skin over it ulcerates, leading to hæmorrhage.

Treatment.—This is very difficult. In limited cases the tumour may be excised. Generally, however, ligature of the peripheral arteries of the growth is more satisfactory. Electrolysis and injections of perchloride of iron into the

mass have been tried, with some success. Occasionally spontaneous cure follows some infection.

V. ARTERIO-VENOUS ANEURYSM

Arterio-venous aneurysms, in which an artery and a vein communicate, are of two kinds: (1) aneurysmal varix, where the two vessels anastomose directly; and (2) varicose aneurysm, where the sac separates the connecting vessels.

(1) ANEURYSMAL VARIX.—The aneurysm is usually traumatic in origin and used to be frequent at the elbow, as the result of venesection. The artery is wounded at the same time as the vein and they become connected, the result being that the vein becomes markedly dilated and tortuous.

The varix forms a soft, compressible, ill-defined tumour, which pulsates. Pain in the tumour is not uncommon. A marked thrill can often be felt and a loud bruit may be heard over the tumour. If the limb is raised the tumour shrinks, while it becomes large and congested if the limb is held downwards. The limb below the tumour is often œdematous.

In some cases the aneurysm remains stationary, and all that is required is an elastic support. If, however, it tends to increase in size, the artery should be ligatured above and below its communication with the vein, and the vein should also be ligatured.

An intrathoracic aneurysm may become adherent to a vein and perforate into it. The most common site is when an aneurysm of the ascending arch of the aorta perforates the superior vena cava, but even this is rare. The latter vessel becomes greatly distended and an arterio-venous aneurysm is formed. There is often a sudden onset when the lumen of the two vessels becomes connected; there is congestion of the head and neck and upper limbs, great distension of the veins, and often œdema. On auscultation over the tumour, a continuous humming murmur is heard, with marked accentuation during systole.

(2) VARICOSE ANEURYSM.—This occurs when an artery and vein are simultaneously wounded. A false aneurysmal sac is formed in the tissues and communicates both with the artery and vein. The symptoms are similar to those of an aneurysmal varix, but in addition there is a pulsating tumour, which can be distinguished from the dilated vein. This form of aneurysm should be excised by open operation combined with four-fold ligature of the artery and of the vein. It is not often seen in civil life but is fairly common in war-time, mainly as a result of gun-shot wounds.

ARTERIO-VENOUS ANEURYSM OF THE ORBIT OR PULSATING EXOPHTHALMOS.—This is a form of aneurysm by anastomosis, due to a communication having formed between the cavernous sinus and internal carotid artery as it passes through it. It is generally the result of a fracture of the base of the skull. It is usually unilateral, but may be bilateral, and the communication may take place immediately after the fracture, or evidence of the lesion may only appear days or weeks after the injury.

The main symptom is protrusion of the eyeball, the globe being displaced outwards and downwards. It may be seen to be visibly pulsating, but if not, slight pressure upon the globe of the eye will bring out pulsation. A loud bruit, either continuous or increased during systole, may be heard anywhere

over the head ; this roaring sound is generally very distressing to the patient. There is great dilatation of the veins around the eyelids, conjunctivæ, and fundus. Headache is common

The condition may last for years, and in a few cases spontaneous recovery has taken place.

The treatment is either compression or ligature of the carotid artery. The great danger in ligaturing the carotid artery is the occurrence of cerebral softening and hemiplegia. To avoid this, it is better to ligature the artery temporarily and see if any cerebral symptoms tend to develop ; if they do, the ligature should be removed after 24 hours ; but if they do not, the ligature may be tightened and the artery completely occluded.

DISEASES OF THE PULMONARY ARTERIES

The pulmonary artery is much less frequently the seat of disease than is the aorta, but it is liable to be affected by pathological changes of a similar character to those that are found in the systemic arteries.

Ætiology and Pathology.—Four main pathological changes are generally recognised, namely

1. **HYPERTROPHY.**—This condition is associated with an increase in the blood pressure in the lesser circulation and is found in cases of disease of the lungs, such as emphysema, pulmonary fibrosis, and bronchiectasis, and also in disease of the heart, such as mitral stenosis. The intima of the pulmonary arteries hypertrophies and is prone to degeneration, so that patches of fatty change (atheroma) appear in the hypertrophied tissue. This form of intimal degeneration is superimposed upon hypertrophy, which is the result of obstruction to the lesser circulation and is independent of inflammatory change.

2. **INFLAMMATION.**—There is no doubt that *syphilitic* inflammation may attack the pulmonary arteries as well as the aorta. The larger trunks may be affected by mesarteritis, and saccular aneurysms of the main branches have been described, but are rare. The smaller arteries and arterioles in the lungs may also be affected by syphilitic arteritis, with endarteritis obliterans. The endarteritis, either by itself or in combination with thrombosis, may lead to complete occlusion of the lumen. Ayerza and his pupils have emphasised the importance of syphilis as an ætiological factor in the production of cyanosis and congestive heart failure in the absence of the usual causes of these conditions, such as emphysema and fibrosis of the lungs, or mitral stenosis. Fatty or calcareous changes may occur as secondary changes in the walls of the inflamed arteries.

Tuberculous inflammation of the branches of the pulmonary arteries is also common. Tuberculous endarteritis obliterans is frequent in phthisis. Tuberculosis of the lung or a bronchial gland may extend through the wall of a large artery to its intima, giving an intimal tubercle which when softened can lead to a general dissemination. The wall of a pulmonary artery exposed in a tuberculous cavity is frequently weakened by tuberculous or pyogenic invasion from without, and an aneurysm results. The profuse hæmoptysis found in the latter stages of chronic pulmonary tuberculosis results from the rupture of one of these aneurysms.

3. **DEGENERATION.**—Degeneration of the intima secondary to intimal hypertrophy or to inflammation has been described above. Slight primary intimal degeneration, or atheroma, is not uncommon in elderly subjects. C. F. Coombs and others have described cases of a severe atheroma of the pulmonary artery in young subjects, in whom there was no evidence of syphilis or of pulmonary or cardiac lesions, and suggest that in these cases there is an inherited tendency to intimal degeneration.

4. **CONGENITAL MALFORMATIONS.**—In the majority of cases a pulmonary stenosis is present, but in rare instances dilatation of the vessel has been found. These conditions are described under congenital heart disease (*q.v.* p. 968).

Symptoms.—The symptoms of disease of the pulmonary artery are those of obstruction of the lesser circulation.

Dyspnoea is often an early symptom, and may occur on exertion or in nocturnal paroxysms. In the later stages it becomes constant, with attacks of orthopnoea. Cyanosis is one of the most characteristic manifestations, but it varies in intensity and also in the stage of the disease at which it appears. Cyanosis is due to an imperfect oxygenation of the blood passing through the lungs at each cardiac cycle. It will, therefore, be most marked in those cases in which only a small proportion of the blood passes through the lungs at each beat, as in congenital pulmonary stenosis, or those in which the capillary area in the lungs has been so reduced by emphysema or the lung itself has been so damaged by fibrosis that the circulating blood is imperfectly oxygenated. In mitral stenosis the degree of cyanosis is remarkably variable and probably depends as to whether secondary changes in the lungs have developed. In Ayerza's disease (syphilitic inflammation of the pulmonary arteries) the cyanosis may be extreme and the patients may have almost a black appearance (*cardiacos negros*). This may be due to the endarteritis obliterans of branches of the pulmonary artery, or to a coexisting syphilitic obliterating bronchitis, or a syphilitic pneumonia causing fibrosis of the lung.

Hæmoptysis may occur before cyanosis has become established or in the later stages. It may be slight or profuse, and may be associated with attacks of pulmonary artery thrombosis. Cough, with mucopurulent expectoration, is common, and attacks of vertigo may occur. Somnolence is not infrequently found when marked cyanosis is present. The fingers are not clubbed, except in cases resulting from bronchiectasis or fibroid lung.

The pulse is usually regular and the heart is much enlarged, especially the right ventricle. If mitral stenosis is present diastolic murmurs may be heard at the apex. There are no constant physical signs in the lungs, but if emphysema, fibrosis, or bronchiectasis has been the determining factor, the physical signs characteristic of these conditions will be found. Œdema is often present and may be extreme and the liver enlarged. The spleen is not palpable. The blood shows an increase in the number of red cells, up to 8,000,000, the number varying with the degree of cyanosis.

The radioscopic findings are characteristic. The right ventricle is enlarged, the pulmonary artery often dilated, and the branches of the pulmonary artery show more clearly than usual and can be followed into the lung and in some cases can be seen to pulsate. The electrocardiogram shows a marked right ventricular preponderance and alterations in the *P* wave, suggesting right auricular hypertrophy.

Diagnosis.—Cases of pulmonary artery affections secondary to pulmonary or cardiac disease can be distinguished by the presence of the symptoms and signs of the underlying lesion. There is no sure method of diagnosis between syphilitic and non-syphilitic cases. In syphilitic pulmonary arteritis the patients are usually between 30 and 50 years of age and may give a history of syphilitic infection. The Wassermann reaction in the blood is positive.

Course.—There may be a history of pulmonary symptoms, such as cough and dyspnoea, for many years. Later the intense cyanosis may develop, and this may last for 4 or 5 years. Some of these patients die in their sleep, but in others myocardial failure, with advanced anasarca, is the cause of death. Others die of complications, such as broncho-pneumonia.

Prognosis and Treatment.—The outlook depends on the causative factor. In early cases in which syphilis has been established as the cause of the inflammation of the pulmonary artery, anti-syphilitic treatment will retard the progress of the disease. In paroxysms of cyanosis, venesection gives marked relief. The usual treatment for congestive heart failure must be adopted, when this has supervened.

PHLEBITIS

Phlebitis or inflammation of the veins may be sharply divided into two great classes—(1) non-suppurative or plastic, and (2) suppurative. The terms endo- and peri-phlebitis have been used to indicate inflammation of the internal and external coats. Peri-phlebitis results from invasion of the veins by inflammatory processes outside it, or from injury. It may extend inwards towards the lumen of the vein, and result in endophlebitis and generally clotting of the blood within the vein. Endophlebitis is usually the result of poisons or microbes circulating within the vein. Inflammatory changes of a plastic type occur in the endothelium, and in consequence a clot or thrombus is set up within the vein. The clot may adhere to the vessel wall and completely obliterate it. Organisation of the clot by fibrous tissue may occur, the vein being transformed into a hard fibrous cord. In other cases the clot may become softened and broken down, and the circulation may be resumed through the vein. In certain cases changes in the composition of the blood may lead to clotting in a vein, and the presence of this clot itself may give rise to a plastic phlebitis; this is sometimes called *thrombo-phlebitis*. In other cases the vein and contained clot may become invaded by pyogenic organisms, and leucocytes will enter the clot and cause it to break down into a purulent fluid.

PLASTIC PHLEBITIS

Ætiology.—(1) Traumatic phlebitis; (2) the formation of a non-infective clot—thrombo-phlebitis; (3) gouty phlebitis may accompany an attack of gouty arthritis or may occur independently; (4) typhoid fever not infrequently causes phlebitis and thrombosis; (5) in pneumonia and influenza phlebitis is not an uncommon complication; (6) post-operative phlebitis is not at all uncommon in cases of operation on the lower abdomen and the

bladder; and (7) puerperal phlebitis or phlegmasia alba dolens frequently follows parturition.

Phlebitis may attack any vein, but is most common in the lower limb, particularly in the saphena vein.

Symptoms.—Phlebitis is accompanied by pain and tenderness in the course of the affected vein, which can be felt as a hard cord. The skin may become reddened over the superficial veins, and the limb is often œdematous when thrombosis has taken place. There is usually more or less febrile disturbance. In gouty phlebitis, the pain is often severe, the areas of inflammation are often multiple; there is a great tendency for more than one vein to be attacked at once, and, in opposition to most forms of phlebitis, the disease may be symmetrical.

Complications and Sequelæ.—The complications and sequelæ of plastic phlebitis are those of the thrombosis that accompanies it, and will be described under thrombosis.

Prognosis.—This is generally good apart from the risk of embolism or of the thrombosis spreading towards the large veins, such as superior or inferior vena cava. Treatment must be carefully carried out to minimize the risk of this.

Treatment.—Patients with phlebitis should be put to bed, and the limbs elevated and wrapped in cotton wool. All sudden movement, friction, or handling should be avoided. The bowels should be freely opened, as chronic constipation and stasis in the colon may impede the circulation in the iliac veins. In gouty phlebitis, the diet should be restricted to fish and light farinaceous foods, but when patients are marasmic and anæmic, the diet should be as liberal as their digestive powers will permit. In all cases of phlebitis foods containing much lime-salts, such as milk, should be limited. Potassium or sodium citrate with carbonate of ammonia and liquor ammonii acetatis are of service. Glycerin of belladonna smeared over the inflamed vein appears to ease the pain.

SUPPURATIVE PHLEBITIS

Ætiology.—Suppurative phlebitis is the result of infection of the walls of the veins with pyogenic organisms. The micrococci may be in the circulating blood, as in some cases of puerperal phlebitis, or they may reach the veins from a focus of suppuration around it, as in facial carbuncle, middle-ear disease, or inflammation of the portal veins—suppurative pylephlebitis.

Pathology.—The coats of the vein are infiltrated with leucocytes, the clot which has formed within the vessel breaks down into yellow pus, and abscesses are not infrequently found along the course of the vein. Not uncommonly the septic inflammation spreads along a vein, splitting up the coats.

Symptoms.—There is a throbbing, smarting pain in the region of the affected vein, and the part drained by it is œdematous. Not infrequently the septic process spreads along a vessel. There is often fever, a rapid pulse, a dry tongue, and delirium, and in many cases a succession of rigors indicates the development of pyæmia.

Complications and Sequelæ.—These depend upon the situation of the vein and the occurrence of emboli, owing to breaking away of the softened

clot. (See Thrombosis.) When the vein is superficial the diagnosis is easy, but when a deeply-seated small vein is affected the only symptoms may be those of the pyæmia to which it gives rise.

Treatment.—The prevention of this disease by asepsis is one of the great advances in modern surgery. As soon as the disease is recognised, a ligature should be placed upon the vein between the affected area and the heart, the inflamed vein should be thoroughly laid open, the septic clot removed and the cavity thoroughly cleansed. In some cases where numerous abscesses are formed, amputation is the only means of arresting the general infection.

Thrombosis of the portal vein is commonly the result of septic conditions within the abdomen, most often suppuration in the region of the appendix. The condition is not always acute. Peptic ulcers may lead to portal thrombosis, and tubercular glands along the course of the portal vein have been found to cause clotting within the lumen of the vessel. Dysenteric ulcers resulting from bacillary dysentery may also give rise to septic portal thrombosis, but typhoid ulceration very rarely does so.

The effect of portal thrombosis is to produce a portal pyæmia, portions of the clot passing into the liver and causing abscesses within the radicles of that organ. Occasionally the portal vein itself becomes converted into a sac containing pus, and the liver is then riddled with abscesses along the course of the portal branches. This condition is known as *portal pylephlebitis*.

Symptoms.—The symptoms of septic portal thrombosis are the occurrence of fever and of rigors. The liver becomes enlarged and tender, and jaundice of a slight degree is quite common.

As the condition is such a generalised one, recovery very seldom takes place, even if treatment is adopted.

Treatment.—This consists in removing the cause and incising any liver abscesses.

MAURICE CAMPBELL.

THROMBOSIS AND EMBOLISM

Thrombosis is the name applied to the coagulation of blood within living vessels, whether in the heart, the veins or the arteries.

Embolism is the process whereby a portion of clot or other substance, such as parasites, fat globules, masses of bacteria or particles of tumour, is carried from one part of the circulation to another, and is impacted when it arrives at a vessel too narrow for its further progress. An infarct is the degenerated or necrosed condition of the tissues due to interference with the circulation of blood within it, and can be caused by embolism, thrombosis, endarteritis, endophlebitis or strangulation of veins. An infarct is generally wedge-shaped in outline, with the base towards the periphery of the organ affected. As seen post mortem, it is either yellowish-white in colour (the white infarct), or blood-red in colour (the hæmorrhagic infarct).

In the case of the white infarct the tissue deprived of its blood supply becomes permeated with lymph from the surrounding living tissue, and coagulative changes take place in it. In the case of the kidney or spleen, the

coagulable material is sufficient to render the infarct hard. In the case of the brain, less coagulable lymph is poured out, and the area of the brain affected becomes softened. In the early stages there is often a zone of congested vessels around a white infarct; this is a reaction on the part of the surrounding living tissues to the presence of the dead material. Later the infarct becomes invaded by fibrous tissue and a scar results.

In hæmorrhagic infarct, coagulation and necrosis also take place, but to this is added hæmorrhage, by diapedesis of the red blood cells from vessels of the collateral circulation. A hæmorrhagic infarct is commonly seen in the lung; a cone-shaped area of lung tissue becomes hard and dark-red in colour. Should the embolus, instead of being aseptic, contain living micro-organisms, a septic process is set up within the infarct and an abscess results. Such purulent abscesses are commonly seen in the lungs, as the result of septic phlebitis, and occasionally in the systemic system, as the result of septic endocarditis.

THROMBOSIS

Ætiology.—The causes of thrombosis are—(1) altered conditions of the blood or increase in its coagulability; (2) slowing of the current of the blood within the vessels; and (3) a lesion of the lining membranes of the vessel or cavity of the heart. Thus, thrombosis may occur in cases of anæmia or after infections or operations, where the blood is more coagulable than normal; in the appendages of the dilated auricles of the heart where the movement of blood is feeble or in peripheral vessels or in the lungs when the circulation as a whole is feeble, *e.g.* with a failing heart; and it may result from inflammation of the lining of the vessel or degenerative changes in its endothelium, especially when there is atheroma associated with much narrowing of the channel obstructing the circulation.

(1) **INTRA-CARDIAC THROMBOSIS.**—This is one of the commonest forms of thrombosis and is very important. It occurs in the left auricle, when it has become extremely dilated as the result of mitral stenosis. The ante-mortem clot generally begins to form in the dilated appendix of the left auricle, but may extend by the deposition of excessive layers of fibrin to invade the auricle itself so that a large ball thrombosis be formed within the cavity. Portions of the ante-mortem clot may break away from the thrombus, and may be carried into the left ventricle and into the general circulation, and embolism may occur in the brain, spleen, kidneys, intestines and the main arteries of the limbs. This danger is mainly to be feared in the first few weeks after the deposition of the thrombus, but may of course recur as fresh thrombi form.

Ante-mortem clot is occasionally deposited among the meshes and cavities of the dilated left ventricle. In cases of cardiac fibrosis and myelomalacia cordis, the result of disease of the coronary arteries, and especially after coronary thrombosis and cardiac infarction, the lining of the heart may become affected and fibrinous deposits occur within either ventricle. Lastly, in septic endocarditis, large vegetations, consisting of clot and masses of micro-organisms, may occur. Inflammation of the ventricle and of the auricle may also be present, and ante-mortem clot may be deposited on these roughened surfaces. Portions of this clot may leave the ventricle and pass into the

general arterial circulation, where the effect produced will depend upon whether the emboli are aseptic or contain micro-organisms.

Thrombosis in the right auricle occurs in many conditions where there is gradual cardiac failure and dilatation of the right side of the heart. Portions of ante-mortem clot form in the right auricular appendix, and parts may break away and pass into the lungs and an embolism of the pulmonary artery result. If the embolus is sufficiently large to cause blocking of the artery or of one of its main branches, sudden death ensues, but if only one of the smaller branches is affected, a pulmonary infarct results. Much more rarely ulcerative endocarditis occurs on the right side of the heart, and portions of the valves or affected clot reach the lungs in the same way.

(2) **ARTERIAL THROMBOSIS.**—Arterial thrombosis is rarer than venous thrombosis. It is sometimes due to arterial embolism, but is also the result of trauma or disease of the arterial walls, such as atheroma or endarteritis.

Thrombosis of the coronary arteries is a very important condition, since it is a frequent cause of sudden death and of serious cardiac disability. It has already been described (pp. 991–994). The usual artery affected is the anterior interventricular branch of the coronary artery. Atheromatous plaques are constantly found within the thrombosed vessel, and sometimes atheroma has occluded the orifice of the artery. In cases where the circulation has been slowed and greatly diminished before the final clotting, changes in the wall of the left ventricle are very common. Syphilitic mesoarteritis sometimes occludes the orifices of the coronary arteries but this is much less common.

Thrombosis is very frequent in the small cerebral arteries, especially when they have become narrowed as the result of disease. In early middle-age this narrowing is usually the result of syphilitic endarteritis, but most patients are older and then the arterial lumen is diminished by atheromatous changes in the wall of the vessel.

Thrombosis of the main artery of a limb usually results in gangrene; the limb becomes first white and pallid, later mottled in appearance, and finally black. If the patient survives the immediate shock and the disease to which the thrombus owes its origin, a line of demarcation will form between the vital and devitalised tissues, and the limb should be amputated well above this level.

Thrombosis of the retinal artery is more common than embolism. There is a sudden painless loss of sight in one eye, and generally the blindness is permanent and complete. Within a short time there is opacity of the central parts of the retina, and the macula shows up by contrast as a bright cherry-red spot.

(3) **VENOUS THROMBOSIS.**—*Thrombosis of the lateral sinus* occurs in disease of the middle ear. The mastoid cells become infected with pyogenic organisms and the disease spreads to the petrosal or sigmoid sinus. The clot in the vein becomes softened by pyogenic organisms, and particles break away and are conveyed to the lungs, in which pyogenic abscesses are formed.

The symptoms of septic thrombosis of the lateral sinus—and its continuation of the jugular vein—are infiltration of the tissues of the neck, with a cord-like induration of the vein itself, with some restriction of the movements of the head. There is a history of a chronic and often offensive discharge from the ear of the same side. A high temperature and rigors,

due to flooding of the circulation by poison, occur when a portion of the septic clot is dislodged. In these circumstances the jugular vein should at once be ligatured below the clot, in order to prevent further portions of the clot gaining access to the blood stream. A radical mastoid operation should also be performed, the sinus opened and its septic contents removed.

Thrombosis of the longitudinal sinus of the brain occurs as the result of injuries and infected wounds of the skull. It is a common war injury, the vertex of the head having been injured by a bullet as the soldier passes along the trench. Thrombosis of the cranial sinuses also occurs in marasmic patients, but it is usually agonal.

In many of these cases, owing to the vertical position of the leg areas in the brain, a paraplegia is produced, while the arms are not affected. The condition should be treated by trephining and draining the cranial cavity. Occasionally the longitudinal sinus becomes thrombosed in septic conditions in children and also in chlorotic anæmia in adults.

Thrombosis of the cavernous sinus is not infrequently the result of the extension of a chronic suppurative process of the sphenoidal cells at the back of the nose. The cavernous sinus is also affected by septic processes on the face; a small boil on the nose or a mosquito sting on the face may produce a septic thrombosis of the angular vein; this vein communicates with the ophthalmic vein, and the septic clot may extend along the latter into the cavernous sinus. As the venous plexuses of the pterygoid and zygomatic fossæ communicate through the foramina in the middle fossa and by the inferior ophthalmic vein, purulent inflammation of the jaw and of the teeth sockets is sometimes a cause of cavernous thrombosis. The result is that a marked degree of exophthalmos and swelling of the lids, and œdema of the optic disc and extensive retinal hæmorrhages occur. Not infrequently the septic condition of one cavernous sinus spreads to that on the other side through the circular sinus, and the exophthalmos may be double. Death from pyæmia or meningitis results. Owing to the position of the sinus, operation is impossible.

Thrombosis of the central retinal vein is common in elderly patients with athero-sclerosis and high blood pressure. The loss of sight is not so sudden or complete as in blockage of the artery. There is often albuminuria. Glaucoma often develops, but if the thrombosis is in a tributary vein the degree of recovery may sometimes be fairly good.

Femoral thrombosis is perhaps the commonest form of thrombosis. It frequently occurs after parturition and in anæmic and marasmic states. It is met with after infectious fevers, especially after typhoid fever, more rarely after influenza and pneumonia. It also follows operations, especially if a septic condition has been dealt with, or results from the operation. The thrombosis generally occurs in the femoral vein, and there is often some rise of temperature and a slight rise in pulse rate. It is most common on the left, because of the pressure of the right common iliac artery on the left common iliac vein. The limb affected becomes œdematous and a hard cord is found in the course of the vein.

In other cases thrombosis of the veins may also occur in marasmic conditions secondary to carcinoma, tuberculosis and tertiary syphilis.

Complications and Sequelæ.—Collateral circulation is usually satisfactorily accomplished in femoral thrombosis. In cases where the arteries

as well as the veins are involved gangrene may occur. Occasionally the thrombosis may spread up into the iliac vein and into the inferior vena cava, in which case both legs may become swollen and œdematous. Even in cases where the inferior vena cava has become thrombosed, recovery may take place, collateral circulation being established by means of veins passing up from the legs into the axillæ. If, however, the clot reaches the entrance to the renal veins, death nearly always results from renal thrombosis. Embolism is not at all uncommon in femoral thrombosis, the clot passing into the right auricle, and then into the right ventricle and pulmonary artery.

Treatment.—Complete rest for at least 3 weeks, as a precaution against embolism, should be insisted upon. Limitation of foods containing quantities of lime salts, such as milk, and the administration of citrates and salts of ammonia will materially help in the treatment of the case. The leg should be elevated and wrapped in cotton wool and kept warm.

EMBOLISM

Embolism may occur in three main situations, namely—(1) in the systemic circulation; (2) in the pulmonary circulation; and (3) in the portal circulation.

Emboli in the systemic circulation are derived from ante-mortem clots in the left auricle and left ventricle. These clots are formed in cases of mitral stenosis and more rarely in mitral regurgitation, and also when the left ventricle is greatly dilated and hypertrophied. In these cases the emboli are aseptic. Systemic emboli also occur in septic endocarditis, when portions of the valve break away or masses of fibrin and micro-organisms pass into the general circulation. These emboli are septic, and when they reach their destination usually form abscesses.

Emboli in the pulmonary circulation may have their origin in clots formed within the right auricle and right ventricle, or more rarely from septic endocarditis of the tricuspid and pulmonary valves; they may also come from any part of the systemic venous system. A very important form of pulmonary embolism is met with after abdominal operations and after childbirth. About the tenth day after an apparently successful abdominal operation or an uneventful parturition, a pulmonary embolism may occur with appalling suddenness. Death may take place at once, or hæmoptysis and pleurisy supervene. The clot forms in the common iliac vein, at the junction of the internal and external iliacs. Not only may a clot pass along the veins, but we also get droplets of fat in fat embolism, air bubbles in air embolism, and masses of parasites in parasitic embolism.

Two recent studies, one of autopsy cases of hæmorrhagic infarct of the lung and one of post-operative cases of thrombosis and embolism, support the view that embolism is much the most common cause of pulmonary infarction (87 per cent.) and that this generally comes from a venous thrombosis. In 14 per cent. of the autopsy cases a massive pulmonary embolism was the main diagnosis and most of these were after operations or fractures. In 47 per cent. heart disease with the accompanying stagnation of the blood stream appeared to be a predisposing factor. A potential source for the

embolus was found in 75 per cent., most often in the pelvic or leg veins. Massive embolism was more likely to occur on the tenth day but was not infrequent at any time in the first fifteen days ; it was independent of sepsis and depended on the stagnation of the blood stream, either from the state of the heart or more often from the mechanical conditions of rest. Smaller infarcts were more closely associated with sepsis (which suggests that the embolism is more often due to the breaking off of a small part of the thrombus) and more erratic in the time of their appearance.

In embolism in the portal circulation portions of clot in the radicles of the portal vein are finally arrested in the liver.

Symptoms.—1. EMBOLISM OF THE CEREBRAL ARTERIES.—The onset is sudden, and the left side of the brain is rather more often affected than the right. Hemiplegia or aphasia is produced ; consciousness is lost only for a few minutes during the attacks.

2. EMBOLISM OF THE SPLENIC ARTERY.—The onset is sudden, with pain in the left side and sudden enlargement of the spleen, which is very tender. During the next few days, the spleen diminishes in size, but it often remains permanently enlarged.

3. EMBOLISM OF THE RENAL ARTERIES.—Sudden pain in the back is produced, and blood and a little albumin are present in the urine. The hæmaturia may last for a day or two, or longer if a larger branch was involved.

4. EMBOLISM OF THE SUPERIOR MESENTERIC ARTERY.—The patient is seized with sudden, violent abdominal pain and distension. The collateral circulation, in spite of the numerous vascular arteries that supply the intestine, fails, and gangrene of the small intestine results. There is a complete intestinal obstruction, and blood finds its way into the stools and into the peritoneal cavity. Operation should be undertaken at once, but owing to the large amount of bowel affected recovery is very rare.

5. EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.—This is not an uncommon event in cases of mitral stenosis and in septic endocarditis of the aortic and mitral valves. The patient is seized with pain in the eye and becomes suddenly blind on one side. The optic disk becomes pale and the retinal vessels small. Occasionally only a single branch of the vessel is affected. The same picture is found more often as a result of arterial thrombosis in elderly patients with athero-sclerosis or high blood pressure.

6. EMBOLISM OF A LARGE ARTERY IN A LIMB.—This sometimes occurs. There is acute pain in the limb, followed by numbness and loss of power. The pulse is imperceptible below the seat of the embolism, and gangrene results.

7. AIR EMBOLISM.—Air may enter the veins during operation on a large vein, or during intravenous injection of saline or other solutions, or after distension of the bladder and the urethra with air. Air embolism is undoubtedly capable of causing death, but a small quantity may enter a vein without any effect whatever, or, if some disturbance should arise, without fatal termination. The exact way in which air embolism causes death is doubtful ; it may be due to arrest of the pulmonary circulation or to cerebral anæmia.

The diagnosis is not difficult ; the respiratory embarrassment, convulsions, the feeble pulse and the characteristic sound upon air entry into the veins are usually sufficient.

Treatment consists in immediately occluding the vein into which the air has entered. Stimulants should be administered hypodermically and nitrite of amyl inhaled. Venesection may be used to relieve the embarrassment of the heart.

8. **FAT EMBOLISM**—Fat may reach the blood vessels in cases of fracture of bones and in cases of hæmorrhage into or rupture of the liver. The fracture is usually situated in the long bones, generally in the tibia or femur, and occasionally in the ribs. Fat embolism may occur within a few hours of fracture of the bones.

The fat droplets first lodge in the capillaries of the lungs. Occasionally they are forced on through the lungs into the general circulation, and the glomeruli of the kidney may be plugged with fat cells, and they may also lodge in the brain or spinal cord.

The patient becomes cyanosed, and crepitations from œdema of the lungs may be heard at the bases. The temperature remains normal. Cerebral complications, such as delirium, coma and rarely localised paralysis, may be found. The urine should always be examined, as oil drops have been detected by staining with osmic acid, and also the retina, as in one case the fat drops were recognised in the retinal vessels before death.

When fat embolism occurs within a few hours of fracture of the bones, it has to be diagnosed from the general shock of the accident. It is doubtful whether fat embolism in the lungs can alone cause death; more probably this is due to the disturbance of the kidneys or lesions of the brain.

The indication for treatment is to sustain the heart. Nitrite of amyl is often useful, and inhalations of oxygen should be given to lessen cyanosis.

9. **PARADOXICAL EMBOLISM**.—In certain cases of venous thrombosis, emboli occur not only in the lungs but also in the systemic arteries. It has been shown that in these cases the embolus has passed from the right auricle to the left auricle through a patent foramen ovale. These crossed or paradoxical emboli are often preceded by pulmonary embolism, which causes a rise in pressure in the right auricle and a fall in the left auricle, so that the embolus can pass from the right to the left side of the heart.

MAURICE CAMPBELL.

ARTERIAL BLOOD PRESSURE

HYPERTENSION

In hypertension the systolic and diastolic blood pressure readings are persistently—not necessarily permanently—above certain levels, from whatever cause. Authorities differ somewhat as regards these levels, for the limits of normal variation are fairly wide, and, moreover, it is now practically universally believed that the blood pressure does not normally increase with advancing age as much as was previously thought to be the case; but it is generally agreed that persistent readings above 150 and 90, or at any rate 160 and 90, in adult males, and 170 and 100 in the elderly are pathological.

It is important to note that a transient increase in the blood pressure

readings, especially of the systolic, may occur with nervous excitement, such as is not infrequently incidental to a medical examination.

The late Sir Clifford Allbutt introduced the term *hyperpiesia* to denote a clinical condition in which there is a persistently raised blood pressure independent of renal disease. Some writers use the term *essential* or *primary* hypertension, or *hypertensive cardiovascular disease* (Janeway). The increased blood pressure is the essential or primary condition. Any changes which may be in the walls of the blood vessels, the heart, the kidneys or other organs are either a secondary result of the hypertension, or co-existent with it, and in the latter case may or may not be due to the same cause as is the hypertension.

Ætiology and Pathology.—The ætiology and pathology of hyperpiesia is obscure, and constitutes one of the most important and difficult questions in clinical medicine.

An inherited constitution is an important factor, as revealed by the high familial incidence in certain cases. The malady is more common in late middle life; in those who tend to worry, to be over-anxious, to take things too seriously, or are of an excitable disposition; in those whose manner of life involves continued mental or emotional stress or strain; and in the obese. It may be the result of poisons circulating in the blood, which act as pressor substances, such as occurs in endocrine disturbances, for example, at the female menopause, in tumours of the adrenal cortex or medulla, with Cushing's Basophilism Syndrome (pituitary basophilism), and in acromegaly; in focal sepsis; and in chronic alcoholic excess and excessive smoking. The general consensus of opinion is that a high protein diet intake in itself is not a cause, but in my view it is.

With regard to the pathogenesis of hyperpiesia, it is generally agreed that the immediate cause of the condition is an increased resistance to the passage of blood through the smaller arteries, especially the arterioles, at first due to hypertonus on their part. If the latter does not cease, either spontaneously or because of therapeutic measures, then, sooner or later, cardiac hypertrophy, especially of the left ventricle, and diffuse hyperplastic sclerosis supervene (see page 1035). The latter gives rise to actual narrowing of the lumen of the blood vessels, which may even be great, and a resultant further increase in the resistance to the passage of blood.

The cause of the initiation of the hypertonus of the arteries cannot yet be explained.

It is to be noted that some now consider that hyperpiesia is primarily of renal origin. Among the reasons adduced for this view are that some cases, in the course of time, develop into what is termed "malignant" hypertension. It may here be observed that this only occurs in a small proportion of cases. The matter will be dealt with further later on. Again, experimentally produced renal ischæmia in dogs, the result of the gradual narrowing of the renal arteries by means of clamps, will give a condition similar to hyperpiesia in man. With regard to this, it may be pointed out that pressor substances circulating in the blood from any source, including the kidneys, will have a like effect.

By far the general consensus of opinion—which I myself share—is that it is not possible to explain hyperpiesia as being due to structural changes in the kidneys, in which connection the following observations may be

made. (1) In hyperpiesia the blood pressure becomes normal under ether anæsthesia, and is diminished during sleep. (2) Hyperpiesia and diffuse hyperplastic sclerosis may occur independently of any renal lesion. (3) While in hyperpiesia and diffuse hyperplastic sclerosis the kidneys generally become involved secondarily, it may be in marked degree, with consequent increased obstruction to the circulation and an increase in the hypertension, in a large proportion of cases there is generally little or no impairment of renal function, which is one of the most characteristic features of hyperpiesia, and less than 10 per cent. die from renal failure. (4) There is frequently an absence of hypertension in polycystic and tuberculous disease of the kidney, occasionally in acute nephritis, and rarely in chronic nephritis. (5) In chronic nephritis changes may take place in the renal vessels of an almost exactly similar nature to those in diffuse hyperplastic sclerosis, leading to hypertension and similar cardiovascular accidents. But there the vascular changes are secondary to the nephritis.

Hypertension is not by any means only the result of kidney disease. There are many other causes. Indeed, it may even be asked whether in some cases of hypertension and diffuse hyperplastic sclerosis, and chronic nephritis, the former may have been the cause of the latter? Or, again, may both be the result of a common cause?

While the great majority of cases of hypertension are of the nature of hyperpiesia, the condition may also be due to diffuse hyperplastic sclerosis (see page 1035), chronic and acute nephritis, polycystic disease of the kidney, increased intracranial pressure, *e.g.* intracranial hæmorrhage, and polycythæmia vera. With regard to the first, however, the condition is rather the result than the cause.

Symptoms.—In hyperpiesia the patient is often well nourished and may be plethoric. There may be a complete absence of subjective symptoms for some years, and the condition may be discovered accidentally, *e.g.* during an examination for the purpose of life assurance.

Usually the onset of symptoms is insidious. The most common early symptoms are a feeling of fullness and it may be of throbbing in the head, transient giddiness, tinnitus aurium, flushing, insomnia, palpitation, languor and early fatigue, the latter especially on mental effort, headache, of a dull aching or it may be of a throbbing character, particularly in the occipital region and especially in the morning, impairment of memory, mental irritability and diminished emotional control. The blood pressure readings are above those mentioned in the first paragraph.

The condition may subside, either spontaneously or the result of therapeutic measures. If not, the blood pressure readings usually increase, and the systolic may reach 240 mm. or even more. Consciousness of the action of the heart may become more prominent. Sooner or later, there is evidence of hypertrophy of the heart, especially of the left ventricle, and of diffuse hyperplastic sclerosis (see page 994). The walls of the radial, the brachial and temporal arteries may be felt to be uniformly thickened—the so-called “whip-cord” artery. The degree of hardening of the arteries is found on palpation to vary at different times. The artery feels hardest when the vessel is most contracted and consequently smallest. The character of the pulse is that of high-tension. The retinal arteries are often thickened, and on ophthalmoscopic examination may show a glistening light along their

course—the so-called “silver wire” arteries, due to reflection of light from the thickened vessel. Even more important is a visible irregularity in calibre of the arteries best seen near the disc. Further, at the arterio-venous crossings there may be seen an obstruction to the flow of blood through it, leading to distension of the peripheral part of the vein and also to deviation of the vein where it crosses the artery.

The subsequent clinical picture and course exhibit considerable diversity.

The cardiac symptoms are by far the most common. The most frequent are shortness of breath, palpitation and precordial discomfort or pain, referred to exertion. Later, cardiac failure, usually left-sided but it may be involving both sides, may supervene (see page 857). Acute left-sided failure may occur (see page 857). Angina pectoris is not very infrequent. There is occasionally coronary occlusion. For further particulars, the reader is referred to page 857.

Various gastro-intestinal symptoms are often present. There may be hæmorrhages from the nose, the stomach, the intestines and other mucous membranes, the kidneys, the conjunctiva, and occasionally the retina, which are sometimes flame-shaped. There may be tingling, numbness and cramp of the limbs, and occasionally intermittent claudication. Hypertensive encephalopathy (see page 1607) may occur. Cerebral hæmorrhage may be a terminal event.

Not infrequently there is albuminuria, generally slight and not constant, and there may be polyuria and nocturnal micturition, and hyaline and granular casts. There is usually no or little impairment of renal function. Uræmia is rarely a terminal event.

There is a small group of cases of hypertension in which, instead of the symptoms being mild or moderate, the course slow, and little or no renal insufficiency, the symptoms are severe, the course is progressively rapid, and the patients die from renal failure. The former, which constitutes the great majority, is called benign and the latter “malignant” hypertension. There are also intermediate forms between the two groups. The renal changes in malignant hypertension are described as malignant nephrosclerosis, and those in benign hypertension may be termed benign nephrosclerosis. It may be remarked, however, that the term malignant is not altogether an appropriate one.

MALIGNANT HYPERTENSION.—There appear to be two forms of this, namely: (1) In a case of benign hypertension of a varying number of years' duration, the symptoms suddenly become severe and the malady pursues a rapid course. (2) In an individual who has not been aware that he had hypertension, from the outset there are severe symptoms in association with hypertension and the course is rapid. The second usually occurs at an earlier age than the first.

There are the same changes in the kidneys as in the benign form, and, in addition, those which are believed to be characteristic, *i.e.* acute necrosis of the walls of the arterioles and also often of the root of the glomeruli, and it may be resultant thrombosis of the glomeruli. While acute necrosis is most marked in the vessels of the kidneys it is also found in those of other organs. There has been much discussion among pathologists regarding the interpretation of the distinctive changes in the kidneys. In the opinion of some, they are an intensified result of the ætiological factor of the changes

in the walls of the arteries in the benign form ; while others think they are due to a special toxin. Again, some are of opinion that malignant hypertension is merely a severe or extreme phase and a terminal result of the benign variety, whereas others think that it is a separate entity.

In malignant hypertension, the blood pressure, especially the diastolic, is usually materially higher than in the case of the benign form, being generally over 200-120, and may be very high and may become extreme. There is lassitude, anorexia, frequently loss of weight, severe headache and anæmia. Changes in the retina occur earlier and are more marked, hæmorrhages being more evident and retinopathy is frequent if not usual. There is polyuria and nocturnal micturition. The urine is of low specific gravity, and contains hyaline and granular casts, a variable amount of albumin and often red blood cells. As the disease progresses, there is evidence of renal impairment. Death occurs from uræmia, cardiac causes or cerebral hæmorrhage.

In hyperpiesia it is probable that more than half the patients die from cardiac causes—in the great majority from congestive failure, not infrequently from angina pectoris, and occasionally from coronary occlusion ; in a fair proportion of cases, cerebral hæmorrhage is a terminal event ; in less than 10 per cent. uræmia ; while in the remainder the cause of death is some intercurrent disease.

Diagnosis.—Hypertension is recognised by persistent blood pressure readings above those mentioned in the first paragraph of this article.

The diagnosis between hyperpiesia and malignant hypertension may readily be made from a consideration of the respective clinical features described above. That between malignant hypertension and chronic glomerular nephritis may be difficult.

Prognosis.—Hyperpiesia usually pursues a slow course. It is probable that the duration of life after the beginning of symptoms is ten to fifteen years, or even more. The outlook is chiefly affected by the blood pressure readings, particularly the diastolic, the state of the heart and of the arteries, and the patient's manner of life. See also page 996. The duration of life in malignant hypertension is usually at the most two years.

Treatment.—As regards the treatment of hyperpiesia, the first indication is a thorough review of the ætiology. Then, the question as to whether a preliminary rest and, if so, how much is advisable should be considered (see page 862).

Hyperpiesia is an excellent example of the value of treating not only the disease but the patient. He should be reassured and encouraged. It is inadvisable to let him know the exact blood pressure readings at any time.

In no cardiovascular malady, excepting perhaps in angina pectoris, is the manner of life of so great consequence.

The cardinal indications are that, while the patient's activities should not be unnecessarily curtailed, they should come within the limits of his strength and even keep something in reserve ; there should be a sufficiency of rest in his life ; and all causes of mental and emotional stress should be carefully avoided. With this object in view, inquiry should be made regarding the patient's environment, the nature and hours of his work, his habits, the question of sleep and other relevant matters, while his temperament should be taken into consideration.

Each patient should be in bed for at least ten hours each night, rest physically and mentally for at least half an hour after the midday meal and have a quiet day each week. If the blood pressure is more pronounced, he should be in bed for at least twelve hours, rest for at least an hour after lunch, and stay in bed on one day a week with, it may be, a diet limited to vegetables, fruit and milk. In addition to an annual holiday of good length, several shorter ones during the year are advisable, in each case with a sufficient element of rest. As indicated, physical exertion and mental effort should be within the limits of the patient's strength (see page 863), with something in reserve. With this proviso, walking in the open air, riding a non-pulling horse, golf and mild bicycling are suitable forms of the former. The patient should be encouraged not to worry, or be over-anxious, or to take things too seriously; to avoid all excitement and other forms of emotional stress; and cultivate the art of "relaxing," physically and mentally.

If the patient tends to worry, or be over-anxious, or to take things too seriously, sedatives (see page 863) are often of special value. The matter of sleep is also of particular importance, and if there is insomnia, it should be treated on the lines laid down on page 863. Again, any obesity should, without fail, be corrected.

The amount of fluid with meals should be diminished, while an ample quantity should be taken between meals. Some prefer the mildly alkaline waters. The benefit of a regulated diet of moderate latitude may be considerable. Its amount should preferably be rather less than is needed. Those articles of food which especially stimulate the cardiovascular system should be reduced and proportionately to the degree of hypertension. The quantity of beef and mutton should be diminished, while twice-cooked meat, salted and preserved meat, liver, kidney, brains, sweetbreads, meat soup, and gravies, and meat extractives are better avoided altogether. Fish, poultry, game (not high), rabbit, vegetables, salads and fresh fruit are suitable. Ruthmol may be used as a substitute for table salt with meals. Moderation in the use of tobacco, tea and especially of coffee should be enjoined. Alcohol is better avoided altogether.

Strict attention to the condition of the bowels, preferably by a saline each morning, and a mercurial preparation at bedtime once or twice weekly are indicated.

Cold and hot baths are contra-indicated; but a warm bath, the temperature of which is gradually lowered, may be taken daily. A Turkish bath once or twice a week may be helpful. Patients often derive much benefit from a course of treatment at a Spa where different kinds of baths are used, on account of the rest, the change of air, the regular mode of life and exercise, the careful dieting and other factors, and in such cases periodic visits are to be recommended.

Iodine, in some form, in small doses appears to be occasionally of help. The same perhaps applies to the nitrites, such as a combination of sodium nitrite, erythrityl tetranitrate and mannityl hexanitrate, when symptoms are present. I have been disappointed with the results of the administration of mistletoe, liver extract, and potassium and sodium thiocyanate.

Diathermy, high frequency currents and other forms of physiotherapy are employed by some.

When the blood pressure is very much raised, particularly if there are

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any head symptoms, venesection may be of great benefit as a temporary measure, especially in plethoric patients. At least a pint of blood should be withdrawn. If required, this method of treatment may be repeated at intervals. It is also indicated when there are manifestations of great distension of the right side of the heart.

If the foregoing measures fail, a prolonged holiday, or complete rest in bed and a diet restricted to vegetables, fruit and milk for 2 to 4 weeks should be tried.

During the last few years a variety of surgical measures have been introduced in the treatment of hyperpiesia with the object of denervation of the kidneys, the suprarenal glands and the splanchnic area. It would appear that (a) the only procedures which afford promise of some success are bilateral section of the splanchnic nerves and decapsulation; (b) a very careful selection of cases is required; and (c) only those which are severe and unaccompanied by cardiac and renal failure should be considered. Surgical treatment of hyperpiesia is in its experimental stage but deserves further investigation.

For headache in hyperpiesia, cold compresses, or the application of menthol, or a cup of tea, or phenacetin and citrate of caffeine, failing which a tablet of trinitrin or one of the other nitrites may be used.

For the treatment of cardiac failure in hyperpiesia, see page 996. That of hypertensive encephalopathy is described on page 1608. For the treatment of renal failure, see pages 1327, 1328.

In conclusion, I would repeat that in the treatment of a case of hyperpiesia the patient's manner of life is of the greatest consequence and its importance can scarcely be exaggerated.

HYPOTENSION

In hypotension the systolic blood pressure is persistently—not necessarily permanently—below 110 mm. in adult males and 105 mm. in adult females, from whatever cause. Usually the diastolic pressure is proportionately less affected.

Ætiology and Pathology.—Hypotension may be physiological, that is, in some healthy individuals the blood pressure is persistently below the level mentioned.

The pathological form of hypotension may occur in a variety of conditions, such as: endocrine glandular disturbances, for example, Addison's disease; acute infective diseases, especially diphtheria; in pulmonary tuberculosis; in shock and collapse; in focal sepsis; in most cases of fatty degeneration and fatty infiltration, and in some of fibrosis of the myocardium; in some cases of chronic valvular disease; in coronary occlusion; in anæmia; and in cachexia and malnutrition.

The chief factors in the causation of hypotension are: diminished contractile force of the left ventricle; and, more frequently, vaso-dilatation, especially of the splanchnic area.

Symptoms.—Among these may be languor; early fatigue on physical and mental effort, and on maintaining the upright position; giddiness and faintness, especially on change of posture; a tendency to mental depression

and headache ; coldness and pallor or lividity of the extremities ; and an abnormal response of the cardiac rate with the assumption of the upright position after lying down. In splanchnic stasis, pressure on the abdomen by the hand while the patient is lying down is sometimes accompanied by distension of the jugular veins.

Treatment.—The ætiology should be reviewed. In acute cases, rest in bed for a time is advisable. In others, care should be taken to avoid over-exertion, physical or mental ; and it is an advantage to rest after effort, and before and after meals. A tepid or cold sponge bath in the morning, followed by simple exercises, may be helpful. An ample amount of nutritious and easily assimilable food, with an increase of those articles which stimulate the cardiovascular system, such as meat, and the periodic administration of general tonics are indicated. Physio-therapy, including massage, is useful in certain cases. Strychnine, citrate of caffeine, nikethamide (coramine), ephedrine, and pituitary (whole gland) may be tried. For Addison's disease, see page 507. Digitalis is of no avail. The wearing of an abdominal belt is very helpful in some cases.

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SECTION XIV

VASOMOTOR NEUROSES (ANGIO-NEUROSES)

INTRODUCTION

UNDER this heading are described several diseases in which vasomotor disturbance is the prominent symptom. Sensory, secretory and trophic disturbances may also be present. These diseases differ from each other both in regard to the nature and location of the vasomotor changes. Thus in Raynaud's disease there is a spasm of peripheral arteries. In erythromelalgia there is vaso-constrictor paralysis or excitation of the vaso-dilator nerves. In angio-neurotic œdema there is disturbance of capillary permeability, and perhaps of capillary tone. These diseases are described as vasomotor neuroses because a lesion of structure is not an essential part of their pathology, and because a considerable functional element is generally present. Thus they are common in persons who have an unstable nervous temperament, and emotional disturbance and fatigue play a not unimportant part in their ætiology. It has been thought in the past that these diseases were primarily due to disorder of the involuntary or vegetative nervous system. Lewis, however, has shown in the case of Raynaud's disease that a local fault of the vessels rather than a disordered vasomotor impulse determines the spasm of the digital vessels, and he finds the explanation of Raynaud's disease in terms of vasomotor dysfunction unconvincing. Too little is known of the ætiology of acroparæsthesia, erythromelalgia and Milroy's disease to throw light on this aspect but whatever the basic pathogeny of these conditions may be, vaso-dilatation is a prominent feature of erythromelalgia.

It is important that as far as possible a distinction should be drawn between these diseases occurring as neuroses and similar syndromes complicating recognised pathological states, such as lesions of the spinal cord or brain (tabes dorsalis or hemiplegia), lesions of peripheral nerves (peripheral neuritis), and lesions of vascular channels, or local pressure effects, such as may result from a cervical rib. But they are not separated by a rigid line from slighter manifestations of vasomotor instability, such as are frequent in women at the climacteric, and in clinical disorders resembling exophthalmic goitre. They are undoubtedly akin to such common symptoms as flushings, cerebral hyperæmia, facial congestion, angio-spasm in all its varieties, tachycardia (in some of its forms), anginal attacks, migraine, vertigo, tinnitus aurium, universal or circumscribed hyperidrosis, and gastric disorders of certain forms of functional gastric dyspepsia.

Lewis's studies on the local vascular reaction to irritation of the human skin have thrown much light on these diseases. He showed that there are three components in the reaction: (1) a primary dilatation of capillaries—the *red line*; (2) an increased permeability of these capillaries, producing the *wheel*,

which is independent of the nerve supply ; and (3) the *flare*, which depends on the integrity of the sensory nerve fibres in the neighbourhood. All these phenomena can be produced by an intradermal injection of histamine, and he attributed them to the liberation of this or some similar chemical substance to which he gave the name of "H-substance." It has been suggested that local liberation of histamine may play a part in producing the vesicles in herpes and the rash in erythema nodosum. It is of special interest that these reactions are partially dependent upon and partially independent of the nervous system. Some of the angio-neuroses are therefore so closely related to allergy that a general description of that condition is called for here.

ALLERGY

Idiosyncrasy has been defined as an unusual physiological personal equation, and allergy is a chemical idiosyncrasy, which expresses itself as an urgent attempt on the part of the cell to conserve its chemical identity. The tendency to allergy is inborn, whereas anaphylaxis is an acquired sensitivity. All the phenomena of allergy can be reproduced by histamine. The question is, how does this substance, which is toxic to everyone, become liberated in the tissues of certain people in answer to stimuli which are quite harmless to everyone else ? Normally there are two antagonists to histamine, adrenalin and the special ferment histaminase. It has therefore been suggested that the chemical basis of allergy is a congenital lack of histaminase, aggravated by an intermittent deficiency in adrenalin. The reaction is usually excited by foreign proteins, but a similar idiosyncrasy may be shown to various drugs.

The manifestations of allergy express themselves chiefly (a) in the *respiratory system*, by asthma, hay fever, paroxysmal rhinorrhœa, recurrent catarrhs ; (b) in the *skin*, as urticaria, purpura and eczema, particularly of the infantile type ; (c) in the *alimentary canal*, by diarrhœa and vomiting, or spastic colon ; and (d) by *effusion into joints*. There are many other conditions which with more or less show of reason might be added to this list. Certainly some cases of migraine seem to be of this order. It will be noted that several of these manifestations could be interpreted as a violent attempt to expel the invader, at any rate from the vital organs.

Most allergic manifestations are worse at night, because of the prominence of vagus control then, so that there is a relative insufficiency of the sympathetic nervous system, and therefore in the supply of adrenalin. The principal conditions produced by allergy are described under their appropriate headings.

ANGIO-NEUROTIC OEDEMA

Synonym.—Quincke's Disease.

Definition.—A paroxysmal affection, characterised by the appearance of circumscribed oedematous swellings of the skin and subcutaneous tissues of transient duration. The mucous membranes are often affected.

Ætiology.—Heredity is an important factor. Osler reported the case of a family in which five generations had been affected, involving 22

members. The condition is more common in men than in women, and generally affects the young. Those attacked are usually of a nervous disposition. Garrod reported a case in which each recurrence of periodic hydrarthrosis was attended by circumscribed œdema, either of the lips or eyelids. The attacks may coincide with menstruation. The exciting cause is generally difficult to determine. It may be emotional strain, exposure to cold, or trauma. Local trauma sometimes determines not only the onset but the site of an attack, as in a case recently reported in which riding provoked an attack on the inner aspect of the thighs and knees.

Pathology.—In the absence of a known pathology various theories have been advanced to explain the condition. Local venous spasm, a direct nervous influence on capillary walls, as a result of which the permeability of the vessels is increased, and, more recently, the local action of a circulating toxin on the capillary walls, are theories which have obtained support. With regard to the last named, Garrod drew attention to the joint swellings that frequently accompany erythematous and urticarial rashes resulting from known toxic causes. Such conditions form a part of the clinical picture of serum sickness, or may occur after taking certain articles of diet, or as the result of stings of insects or nettles. Lewis has shown that a modification of the same toxin may produce a dermolysin or a hæmolysin. In the former instance œdema; in the latter purpura results. Thus, *B. welchii* may produce either condition, depending on the intensity of the infection. This thesis illustrates the present view of angio-neurotic œdema as being a local expression of the presence of a circulating toxin, prone to occur in persons of nervous temperament, rather than a disease *sui generis*. The patients often show other signs of allergy, especially in their sensitiveness to foreign proteins.

Symptoms.—The complaint takes the form of acute circumscribed swellings of the skin and subcutaneous tissues, 1 to 4 inches in diameter. The swellings are rounded, painless, rarely itch, and are generally pale or sometimes redder than the surrounding skin, from which they stand out prominently. They may develop simultaneously in different parts of the body, and disappear in a short time. They may recur repeatedly, or only after a period of years; the recurrence is occasionally periodic. They occur most commonly in the eyelids, lips, cheeks and backs of the hands, and are asymmetrical. The whole side of the face, one side of the scrotum, the penis, a whole limb, or in fact any part of the skin, may be involved. The pharynx, tongue and conjunctivæ may be implicated. Œdema of the glottis is rare, and has proved fatal. Swelling of mucous membranes may lead to symptoms of gastro-intestinal disturbance, such as nausea, vomiting and colic. Cases in which hæmorrhage from mucous membranes, stomach, bronchi, bladder, etc., occurred have been reported. Hæmoglobinuria has been observed; in such a case a Wassermann test is indicated. The attacks are generally afebrile, and there is no constitutional disturbance, unless the stomach or intestine is involved.

Course.—This is variable. Recurrence is frequent, often at intervals of 3 to 4 weeks, but sometimes after long intervals. It is rarely periodic.

Diagnosis.—The complaint is so characteristic, in the sudden onset and rapid subsidence of asymmetrical rounded swellings, that it is hardly likely to be confused with other affections. The condition is nearly allied to

urticaria, from which it is distinguished by the circumscribed and deep-seated nature of the swellings and the absence of itching. No distinction is made between angio-neurotic œdema and giant urticaria.

Treatment.—The general health must receive first attention, and a saline purge is indicated. Both arsenic and quinine have been advocated. It is advisable to avoid any particular protein in the food which is found to excite attacks. When the attacks occur after a particular meal of the day, a capsule containing 1 to 2 grains of peptone half an hour before that meal appears to have an effect in temporarily desensitising the body against foreign protein. This, combined with 5 to 7½ minims of tincture of belladonna and 10 to 15 grains of calcium lactate after meals, has prevented recurrences in several cases. The former drug diminishes the vagal hypersensitiveness, and the latter increases the viscosity of the blood. One of the most useful forms of treatment for the relief of the paroxysm is a subcutaneous injection of 3 to 7 minims of liquor adrenalinæ hydrochloridi. This excites the antagonistic action of the sympathetic. For the same reason half a grain of ephedrine orally administered may be tried. When the tongue is involved the patient should be given one or two of Armour's suprarenal tablets to suck. Pituitary (posterior lobe) extract injections have also been recommended. Bromide is often helpful as an additional measure when the symptoms are marked. In severe cases the intravenous injection of small doses of peptone might be considered. Autohæmotherapy has proved decidedly useful in some cases. Vitamin K is also worthy of a trial.

Intermittent Hydrarthrosis, which is described under "Diseases of the Joints" (p. 1367), presents some interesting affinities with the vasomotor neuroses, particularly in its association with angio-neurotic œdema.

RAYNAUD'S DISEASE

Definition.—"Intermittent pallor or cyanosis of the extremities brought on by cold, with the skin a normal colour between attacks" constitutes Raynaud's phenomenon, which may occur however in conditions other than Raynaud's disease.

Ætiology.—The cause of this malady is unknown; it almost exclusively affects young women, and symptoms may begin any time between adolescence and middle age. The diagnosis of Raynaud's disease in a man is nearly always wrong. Jonathan Hutchinson preferred to speak of Raynaud's phenomenon, regarding it not as an entity but as a syndrome occurring in many different conditions. As he first suggested, a few cases are due to syphilis, congenital or acquired. It has occasionally been observed after acute infections.

Pathology.—Lewis has shown that there is an abnormality of the digital arteries, which expresses itself in a hypersensitiveness of these vessels to relatively low temperatures. It seems therefore that the fault lies primarily in the vessel wall rather than in the nerve supply to the muscle fibres. In advanced cases there is endarteritis, with partial occlusion of the lumen of the artery.

Symptoms.—The patients complain of attacks of pallor, blueness or numbness of the fingers, brought on by contact of the hands with anything

cold. Keeping the palms of the hands in cold water (15° C.) for 15 minutes in a cool room (18° C.) is usually sufficient to induce an attack. The body temperature is just as important, if not more so, than that of the hands in determining the onset of cyanosis. When such patients feel chilly in themselves their hands go blue on the slightest provocation, *e.g.* when sitting in a draught; when walking, cycling or driving a car in cold weather; or when swimming. When the body is really warm it is impossible to induce an attack no matter how cold the hands are. Emotion also may bring on an attack. In this connection it is interesting that an injection of adrenaline may have a similar effect. The attacks vary from slight pallor of one finger-tip to cyanosis and numbness of all the fingers of both hands. The cyanosis begins as a light-bluish tint, and later becomes a deeper blue. It always starts at the finger-tips, and spreads proximally to the base of the fingers and perhaps to the palm; rarely, if ever, does it reach above the wrist. If the attack persists for long, a secondary waxy pallor replaces the cyanosis. The hands remain blue or pale until they are warmed. They feel cold to the observer's touch. When the hands are warmed (40° C. for 3 minutes), or when the body temperature rises, the blueness begins to pass off, and irregular red blotches appear in its midst "like the spots on a plaiice." Some of these appear and fade away, but in the end they coalesce until the dorsum of the hand and the palm are fiery-red or scarlet. Gradually this redness spreads up each finger from base to tip.

Throughout the period of cyanosis the patients complain of a "tingling" or of a "feeling of numbness" in the fingers; some of an "uncomfortable sensation"; some of a "slight pain"; severe pain is unusual. When warmth is applied in any form—hot air in front of a fire, or by friction—the fingers quickly recover their normal colour. During this stage there are paræsthesiæ, "pins and needles," etc. In only a few patients is sweating of the hands a marked feature during attacks. Swelling of the fingers is rare. In a severe attack local pressure on a finger leaves an indentation which takes longer to disappear than when the circulation is normal. While the hands remain cold the radial pulses are of smaller volume and the veins less prominent than when warm. If a finger is accidentally cut when cold it does not bleed; "only a little dark blood oozes out." If symptoms occur in the feet they are usually less severe than in the hands. Nutritional changes at the extremities are rare in true Raynaud's disease. But in the later stages, when secondary arterial changes in the arterial walls have occurred, small areas of superficial necrosis at the finger-tips may be found, which leave small depressed tender scars.

Diagnosis.—This has been considerably clarified by John Hunt. The first question to be answered is: Is the complaint "Raynaud's phenomenon"? *i.e.* Is there *intermittent* cyanosis of the extremities, brought on by cold, with the skin a normal colour between attacks? The following conditions with their vascular phenomena are eliminated by this simple definition, many of them because their symptoms are continuous and not intermittent: chilblains; frost-bite; acrocyanosis; erythrocyanosis; clubbed fingers and cyanosis of the fingers and toes due to lesions of the heart and lungs; enterogenous cyanosis; incipient gangrene from arterial thrombosis (in advanced arteriosclerosis, thrombo-angitis obliterans, or ergotism); arterial embolism; diseases of the nervous system

(neuritis, poliomyelitis, syringomyelia, pyramidal lesions, and hysterical paralysis), cervical rib. If true Raynaud's phenomenon is present then the other conditions in which this phenomenon occurs must be considered :

(1) *Hereditary cold fingers*.—Many healthy young people find that their fingers go white and numb on exposure to cold. This is the commonest cause of Raynaud's phenomenon, and is local syncope in its simplest form. The onset is usually during childhood up to about the eighth year. Both sexes are affected, as are often members of the same family. These three points clearly differentiate the condition from Raynaud's disease.

(2) *After local injury or infection of the hands or feet, and in workmen using vibrating instruments*—pneumatic chisels, hammers, riveters, road drills, etc.

(3) *Scleroderma*.—This is diagnosed from Raynaud's disease by the following points : The fingers soon become stiff, and the stretched shiny skin cannot be picked up from the underlying tissues. Nutritional changes in the finger-tips are frequent. It is not confined to the extremities, and is much more rapidly progressive, painful and depressing than Raynaud's disease.

(4) *Syphilitic Arteritis*.—This may be diagnosed when Raynaud's phenomenon is associated with severe necrosis of the nose and ears, and when hæmoglobinuria is present. A blood Wassermann may be positive.

(5) *Other rare causes of Raynaud's phenomenon*.—Erythræmia is one.

Treatment.—Cold in any form should be avoided. The temperature of the body as a whole is almost more important than that of the hands and feet, and warm clothing is essential. The temperature of a living-room should be about 20° C. (68° F.). Cold water should be banned, and gardening in cold weather left to others. Gloves should be loose and long, overlapping the coat-sleeves. For people who work with their fingers, mittens are invaluable. A muff, a small hot-water bottle, or an electric heater in a handbag or pocket may be recommended. Cracks at the ends of the fingers may be covered with a collodion preparation, or with narrow strips of elastic adhesive plaster. The skin of the fingers may be kept soft by applying liquid paraffin. Care should be taken to avoid minor injuries to the fingers, and when these occur they should be treated at once. Boots or shoes should be of stout construction, allowing plenty of room for the toes, and stockings or socks should be thick and soft ; two thin socks often keep a foot warmer than one thick one. Tight suspenders should be avoided. Bedsocks and hot-water bottles are helpful at night.

The number of drugs that have been recommended to relieve symptoms indicates how few are really valuable. Thyroid, calcium lactate or gluconate, potassium iodide, and belladonna are perhaps the favourites. In some patients physical therapy is needed to give relief : postural exercises ; radiant heat, taking special care to avoid burns ; contrast baths ; intermittent venous occlusion by alternating positive and negative pressures ; and galvanic baths. Sympathectomy is indicated when the attacks of cyanosis are causing definite distress or recur so frequently as to interfere with work, and when temperature tests suggest that there is a considerable degree of vasomotor tone. The immediate results of the operation are excellent ; but after some months, perhaps two years or more, slight symptoms may return. In spite of this disappointment the great majority

of patients insist, even several years after the operation, that their hands are better than they were before it. The relief of major symptoms is due to removing from the vessels the burden of their normal vasomotor tone. The local fault in the vessels remains untouched, for on this the operation has no effect.

ACROPARÆSTHESIA

Definition.—A vasomotor neurosis, characterised by paræsthesiæ of the hands, especially affecting the finger-tips.

Ætiology.—The condition is usually observed in women, especially at the climacteric. It rarely occurs before the age of 30. It is frequently associated with a neuropathic diathesis and a lowered vitality due to any cause. General causes include inanition, anæmia and pregnancy. Local causes are exposure to cold, particularly cold water, or to alternate hot and cold water as experienced by washerwomen.

Symptoms.—The onset is insidious and the symptoms are almost entirely subjective. The affection is often limited to one hand or certain fingers, the toes rarely being affected. The patient complains of numbness, tingling, formication of the fingers or tenderness of the finger-tips. There may be slight loss of sensibility in the finger-tips and occasionally evident pallor.

Diagnosis.—The condition is readily distinguished from Raynaud's disease by the absence of local asphyxia. It is important to exclude any affections of the spinal cord, such as tabes dorsalis.

Prognosis.—The complaint tends to be continuous and persistent. The outlook regarding recovery is not good, unless the condition is due to a recognisable and removable cause. There are, however, no complications.

Treatment.—This is directed to the removal of the cause, and improvement of the general health and of the local circulation. Sodium salicylate and bromides are often helpful, and radiant heat and massage are of value.

ERYTHROMELALGIA

Definition.—A rare condition characterised by pain, redness and swelling of the toes and feet, and less often of the hands.

Ætiology.—Little is known of the ætiology of the disease. Men are more often affected than women. The condition may occur in the course of a disease of the central nervous system, such as hemiplegia, disease of the cauda equina, and disseminate sclerosis. The swelling and pain are aggravated by standing and by warmth.

Pathology.—Disease of the peripheral arteries—a chronic endarteritis—has been described in three cases by Batty Shaw. Changes in the peripheral nerves have been held responsible, and Weir Mitchell found marked degeneration of the fine nerve branches in one case. Others regard the malady as an angio-neurosis, allied to acroparæsthesia and Raynaud's disease. Lewis prefers to regard erythromelalgia as a special instance of a condition he designates as *erythræmia*, which he maintains has nothing to do with abnormality of the vasomotor system, but always results from a local process.

There is a release into the skin of an unknown substance not histamine, which lowers the threshold of the pain nerve endings.

Symptoms.—The first case was described by Weir Mitchell, and was that of a sailor, aged 40, whose first complaint, following an African fever, was of "dull, heavy pains at first in the left, and soon after in the right foot. There was no swelling at first. When at rest he was comfortable, and the feet were not painful; after walking the feet were swollen. They scarcely pitted on pressure, but were purple with congestion; the veins were everywhere singularly enlarged, and the arteries were throbbing visibly. The whole foot was said to be aching and burning, but above the ankle there was neither swelling, pain . . . nor flushing."

Pain is generally the first symptom, soon followed by redness and swelling, most marked in the terminal phalanges of the toes or fingers. The pain is generally severe; at first it occurs only in the evening, but later it becomes chronic or remittent and may be agonising. The redness may increase to cyanosis. The swelling is more marked in the latter part of the day, and is aggravated by standing, walking, dependence of the limb and by heat. These symptoms are relieved by cold and recumbency. Hyperidrosis of the affected part is not uncommon. The condition may be complicated by general weakness, vertigo, headache, palpitations and tachycardia. Its complication with erythraemia has been described. Pellagra has been mistaken for erythromelalgia.

Prognosis.—The complaint is intractable, and tends to persist, with exacerbations and remissions, for many years.

Treatment.—The affected part should be elevated and immobilised. Faradism and cold have been recommended. Analgesics are required for the relief of pain, which may even necessitate amputation (Shaw). Sympathectomy is contra-indicated and should never be performed for this condition.

ERYTHROCYANOSIS

This condition occurs, as its full name, Erythrocyanosis Crurum Puellarum Frigida, implies, chiefly in the legs of young women. The feet and legs are cold, and the back of the leg and ankle is swollen and blue, especially at the insertion of the tendo Achillis. Chilblains are often present, and ulceration may occur where the swelling is most severe. The condition is due to vascular spasm, in which the modern fashion of inadequately protected extremities is presumably a factor. Treatment includes warmer stockings, with silk ones outside to gratify the usual desire for appearance, and the loosening of all tight bands above and below the knee. Exercises, such as walking and skipping, with local massage should be ordered, and elevation of the heel may give some relief. Calcium gluconate, vitamin D and small doses of thyroid have been advocated, and daily subcutaneous injections of acetyl choline bromide, beginning with $\frac{1}{4}$ c.c. and gradually increased to 1 c.c., have given good results. After a fortnight the injections may be administered less frequently, and at the end of six weeks discontinued. Bilateral lumbar ganglionectomy has been recommended, but should not be considered unless thorough medical treatment fails. It may improve the colour of the skin and promote healing of any ulcers, but it does not diminish the swelling.

CAUSALGIA

This is a form of neuralgia usually following injuries in the neighbourhood of certain nerves, particularly the median and sciatic. It is probably due to irritation of the peri-arterial sympathetic fibres. On account of the burning character of the pain, Stopford has suggested the name *thermalgia*. The malady is fully described under lesions of the median nerve.

MILROY'S DISEASE

Synonym.—Hereditary Œdema.

Definition.—In 1892, Milroy described a persistent œdema of the legs, occurring in the absence of any of the known causes of œdema, affecting members of the same family in successive generations.

Ætiology.—The disease has occurred in six generations of the same family, but the percentage of incidence in the families has varied greatly. It is apt to appear in neurotic families. Both sexes are affected about equally, and the œdema may either appear soon after birth, or its onset may be delayed till puberty or even till adult life. Thirty-five years after his original description, Milroy found that the disease was tending to die out in the family in which he first observed it.

Pathology.—Nothing is known of the pathology of the condition. There is no evidence of venous or lymphatic obstruction.

Symptoms.—Only the legs are affected, and these to a variable extent. Thus the swelling may be limited to the ankles; it usually does not extend beyond the knees, but may reach the thighs in long-standing cases. It never extends above Poupart's ligament. Gradually the affected part becomes hard and brawny. The swelling increases in the standing posture, and, once established, it is permanent. There is no pain or redness, the veins are not enlarged, and the general health is not affected.

In some cases there are acute attacks accompanied by fever and pain. During this phase the condition resembles erythromelalgia.

Diagnosis.—This is made on the familial incidence, and the absence of all other recognised causes of œdema. A group of cases in which there is swelling of the feet, ankles and legs without albuminuria or discoverable organic disease to account for the swelling, has recently been recognised by Osman. The patients the writers have seen have been women in the third decade. The swelling is pale, brawny, and does not pit on pressure. This type may respond to rest in bed and intensive alkali therapy.

Prognosis.—The affection does not tend to shorten life.

Treatment.—The affected parts should always be kept bandaged with crepe, as by this means the swelling can be kept under control, and the patient remains able to lead an active life; but if such measures be not employed the œdema gradually extends. Acute attacks may require opium internally, and evaporating lotions locally.

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GEOFFREY EVANS.

SECTION XV

DISEASES OF THE RESPIRATORY SYSTEM

THE PHYSICAL SIGNS IN THE CHEST IN HEALTH AND DISEASE

ACCURATE diagnosis in diseases of the air-passages and lungs depends largely upon careful observation and record of physical signs, especially in their relation to subjective symptoms. Unfortunately, there is no strict uniformity in regard to the nomenclature of physical signs. It is, therefore, desirable to define explicitly the sense in which the various technical terms used in this section are employed. At the outset, it is well to emphasise the importance of a careful and methodical examination in every case. The magistral sequence of inspection, palpation, percussion, auscultation and mensuration has more than the sanction of tradition to commend it. Unless confined to bed, the patient should be examined both in the erect and recumbent positions in all cases of difficulty.

INSPECTION.—The patient being placed in a good light, the configuration of the chest, the range and character of the respiratory movements and the position of the cardiac pulsations should be carefully noted. Most of the terms used in this connection, such as flattening, retraction, recession of intercostal spaces and diminished movement are self-explanatory.

The respiration may be unduly slow (*bradypnoea*), rapid (*tachypnoea*) or distressed (*dyspnoea*). *Dyspnoea* may be inspiratory, expiratory or spasmodic. A peculiar periodic disturbance of the respiratory rhythm is that referred to as *Cheyne-Stokes breathing*. In this condition, the respiratory movements wax and wane in short periods of *dyspnoea*, each followed by an interval of *apnoea* or cessation of respiration lasting up to 30 or 40 seconds. It is due to deficient aeration of the blood and is met with in respiratory, cardiac and renal disease, and also in cerebral lesions and after some poisons.

A variety of grouped breathing is *Biot's breathing*, sometimes seen in tuberculous meningitis. The hyperpnoeic period consists of a few breaths, deep or of increasing depth, followed by *apnoea* without the waning.

Certain abnormal forms of chest configuration are described: The *alar*, *phthinoid* or *pterygoid* type of chest is long, narrow and flat, with winging of the scapulæ; the subcostal angle is narrow and the upper interspaces are wide. The *emphysematous* or barrel-shaped chest is broad and rounded, the angle of Louis is prominent, the subcostal angle is wide, and the movements are restricted. The *pigeon breast* is characterised by prominence of the sternum, with sloping anterior thoracic walls. The *funnel breast* is

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the converse of this, with depression of the lower end of the sternum and of the cartilages attached to it.

PALPATION.—Vocal fremitus or tactile fremitus is the vibration felt over the lung when the hand is placed flat upon the chest-wall without pressure and the patient says “*ninety-nine*” or some other resonant syllables. Vocal fremitus may be increased, decreased or absent in disease.

Tussive fremitus.—The similar vibration felt during cough.

Rhonchal fremitus.—The vibrations communicated to the chest-wall by sonorous rhonchi in bronchitis, and felt by the hand.

Friction fremitus or pleural fremitus.—A rubbing sensation communicated to the palpating hand in certain cases of dry pleurisy. A similar fremitus is occasionally felt in pericarditis.

PERCUSSION.—This consists in tapping the chest-wall over the lung and observing the note produced and the sense of resistance felt. Percussion may be *direct* on to the chest-wall or *mediate*, when the tap is made on to a finger or an instrument placed on the chest. The applied finger or instrument is called the pleximeter, the tapping finger or instrument, the plessor. Percussion should always be light, except over very muscular parts of the chest.

Normal resonance is the note obtained over healthy lung tissue.

Hyper-resonance is an increased resonance, with diminished sense of resistance obtained over emphysematous lung tissue.

Dullness is diminution or loss of resonance, with increase in the sense of resistance. Various degrees of dullness are described, such as impaired percussion, slight dullness, flat, wooden or stony note.

Tympanitic resonance—a hollow drum-like note.

Skodaic resonance—a clear, high-pitched note intermediate in character between the hyper-resonant and tympanitic notes.

Cracked-pot sound—or *bruit de pot fêlé*—a hollow note with a slight jingle added to it, obtained by smart percussion over a fair-sized cavity. It is also heard on percussion of a crying baby.

AUSCULTATION.—The breath sounds should be listened to first, then the adventitious or added sounds, and lastly the vocal resonance.

(a) **BREATH SOUNDS.**—The following varieties of breath sounds may be differentiated:

Vesicular breathing.—The normal respiratory murmur or faint rustling sound audible during inspiration and expiration, the former phase being two or three times as long as the latter. The pause between inspiration and expiration is short.

Cog-wheel, jerky or interrupted breathing is a form of vesicular breathing in which inspiration waxes and wanes, or is divided into two or more parts.

Harsh, exaggerated or puerile breathing.—An intense form of vesicular breathing heard in children and in some forms of emphysema.

Vesicular breathing with prolonged expiration.—There is no alteration in the intensity or pitch of inspiration, but expiration is more prolonged and often harsher.

Absent, diminished, weak and suppressed breathing are self-explanatory.

Bronchial breathing.—The pitch of both inspiration and expiration is raised. Expiration is as long as inspiration and is separated from it by a distinct pause.

Broncho-vesicular and vesiculo-bronchial breathing are incomplete forms of bronchial breathing in which inspiration or expiration respectively assume the bronchial type.

Tubular breathing is a peculiar form of high-pitched bronchial breathing of whiffing character, sounding as if produced close under the stethoscope. This term is often used as if synonymous with bronchial breathing, but should be restricted to breathing of the type just described, which is only heard in consolidation from lobar pneumonia and broncho-pneumonia and in collapse of the lung.

Cavernous breathing is bronchial in type, but both inspiration and expiration have a peculiar hollow character. Expiration is more hollow and more prolonged than inspiration.

Broncho-cavernous breathing is incomplete cavernous breathing, inspiration being bronchial, while expiration is cavernous.

Amphoric breathing.—An intense form of cavernous breathing, often having a very hollow metallic sound.

(b) **ADVENTITIOUS SOUNDS.**—These were formerly divided into dry and moist. The former are now called rhonchi, the latter râles.

Rhonchi are musical sounds produced by the passage of air over mucus or muco-pus in the bronchi. Those arising in the larger tubes are called sonorous rhonchi, those in the smaller tubes sibilant or whistling rhonchi.

Râles are bubbling or crackling sounds produced in the bronchi or alveoli by the passage of air through fluid exudate or secretion. They are usually divided into bubbling and crackling râles. Bubbling râles are heard when the lung tissue is still spongy. Crackling or crepitant râles are produced in consolidated or softening areas of lung. Both varieties are arbitrarily subdivided into fine, medium and coarse râles. Crepitant râles are sometimes referred to as “creps”; this practice may lead to confusion with crepitation and is better avoided. Crepitant râles were formerly called consonating, bubbling râles non-consonating. Gurgling râles are coarse, low-pitched râles, usually heard over a cavity, especially after a cough.

Crepitations are fine “hair-like” crackling sounds. They may be produced either in the pleura or in the lung. In the latter they occur only in pneumonia, broncho-pneumonia, collapse and œdema. They are heard chiefly with inspiration and may be increased in number and intensity by coughing. A coarse variety heard in resolving pneumonic lung is called *redux crepitation*.

Pleural crepitations are fine sounds of similar character occurring in the early or dry stage of pleurisy. They are heard rather towards the end of inspiration and are usually unaffected by cough.

Friction is a coarse rubbing, creaking or grating sound heard in pleurisy when there is rough exudate on the pleural surfaces. It may occur with either inspiration or expiration or with both.

Stridor is a loud, coarse sound, heard chiefly during inspiration in cases of obstruction of the larynx, trachea or main bronchi. It is louder and lower pitched than a rhonchus.

Post-tussive suction is a hissing sound, audible directly after cough. It is heard only over a cavity, and is caused by the influx of air to replace that expelled by cough.

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Veiled puff of Skoda is a puffing sound heard towards the end of inspiration. It is said to indicate small and sacculated bronchiectatic dilatations.

Metallic tinkling and amphoric echo are terms used to describe the quality of certain sounds produced near a very large cavity or a pneumothorax, in breathing or coughing, or by the heart's action.

Succussion splash is a splashing sound produced in a hydro- or pyopneumothorax by shaking the patient, or getting him to shake his thorax. If a gastric splash can be excluded, it is pathognomonic of a pleural or subphrenic hydro- or pyopneumo-thorax.

Bell sound or *bruit d'airain*.—A ringing sound heard on auscultation over a pneumothorax or any large cavity when a coin placed flat on the chest-wall over the air-containing space is tapped by a second coin. A similar sound is often audible on flicking with the finger and thumb over the chest-wall under similar conditions.

VOICE SOUNDS or Voice Conduction.

Vocal resonance is the muffled sound on listening over normal lung when the patient articulates "ninety-nine" or some other resonant syllables.

Bronchophony is an increase in the intensity of the normal vocal resonance.

Pectoriloquy is conduction of the articulate voice sounds which are clearly heard as if spoken into the stethoscope. It is best appreciated by auscultating the whispered voice, and is then called whispering pectoriloquy.

Ægophony denotes a peculiar bleating or nasal modification of the voice sounds, sometimes heard on listening to them through fluid in the pleural cavity.

Physical examination of the chest includes mensuration, estimation of vital capacity and examination by the X-Rays when these are necessary.

Vital capacity is determined by a spirometer, which measures the amount of air which can be expired by a full expiration after the deepest possible inspiration. The average for an adult man is about 3600 c.c. The vital capacity is diminished in many diseases of the respiratory system, notably in acute pneumonia, pulmonary tuberculosis and in attacks of asthma.

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DISEASES OF THE NOSE

ACUTE CATARRHAL RHINITIS

See The Common Cold, p. 133

CHRONIC CATARRHAL RHINITIS

Ætiology.—Simple chronic rhinitis appears to result from frequently occurring attacks of coryza, or even from the persistence of a single acute attack. Predisposing causes of this persistence are deficient resistance, local irritation, auto-intoxication from the gastro-intestinal tract, and reflex vasomotor disturbance. Thus we find chronic catarrh associated with

anæmia, stuffy and over-heated rooms, occupations involving inhalation of irritating dust or vapour, excessive smoking, snuff-taking (a commoner habit than is generally realised among shop-assistants and clerks who are prevented from smoking during working hours), dyspepsia and constipation, alcoholism, sexual excess and masturbation. Nasal obstruction is an important factor in keeping up chronic catarrh by preventing ventilation of the passages, allowing mucus to collect and encouraging the growth of micro-organisms. In children the presence of adenoids is the commonest cause of catarrh.

Symptoms.—The symptoms are nasal obstruction and excessive secretion, which may be watery or muco-purulent, and may come forwards to the nostrils or pass backwards into the throat ("post-nasal catarrh"). Secondary results, from the extension of the inflammation, include catarrhal and suppurative otitis media, pharyngitis, laryngitis and bronchitis.

The nasal mucosa may be reddened, but often has a pale, sodden appearance. The turbinals are swollen and are at first quite soft, but later, when definite thickening has occurred, they feel firmer, and no longer shrink after the application of cocaine or adrenaline. At this stage the condition may be called "hypertrophic rhinitis," and the mucosal thickening, most marked over the two ends of the inferior turbinals and over the lower margin of the middle turbinal, may form large lobulated masses.

Diagnosis.—The diagnosis, in cases of hypersecretion, can only be made after excluding by rhinoscopic examination all other causes, such as a foreign body, mucous polypus, syphilitic, tuberculous and lupoid ulceration, and, more especially, suppuration in any of the accessory sinuses; in the latter, the discharge is not scattered over the nasal passages, but emerges in a localised stream from one or other of the ostia and reappears in the same situation after being removed. The discharge of chronic rhinitis is bilateral, and, though often muco-purulent, is never true pus. The diagnosis from vasomotor rhinorrhœa is often difficult; the symptoms of the latter come and go with great suddenness, often as a result of definite causes such as going into a hot room, and there is complete absence of symptoms between the attacks. A bacteriological examination is often helpful.

Treatment.—General treatment, directed to the predisposing causes mentioned above, is necessary if a good result is to be obtained. Next, nasal obstruction must be removed, and operative treatment is called for if the cause be adenoids, deviation of the septum, or great hypertrophy of the extremities of the inferior turbinal bodies. For details, the reader is referred to surgical works; but it must be emphasised that the valuable secreting surface of the nasal mucosa must not be recklessly sacrificed, and that large portions of inferior turbinal must on no account be removed, nor should every slight deviation of the septum be submitted to operation, for it is rarely quite straight. When the turbinal enlargement is soft and shrinks after the applications of cocaine, the galvano-cautery should be used under local anæsthesia to draw one or two lines along the length of the inferior turbinal, the result of which is to produce a scar binding the mucosa to the bone. When the case has not gone on to definite hypertrophy, the most valuable form of local treatment is cleansing of the nasal passages. The lotion must be warm, about 90° F., and quite unirritating; for this reason it should have approximately the same specific gravity as serum, and

normal saline solution does very well. A mildly antiseptic and alkaline lotion is usually preferred, of which Dobell's solution is the type, such as sodium bicarbonate, grs. 3; sodium chloride, grs. 3; phenol, gr. 1; glycerin, min. 45; water, fl. oz. 1. A convenient instrument for the purpose is a small rubber ball of a capacity of 2 oz. with a blunt nozzle moulded in one piece, which is slowly emptied into the nostril, while the patient breathes deeply through the mouth and inclines the head forward over a basin; in this way the palate is raised and the lotion passes through the naso-pharynx and out by the other nostril. The nose must not be violently blown afterwards, nor must any force be used during syringing, or fluid may be injected into the Eustachian tubes. Occasionally syringing causes headache, in which case the lotion may be used in a coarse spray-producer. Chronic nasal catarrh is, however, in certain cases notoriously resistant to treatment, especially under the conditions of civilised town life; indeed, many sufferers found themselves better in the wet and exposed conditions of life in the trenches during the War of 1914-1918. Vaccine therapy is uncertain in its effect, but gives good results in a proportion of cases; an autogenous vaccine should be prepared from the patient's nasal secretion.

ATROPHIC RHINITIS

Synonym.—Ozoena.

Ætiology.—Cases usually first come under treatment between the ages of 15 and 18, but its insidious beginnings date from an earlier age, and a history of nasal discharge through childhood is often obtainable. It affects females at least three times as often as males. A peculiar physiognomy is to be noticed in nearly half the cases; the skull is brachycephalic, the nose wide and flat, and the nostrils broad and so directed forwards as to be more than usually conspicuous. The affection is sometimes unilateral, in which case the septum is deflected and the disease occurs on the wider side. Occasionally it is found among several members of a family, which might be the result of contagion, but it is also inherited in circumstances where contagion cannot apply, and this may be due to inheritance of the disease or merely of the predisposing physiognomy. It is more often seen in the poorer than in the more well-to-do classes and, in England, it has become much rarer during the last twenty or thirty years. Of the many bacteria found in association with the affection, the most important are the Klebs-Loeffler bacillus, the *Cocco-bacillus fatidus* of Perez, and the *Cocco-bacillus mucosus* of Beeritz, but the consensus of opinion is that they are secondary, and, though helping to produce the fœtor, are not the primary cause of the disease. The condition occurs at too early an age to be the final stage of hypertrophic rhinitis, nor is it usually due to accessory-sinus disease, which can be excluded in the majority of cases. It is probably the sequel of prolonged purulent rhinitis in childhood, which results in the replacement of the ciliated by squamous epithelium, and thus destroys the principal agent for the removal of secretion; the undue width of the nasal passages promotes this retention by diminishing the force of the expulsive current of air, by drying the secretions and by unduly admitting dust and micro-organisms. In this way crusts of dried mucus are formed and decompose, and the resulting inflammation prevents the

development of the turbinals and thus further increases the width of the nasal fossæ.

Pathology.—There is a chronic inflammation resulting in sclerosis and atrophy of the mucosa; the ciliated epithelium is replaced by squamous, the mucous glands are degenerated and the venous sinuses have disappeared. These changes are most marked over the inferior turbinals; the middle turbinals are frequently large and cedematous. The discharge is not pus, but mucus precipitated by evaporation, mixed with shed epithelial cells and teeming with micro-organisms. This collects and dries into large greenish-black crusts which give rise to the peculiar sweetish and horribly offensive odour. There is never true ulceration nor necrosis of bone.

Symptoms.—The symptoms are chiefly those of the nasal discharge and the offensive stench; the latter is rarely perceptible to the patient, who usually, in established cases, has complete anosmia. There are also obstruction from the crusts, dryness of the throat and cough, and often some degree of ill-health from toxic absorption.

The inside of the nose is full of crusts; the inferior turbinals are reduced to mere ridges, the mucosa is pale and thin, and through the widened nasal passages the body of the sphenoid and the wall of the pharynx are plainly visible.

Complications.—Infection of an accessory sinus may result by extension from the septic nasal cavities, but is not very common; conversely suppuration of the sphenoidal or a posterior ethmoidal cell is a possible, and has been considered by some authorities as the usual, cause of the affection. The lymphoid tissue of the throat is conspicuously absent; there is often a dry pharyngitis and laryngitis, and sometimes the crusting extends to these parts, or even down the trachea. Catarrhal and suppurative otitis are common, and the disease is thought by many to predispose to pulmonary phthisis.

Prognosis.—As the ciliated epithelium can never be replaced, the affection is not truly curable, though regular treatment can keep it in an inoffensive condition, and it is common to see a suggestive degree of atrophy of the turbinals in young people with rhinitis completely disappear under treatment. Also, the crusting tends to become less troublesome as time goes on and ultimately to cease, a state of things which is difficult of explanation.

Treatment.—The nose must be kept clean by regular syringing with a mild alkaline antiseptic lotion, of which a large quantity should be used with a Higginson syringe provided with a fine nozzle which cannot block the nares. An oily, stimulating or emollient application, such as oleum eucalypti 15 minims to 1 ounce of paraffinum liquidum may be applied as a paint or spray, or a 25 per cent. solution of dextrose in glycerin as a paint. The crusting can be prevented by excluding the air and, when syringing is insufficient, this should be done by introducing a plug of gauze or cotton wool loosely into the anterior nares, which should be changed twice a day by the patient. After some weeks of treatment the packing may be omitted, but resumed in the event of a relapse. When the surgeon removes the plug the discharge is seen to be a clear mucus; if pus be found, it must be traced to its source in an accessory sinus. Paraffin wax may be injected under the mucosa, to narrow the nasal passages, but it is liable to slough out, and a piece of costal cartilage has been implanted with the same object. A plastic operation

has been devised to shift the antro-nasal wall inwards and has given encouraging results. The treatment of anæmia is important; good food and an open-air life, especially at the seaside, are beneficial.

EPISTAXIS

Ætiology.—The causes of epistaxis may be classified as follows:

Local causes.—Traumatism, including blows on the nose, fracture of the base of the skull, surgical operations and foreign bodies; the small septal erosion of rhinitis sicca, which is the commonest of all causes; malignant disease; angio-fibroma, or “bleeding polypus,” of the septum; multiple telangiectasis, a curious hereditary affection characterised by numerous minute dilations of the capillaries on the face and mucous membranes of the nose, mouth and throat; the general congestion caused by adenoids; and syphilis, lupus and the rarer granulomata, though in these the bleeding is usually an insignificant symptom.

General causes.—High blood pressure, as in arterial disease, chronic nephritis, cirrhosis of the liver, violent exertion, extremes of heat and cold, congestion at the menstrual period or “vicarious menstruation”; venous congestion, as in mitral stenosis, tumours in the thorax or root of the neck, emphysema, bronchitis and whooping-cough; toxic blood conditions, as pernicious anæmia, leukæmia, purpura, scurvy, and all the acute infectious fevers, especially in the prodromal stage. To these may be added rarefaction of the air, as in aeroplane ascents and mountaineering, and poisoning by some drugs, especially salicylates and quinine.

The source of the bleeding is, in the large majority of cases, in a region called Little’s or Kiesselbach’s area, situated on the front and lower part of the septum just beyond the vestibule.

Treatment.—Epistaxis, of sufficient severity to call for the attention of the doctor, should always be treated, though it is of comparatively little importance in healthy young people; in older patients with high blood pressure the loss of blood may be beneficial, but the occurrence is so distressing and alarming to the patient that other means to lower the pressure should be adopted.

The source of the bleeding is usually so far forward that a pledget of wool introduced for less than an inch into the naris, and held by compressing the nostril, will generally control it temporarily. To arrest it and prevent recurrence the bleeding spot must be found, started if necessary with a probe, controlled by application of cocaine and adrenaline on a plug of wool, and sealed by the galvano-cautery at dull-red heat. The use of an emollient ointment during the separation of the scab is advisable. In obstinate cases the bleeding may recur from another spot or from the opposite naris, when the treatment must be repeated. As in other forms of hæmorrhage, a rapid excited heart’s action, associated with restlessness and fright, is often present, and an injection of morphine is of great value. Calcium lactate is often recommended and may be given in 20-grain doses three times a day for 2 or 3 days; or colloidal calcium may be injected subcutaneously. If the bleeding is from the usual situation, formal plugging of the nose is seldom called for; but sometimes the bleeding proceeds from farther back in the nose, or is so

profuse that its situation cannot at first be determined. In such cases the naris should be evenly packed with ribbon-gauze introduced on forceps under inspection. The older method of plugging the posterior nares is seldom required, and carries the risk of causing otitis media. Nasal plugs quickly become septic, and should ordinarily be removed in 24 hours; but they may be kept sweet for several days, should it be necessary to retain them, by moistening them frequently with peroxide of hydrogen.

MUCOUS POLYPUS

Ætiology and Pathology.—Nasal polypi are rare before puberty and are somewhat commoner in men than in women. They never grow from the septum, inferior meatus or inferior turbinal, but only from the ethmoidal region and interior of the accessory sinuses. They are not neoplasms, but are essentially due to a local œdema of the mucous membrane; the swelling thus produced is acted upon by the expulsive forces of the nose, and, being so pulled down and elongated, the return flow of its blood vessels and lymphatics is further impeded and a greater degree of œdema results. All stages of polypus formation may be found, ranging from an œdematous fringe along the border of the middle turbinal to enormous pedunculated masses which block the nose and expand its bony walls. In the majority of cases the œdema is due to inflammatory infiltration of the muco-periosteum of the ethmoidal labyrinth and is often associated with inflammation in the ethmoidal cells; sometimes, however, the cause is vasomotor disturbance, for polypi are found in cases of hay fever and paroxysmal rhinorrhœa in the absence of true inflammation. There is also another form, the so-called "choanal polypus," in which a large single polypus hangs into the nasopharynx from a long pedicle attached within the antrum and passing through the ostium into the nose.

Symptoms.—The cardinal symptoms are nasal obstruction and discharge, which is profuse and watery. The symptoms are worse in damp weather. Cough, headache and asthma are not infrequent, and a loss of the power of mental concentration often occurs.

Polypi are smooth, shiny, white, translucent bodies, pedunculated and extremely soft and movable to the probe; their appearance is so characteristic that they cannot properly be mistaken for anything else. If they project into the nostril they become pinker and more opaque.

Treatment.—The best method of removal is in most cases with a wire snare, a process which can be rendered quite painless with skilful manipulation and the application of cocaine. Any polypoid mucosa in the neighbourhood should be removed with punch-forceps, but the application of caustics or the cautery only does harm. Recurrence is common, but becomes less rapid if the new polypi are removed at regular intervals before they have grown large. Inflammatory disease in the ethmoidal cells and other sinuses must, of course, receive treatment. In the worst cases, the polypi are so numerous and return so rapidly that the snare cannot deal with them adequately; in such they should be removed with a ring-knife or suitable forceps under general anæsthesia, together with the softened ethmoidal tissue, and any suppurating sinuses be opened at the same time.

PAROXYSMAL OR VASOMOTOR RHINORRHOEA

Synonym.—Allergic Rhinitis.

In this condition fits of sneezing are associated with a profuse watery nasal discharge, irritation of the nasal and conjunctival mucosa, nasal obstruction, and often marked depression and prostration. The discomfort is usually worse in the morning, in overheated rooms, or on going out into the cold. The rapidity with which the attacks come and go is sufficient to distinguish them from an ordinary coryza. The affection usually shows itself in early adolescence and tends to improve with advancing age; it is distinctly hereditary and is often associated, either in the patient or in his relations, with other symptoms of allergy, such as asthma, urticaria or chilblains. Males and females are equally affected, and it is most frequent among the cultivated classes; a mental shock is sometimes the starting-point of the attacks.

Of these cases, *hay fever* is the best known and most marked variety, and is due to specific susceptibility to a proteid substance contained in the pollen of certain grasses; in this country, attacks begin about the end of May and terminate in August. Hay-fever subjects are affected by very minute quantities of this toxin, whereas ordinary people are completely immune. Other individuals are susceptible to the pollen, seedlets or scent of other plants and flowers, and others again to the emanations from horses,* cats, dogs and other animals; and researches show that asthma, urticaria, eczema or rhinorrhœa may be variously produced by many kinds of proteid substances, including common and uncommon articles of diet, such as eggs or lobsters, in certain people who are specifically susceptible to these substances.

Treatment.—The determining factors, which should receive attention, are heightened irritability of the nervous system, occasionally some intranasal abnormality which increases the sensitiveness, and the specific irritant. Nervine tonics, strychnine, arsenic and valerian, are indicated, and attention to the general health. Hay-fever patients are better in a locality as free as possible from pollen; some remain comparatively well at the seaside others only on board ship, while some have to spend the best days of the year in a darkened room. Occasionally great benefit results from the removal of some nasal abnormality, a polypus or a sharp spur impinging on the turbinal, but the result of operative treatment is uncertain. In most cases the nares are normal, and in many of these a light cauterisation of the most sensitive areas is very helpful; the sites usually chosen are on the upper anterior part of the septum, and on the anterior part of the inferior turbinal. Ionisation of the nasal mucosa with sulphate of zinc is also employed. True hay-fever patients may have their susceptibility to pollen lessened by inoculation with dilute extract of pollen; the use of these extracts gives excellent results in a proportion of cases, and, more recently, the attempt has been made to test susceptibility to, and to immunise against, other proteid poisons.

ACCESSORY-SINUS SUPPURATION

Ætiology.—In the large majority of cases infection reaches the accessory sinuses from the nasal cavity, and may result from a simple coryza or from

one of the acute infectious fevers. Influenza is especially liable to produce disease of the sinuses, which may also be caused by measles, scarlet fever, erysipelas, enteric, pneumonia or small-pox. In addition, antral suppuration is caused by infection from the teeth, particularly the second bicuspid and first two molars, whose sockets are in closest proximity to the antral floor. The discharge from one sinus readily enters and infects another, so that disease of several cavities often coexists.

Symptoms and Diagnosis.—If the ostium of a suppurating sinus be occluded the pus is secreted under pressure, and the local symptoms are severe, whereas if the secretion can escape freely there may be no symptoms except discharge. The former class of case has been called "closed" and the latter "open" empyema. The difference between the two is, however, only relative, and many cases are alternating, the severe symptoms being relieved by periodical discharge. As the pressure of the pus in the cavity depends on the rapidity of its secretion, and the degree of occlusion of the ostium by inflammatory swelling, it follows that the closed and open cases correspond generally to acute and chronic suppuration; acute suppuration is usually fairly obvious, but some chronic cases with scanty secretion are only to be detected after very careful examination and may be for long the undiscovered cause of post-nasal catarrh, pharyngitis or chronic toxæmia.

The symptoms, then, are swelling, pain, tenderness and discharge, together with the secondary effects of the suppuration. Swelling is rare; the bony walls are not bulged by an empyema, and this is a point of distinction between it and a tumour or cyst, but occasionally spread of the inflammation causes periostitis, or a fistula in the bone is formed leading to an abscess outside the sinus. Thus, in frontal sinusitis a swelling may appear at the junction of the inner and upper walls of the orbit, displacing the eye downwards and outwards, or an abscess may form here and, after opening, leave a fistula. Similarly, ethmoidal disease may produce a swelling farther back on the inner wall of the orbit, displacing the eyeball outwards. In antral empyema, a little cedema of the cheek, or slight swelling in the canine fossa, may be found, but a swollen cheek is more likely to be due to dental periostitis, while any definite bulging of its bony walls is an indication of a tumour. Pain is often severe in acute cases, and in chronic suppuration there may be considerable neuralgic pain. Pain of an intermittent character, relieved by a sudden gush of discharge from the nose, is highly characteristic of sinus disease, as also is a peculiar periodicity, for it tends to begin regularly at the same time every morning and to get better during the afternoon. The pain may be of a local inflammatory character, or may be of a neuralgic type and referred to various parts. In antral suppuration it is over the cheek, or may be referred to the teeth or frequently to the supra-orbital region. The pain of frontal sinusitis is over the cavity or along the supra-orbital nerve; that of ethmoidal disease is over the nasal bridge, behind the eye or in the temple, and in sphenoidal suppuration, in the middle of the head, behind the eye, on the vertex or in the occipital region. Tenderness can usually be elicited in frontal empyema by percussion over the anterior wall, and especially by pressing upwards against the floor of the cavity; it is less marked in antral disease, in the canine fossa. Discharge into the nose is the most important, and often the only, symptom. A localised stream of pus in the nose, which reappears after removal, is, in the absence of a foreign body, conclusive

evidence of suppuration in an accessory sinus. The differentiation of the affected sinus is made by following the pus to its source with a probe and, in the case of the antrum, by tapping with a trocar and cannula. The antrum, frontal and anterior ethmoidal cells open into the middle meatus, and the posterior ethmoidal and sphenoidal into the superior meatus. Further assistance is afforded by transillumination and skiagraphy. Fœtor, both subjective and objective, is often present, and a serious degree of anæmia and ill-health frequently results.

Complications.—These include pharyngitis, laryngitis, bronchitis, and otitis media; the swallowed pus causes various forms of gastric and intestinal disorder, including appendicitis. Acute septicæmia, and pyæmia are rare, but symptoms of chronic poisoning are common, and include anæmia, arthritis, fibrositis, and even mental aberrations. A very important series of complications results from extension of the inflammation to surrounding parts: orbital abscess or cellulitis, osteo-myelitis of the frontal bone, cerebral abscess, thrombosis of the cavernous sinus, paralysis of the oculo-motor nerves and, from the sphenoidal sinus, papillœdema and optic atrophy.

Treatment.—This, in acute cases, consists in rest in bed, hot fomentations to the affected part, aperients, a light diet, and a few doses of aspirin. Inhalations of mentholised steam at frequent intervals are of value, and may be prepared by adding 10 drops of 25 per cent. solution of menthol in spirit to a pint of steaming water in an inhaler. In recent cases of antral suppuration, the cavity should be tapped with trocar and cannula and washed out with a warm saline lotion; this should be repeated daily or every two or three days, according to the severity of the disease, and will effect a cure in a large proportion of cases in an early stage. Frontal sinusitis has a greater tendency to spontaneous cure; the anterior end of the middle turbinal may be amputated and occasionally a cannula can be passed and the cavity washed out. Cases which fail to recover under such treatment, and those of chronic suppuration, must be submitted to operation.

SYPHILIS

CONGENITAL SYPHILIS

The *early form* appears at any time within 3 months after birth, usually within the first few weeks. The symptoms, frequently called "the snuffles," are those of nasal discharge and obstruction; the former may be thin and ichorous, or purulent and bloodstained, and is often associated with cracks and excoriations about the nostrils, upper lip and angles of the mouth; the obstruction may cause attacks of choking and frequently prevents the baby from taking the breast, and so produces wasting and malnutrition. These symptoms are not pathognomonic of syphilis, but may also be caused by catarrhal and purulent rhinitis, therefore the diagnosis must be established by the concomitant lesions.

The *late form* appears usually about the period of puberty, but may occur at any time after the age of about 5 years. It is characterised by a slow destructive gummatous infiltration and ulceration, and the symptoms are those of nasal catarrh and obstruction, frequently with fœtor and crusting;

this chronic rhinitis destroys the ciliated epithelium, and may thus cause a true atrophic rhinitis which persists after the syphilis has become quiescent or cured. Congenital syphilis is apt to produce a very characteristic "saddle-back" flattening of the bridge of the nose.

ACQUIRED SYPHILIS

Primary chancre is very rarely seen on the ala of the nose, and is accompanied by bubo of the submaxillary and pre-auricular glands, and by much induration and swelling.

Secondary syphilis does not produce noticeable symptoms in the nose; there may be rhinorrhœa and obstruction associated with hyperæmia of the mucosa.

Tertiary syphilis occurs usually in the form of a diffuse gummatous infiltration and ulceration, which may proceed to necrosis of any of the bony or cartilaginous walls of the nose; there is profuse purulent discharge, often bloodstained, which tends to dry into greenish-black crusts, the odour of which is extremely offensive. A localised gumma may occur on the septum, where it forms a smooth round swelling projecting into both nostrils which, by its contraction after healing, produces a steep depression of the bridge just below the nasal bones. Syphilitic ulceration sometimes attacks the external parts of the nose, causing perforation of the ala, or destruction of the columella with a characteristic depression of the nasal tip.

Diagnosis.—This seldom presents much difficulty; the form with crusting and ozæna imitates atrophic rhinitis, but in the latter there is never necrosis or decided ulceration—indeed intranasal necrosis may be considered pathognomonic of syphilis. A septal gumma has an appearance identical with that of a hæmatoma, but without the sudden onset and history of traumatism. Syphilitic perforations nearly always involve the bone, whereas those due to rhinitis sicca or lupus never do. Some cases of diffuse infiltration resemble lupus; but in the latter there is no necrosis or offensive odour, the characteristic nodules are usually to be seen at the edges of the lesion, and other patches of lupus may be found on the skin or in the fauces. The chief difficulty of diagnosis lies between severe syphilitic infiltration and malignant disease, but it can usually be determined by the clinical appearance, especially by the characteristic edge of the syphilitic ulcer, by the examination of an excised portion, by the Wassermann reaction, and by the results of anti-syphilitic treatment.

Treatment.—General treatment must be very prompt and energetic to prevent irremediable deformity, and should ordinarily be begun with the injection of arsphenamine or of one of its congeners. Of local treatment, the lesions should be kept clean by frequent syringing with a saline lotion, to which lysol, sanitas or listerine may be added when the odour is offensive; any necrosed bone must be removed as soon as it is loose.

LUPUS AND TUBERCULOSIS

Ætiology.—With the exception of the rare occurrence of tuberculous ulceration as a terminal infection in advanced phthisis, the lesions produced

in the nose by lupus and by tuberculosis are indistinguishable; it appears that the tubercle bacillus finds in the nasal mucosa a medium unsuitable for its development, its virulence is diminished, and it can only produce the modified lesions known as lupus. It is possible, also, that this modification of the bacillus by sojourn in the nose is the ordinary cause of lupus; at any rate it is frequently primary in the nares, whence it spreads to the fauces and larynx and on to the face and hands. The disease begins most often between the ages of 15 and 30, is twice as common in females as in males, and is usually seen in badly nourished people of the poorer classes.

Symptoms.—The early lesions are found on the antero-inferior part of the septum, the nasal floor and the front end of the inferior turbinal, within reach of the finger-nail, which probably conveys the infection. The characteristic "apple-jelly" nodules are seen, with or without ulceration, the latter with rounded slightly raised margins, and tending to spread in some directions and cicatrize in others. The lesions are covered by small adherent scabs, and perforation of the septal cartilage is common. The alæ often become involved with destruction of the margin or with perforation, and the nostrils may be much narrowed and deformed by scarring, while the lachrymal duct is frequently involved. The progress of the disease is extremely slow and may continue over many years. The subjective symptoms are nasal obstruction with a slight sticky discharge.

Diagnosis.—The nares should be examined in all cases of cutaneous lupus, for, if the disease remain unhealed in the nose, relapses will continually occur. In the majority of cases of nasal lupus the diagnosis is cleared up by the presence of lesions or scars on the face, fauces or larynx. The difficulties of diagnosis are from rhinitis sicca with perforation, and from syphilis. The typical brownish nodules are pathognomonic and can always be found by careful examination when the lesions are progressing; they can be made more conspicuous by blanching the mucosa with adrenaline. The scabbing of rhinitis sicca quickly clears up under simple emollient treatment, while the lesions of syphilis are more rapidly progressive and tend to involve bone.

Treatment.—The affected areas are defined by the application of adrenaline and thoroughly and carefully scraped away with a sharp spoon, a general anæsthetic being employed if the lesions are extensive; small lesions and recurrences are destroyed with the galvano-cautery. Nascent iodine by Pfannenstiel's method may be employed; sodium iodide is given in 7-grain doses six times a day, while the nose is packed with gauze kept moist with peroxide of hydrogen, 10 volume strength, with 5 per cent. of acetic acid added; when a marked reaction has been obtained this solution should be diluted to half its strength, the iodide being continued as before; the treatment may have to be persisted in for several months. Radium is still under trial, and tuberculin has not established its value in this affection.

Of general treatment, arsenic in full doses, fresh air, cod-liver oil and fattening foods are of value. Lupus does not show the same tendency to spontaneous cure in the nose as in the larynx; it is easy to obtain improvement, but complete cure is difficult.

TUMOURS OF NOSE

Papilloma occurs on the skin lining the vestibule and differs in no respect from cutaneous warts elsewhere. On the nasal mucosa it is excessively rare, occurs usually on the septum, has a narrow pedicle, a rough red or greyish surface, and bleeds readily when touched; occasionally the growths are multiple and, when large, are difficult to diagnose from a malignant growth, but they do not erode bone; they tend to recur locally after removal.

Fibroma, similar to the naso-pharyngeal fibromata, occurs, though rarely, as a smooth pink growth attached to the posterior region of the nares. It bleeds readily and spontaneously and demands great caution in removal.

Angio-fibroma, or "bleeding polypus of the septum," is less uncommon. It varies from the size of a pin's head to that of a filbert, is red or purple, smooth or finely lobulated, sessile or pedunculated, and grows from the anterior part of the septum. The prominent symptom is epistaxis, for the tumour bleeds freely and spontaneously. When pedunculated, it may be removed with the snare and the base cauterised; when sessile, it should be stripped off the underlying cartilage with an elevator. Recurrence is common.

Osteoma and *enchondroma* are extremely rare, usually grow from the ethmoidal region and produce obstruction and deformity.

Cysts.—Mucous polypi are occasionally cystic; the so-called "ethmoidal cyst" is an enlarged cell in the anterior part of the middle turbinal pressing on the septum and causing obstruction and headache. True cysts are sometimes seen on the anterior part of the nasal floor, and arise from the roots of incisor teeth. When small, they may be treated by intranasal removal of part of the wall; when large, they should be dissected out from the gingival fold. *Mucocele*s are cystic dilatations of the antrum, frontal sinus, or an ethmoidal cell.

Malignant tumours occur in all varieties: carcinoma, sarcoma, and endothelioma. Though they sometimes appear on the septum or nasal floor, the usual site of origin is the ethmoidal region. They tend to expand the bones of the face, producing a characteristic frog-like deformity, and they frequently invade the antrum and expand its walls, constituting a common variety of tumour of the upper jaw. The facial, palatine and orbital walls of the antrum may each be bulged outwards, the latter with displacement of the eyeball, and egg-shell crackling can sometimes be elicited. Sanious discharge and free spontaneous hæmorrhage are prominent symptoms and important for diagnosis. Surgical excision produces permanent cure in only a small proportion of cases; better results, especially in endothelioma and sarcoma, are obtained by the implantation of radium. According to the situation of the growth, access is gained by opening the antrum through the canine fossa, by lateral rhinotomy, or for the lower part of the nose by an incision in the gingivo-labial fold from the molar teeth of one side to the other.

DISEASES OF THE NASO-PHARYNX

ADENOIDS

Ætiology.—By this term is implied a chronic enlargement of the lymphoid tissue of the naso-pharynx, the “pharyngeal tonsil.” This is normally present in childhood and disappears by the age of 20 or thereabouts, but if chronically enlarged may remain up to any age. The precise stage at which the enlargement becomes pathological can only be determined by the symptoms which it produces; these usually become manifest between the ages of 3 and 8, but occasionally show themselves at or soon after birth. The incidence of adenoids is universal, but they are most common in damp temperate climates, and chronic or recurrent nasal catarrh is the principal factor in the causation; the infectious fevers, particularly measles, scarlet fever, and diphtheria, are also a frequent cause of the hypertrophy.

Pathology.—The adenoid, as it should really be called, or enlarged pharyngeal tonsil, is a mass of lymphoid tissue of definite anatomical shape; it is thickest above and tapers away below, and presents a series of ridges which radiate from below upwards and slightly outwards. In older patients the mass is firmer and more fibrous, and the ridges are often adherent in places, leaving deep clefts and furrows in which secretion can collect and decompose.

Symptoms.—The symptoms of adenoids are many and various, and include those due to nasal obstruction, those caused by infection and by the extension of inflammation, and reflex processes attributable to irritation and lowered vitality. In infants the nasal obstruction interferes with sucking and a serious degree of malnutrition will result unless the baby be carefully spoon-fed. Older children snore at night, breathe heavily in the day, and either bolt their food or eat very slowly owing to the necessity of breathing through the mouth. Owing to lack of oxygen the patients sleep restlessly, wake unrefreshed and often suffer from a peculiar inability to concentrate the attention sometimes called “aproxexia.” Persistent nasal obstruction during the period of growth mechanically produces permanent deformities of the jaws and face which narrow the nasal passages, prevent the mouth from closing naturally and thus perpetuate mouth-breathing. When the mouth is habitually held open, the *alæ nasi* are pulled downwards with the cheeks, and become narrow and slit-like and fall in like valves with each inspiration; this “alar collapse” is an important cause of obstruction in neglected cases of adenoids. The palate is narrow and highly arched; the dental arch is narrow and V-shaped, so that the upper incisors, crowded and prominent, look outwards rather than forwards, and are not covered by the short upper lip; the lower jaw retains its infantile obtuse angle, and the lower incisors lie behind the upper; the chin is receding and, in the worst cases, when the molar teeth come into contact on biting, the incisors cannot meet. Only a proportion of cases of adenoids show these deformities, and there is, indeed, considerable uncertainty as to the importance of adenoids in their ætiology; undue softness of the bones, such as occurs in rickets, is doubtless an additional factor, and also in the causation of the malformations of the chest which result from the obstruction to the entry of air. The long

narrow unexpanded chest with acute costal angle and prominent scapulae is the commonest deformity. Harrison's sulcus, a transverse depression corresponding to the attachment of the diaphragm; pigeon-breast, a prominent sternum with depressed costal cartilages; and funnel-breast, a sharp depression at the lower end of the sternum, are also encountered.

Various infective processes result from the spread of inflammation and, if the naso-pharynx be large, are not necessarily associated with nasal obstruction. The terribly common catarrhal and suppurative affections of the ear in children are, in an overwhelming majority of cases, the result of adenoids. Blepharitis and phlyctenular conjunctivitis are also associated with adenoid vegetations. Feverish attacks, often with tender enlargement of the cervical glands, are caused by infection of the pharyngeal and faucial tonsils, and tuberculous disease of the glands is usually due to passage of the bacilli through these portals; in such cases the tonsils and adenoids may remain unaffected or may themselves show tubercles under the microscope. Chronic or recurrent bronchitis frequently results from the infection spreading to the lower air-passages. The mucus secreted by the adenoids is swallowed in large quantities, and produces derangements of stomach and intestines with failure of growth and general health. Finally, mouth-breathing predisposes to dental caries. The irritation of these vegetations, and their effect on respiration and the general health, account for numerous reflex and nervous disturbances. Among them may be enumerated laryngitis with spasm called "laryngitis stridula," spasm of the glottis without laryngitis or "laryngismus stridulus," stammering, reflex cough, asthma, night terrors and nocturnal enuresis; it should be stated that the latter disorder is by no means always to be cured by removal of the concomitant adenoids, and that in general too much stress must not be laid upon the presence of adenoids as the causative factor in all these reflex disturbances.

Diagnosis.—In the majority of tractable children a view of the naso-pharynx can with patience be obtained with a good light and a very small rhinoscopic mirror, when the upper part of the septum and the concavity above it are seen to be occupied by an irregular convex mass. Where this is impossible a rapid digital examination may be required; this is extremely unpleasant to the little patient, and may be postponed, in those cases where the tonsils are sufficiently large to call for removal, until the child is anæsthetised. Similarly, in very frightened, intractable children, if the symptoms point strongly to adenoids it is wiser to give an anæsthetic for examination, being prepared to remove the vegetations if present. In the mongolian type of idiocy the tongue is large and the mouth persistently open, and in microcephaly the extremely undeveloped naso-pharynx causes nasal obstruction; cases of both these types of maldevelopment are often brought to the doctor in the hope that removal of their adenoids will cure their "backwardness," and care should be taken not to fall into the error of performing a useless operation, though if a well-marked adenoid be present it should be removed under a guarded prognosis. On the other hand, adenoids can be present and produce serious secondary effects without causing nasal obstruction or any appearance of the typical "adenoid facies."

Treatment.—The normal naso-pharyngeal tonsil becomes swollen during a coryza, and such temporary swelling should not be diagnosed as "adenoids," by which term chronic hypertrophy is understood, and does not call for

removal provided that it subsides promptly, does not frequently recur, and is not associated with otitis media or other important complications. In such cases, and when the only symptom is a mild catarrh, the regular use of a simple warm saline lotion with a rubber ball-syringe (see p. 1086) will often effect a cure; in children below the age of 5 or 6 syringing is apt to be difficult and the lotion may be used in a spray, while in infants it is best to drop it into the nostrils from a small pipette like the filler of a fountain-pen. This treatment should be combined with open air—if possible a change to the seaside or a bracing country district—cod-liver oil, iodide of iron, or arsenic. Breathing exercises are of great value in these slight catarrhal cases, but only do harm where there is marked obstruction.

When the enlargement frequently recurs or has gone on to chronic hypertrophy, operative removal is the only treatment, and this is especially called for when any aural symptoms supervene, or when cervical adenitis is present. After operation the general treatment referred to above is valuable, but the nose should on no account be syringed until healing is complete, as this encourages aural complications. If the alae nasi are collapsing, or the chest narrow, breathing exercises are of use, but healthy open-air occupations are more useful still. In patients in their teens, or upwards, turbinal hypertrophy has not infrequently resulted; the surgeon should be prepared to snare off enlarged posterior ends of the inferior turbinates at the time of the operation, and intranasal cauterisation may be required later.

TUMOURS

Innocent tumours in the naso-pharynx are exceedingly rare; the so-called "choanal polypus" is a variety of nasal mucous polypus which hangs into the naso-pharynx from a long pedicle attached within the nares.

Fibroma of the naso-pharynx, or naso-pharyngeal polypus, occurs usually in males between the ages of 10 and 25, grows by a broad pedicle from the periosteum of any part of the walls of the naso-pharynx, usually from the basi-sphenoid, and forms a smooth, rounded, pink mass which fills the naso-pharynx and sends prolongations into the nasal cavities. The palate is pushed downwards, the bones of the face expanded, and the eyeballs separated and displaced, producing the "frog-face" deformity. The cardinal symptoms are nasal obstruction and discharge, with headache and severe epistaxis; aural complications may follow, or the eyes may be involved, with diplopia, exophthalmos and compression of the optic nerve; and, finally, death results from exhaustion, hæmorrhage, sepsis or cerebral invasion. Histologically the tumour is composed of fibrous tissue containing numerous thin-walled blood vessels and a variable admixture of round or fusiform cells, so that in some cases it might be described as a fibro-sarcoma; but it is not truly malignant, for it neither involves the glands nor becomes disseminated, though there is a tendency to local recurrence after removal.

Radium has proved of considerable value in reducing the size and vascularity of these growths, as a preliminary to operation.

Malignant tumours are not common in the naso-pharynx, but epithelioma, sarcoma and endothelioma all occur. The early symptoms are chiefly pains of a neuralgic character and those produced by Eustachian obstruction;

later, epistaxis, nasal obstruction, secondary involvement of glands and affections of the eye and cranial cavity may appear. Surgical removal is rarely feasible, but sarcomata and endotheliomata in this region sometimes yield remarkably to treatment by radium.

HAROLD S. BARWELL.

DISEASES OF THE LARYNX

ACUTE CATARRHAL LARYNGITIS

Ætiology.—The affection ordinarily occurs as part of a coryza, or cold, the inflammation spreading downwards from the nose or naso-pharynx. It is also caused by over-use of the voice, especially with faulty voice-production, and frequently a slight catarrh is made worse by using the voice during a cold. It occurs in many infectious fevers, *e.g.* influenza, measles, scarlet fever, typhoid and small-pox, and it is occasionally a result of traumatism, instrumentation, or the inhalation of irritating fumes in chemical works or of the gases used in warfar. Predisposing causes are chiefly those factors which favour attacks of coryza, such as nasal obstruction or discharge, sedentary occupations and overheated rooms; apart from local tuberculous lesions, consumptives are very subject to laryngeal catarrh.

Symptoms.—The symptoms consist of hoarseness or aphonia, local discomfort varying from dryness or tickling to a burning sensation or actual pain, and irritable cough. There is little expectoration, unless the trachea and bronchi are involved. At the onset there may be slight feverishness and malaise. The degree of hoarseness is by no means proportionate to the objective appearances; the voice may be quite good in cases of decided hyperæmia, and may be completely lost when little abnormal is to be seen. This depends largely on the neuro-muscular tone; a muscular man will retain a strong voice with a degree of inflammation which would render a weakly woman completely aphonic—indeed some women lose the voice with every slight cold, so that it becomes difficult to differentiate between laryngeal catarrh and “functional aphonia.” On the other hand, in some voice-users redness of the cords appears to be the normal condition and causes no interference with function. This variable effect on the voice is to be observed in all forms of laryngeal disease. In children, acute laryngitis is a serious affection. They show a far greater tendency to œdema and to spasm, and as the glottis is not only absolutely but relatively smaller than in adults, a dangerous dyspnoea may ensue with great rapidity. The larynx is reddened, and this is most obvious on the parts usually pale—the epiglottis and vocal cords, the vessels on the former being unduly prominent. The cords may be red, pink, yellowish, or merely have lost their bright pearly lustre. A small amount of mucous secretion is generally present, but no large accumulations or strings of mucus, such as are seen in chronic laryngitis. There is often a little swelling of the cords so that, on phonation, their edges come into contact at the centre; this explains how singers’ nodules are caused by use of the voice during a laryngitis.

Treatment.—People suffering from the slighter degrees of laryngitis

rarely apply for treatment, unless they are professional voice-users. The patient should remain in a warm (65° F.), well-ventilated room, preferably in bed, and must not attempt to use the voice. The coryza, if present, should be treated at the same time. Steam inhalations are of value and may be used from an inhaler or from a jug round the mouth of which a towel has been wrapped in the shape of a cone. The water should be at a temperature of 130° to 140° F., and one of the following medicaments may be added in the proportion of one drachm to the pint: Compound tincture of benzoin (Friar's balsam) fl. oz. 1, with or without menthol, grs. 10–15; or benzoic acid, grs. 3, kaolin, grs. 12, tincture of tolu, min. 18, and water to fl. oz. 1, these being sedative, while oleum pini sylvestris, min. 40, magnesii carb. levis, grs., 20, water to fl. oz. 1 is mildly stimulating. Steam inhalations should only be ordered when the patient can remain in a warm room; when he is not confined to the house, or in a later stage, an oily solution from an atomiser is preferable, such as menthol, grs. 7, camphor, grs. 3, chlorbutol, grs. 5, liquid paraffin, fl. oz. 1. Internally, expectorants are indicated; tinctura ipecacuanhæ, min. 10, or vinum antimoniale, min. 5, potassium iodide, grs. h2 or 3, ammonium carbonate, gr. 4—singly or in combination—or amonium chloride, grs. 5, or oil of cubebs, min. 5 in syrup, every 4 or 6 hours. If cough is severe it should be restrained; a lozenge of morphine and ipecacuanha is useful, or a linctus containing diamorphine hydrochloride, gr. $\frac{1}{4}$, or liquor morphinæ, min. 2 to 4 to each drachm.

The acute laryngitis of children calls for prompt treatment. One or two grains of calomel may be given every 3 hours until the bowels have acted freely, after which it may be continued in $\frac{1}{2}$ -gr. doses three times a day. Hot fomentations to the neck and a steam-kettle are advisable, and in acute febrile cases, 1 minim each of tincture of aconite and vinum antimoniale every 3 hours. If dyspnœa occurs, an emetic dose of ipecacuanha often gives prompt relief; 60 minims of the tincture followed by 30 minim doses every half-hour until vomiting occurs: in very young or weakly children, 15 minims every quarter-hour is to be preferred.

SPASMODIC LARYNGITIS

Synonym.—Laryngitis Stridula.

This is simply catarrhal laryngitis with spasm of the glottis as a marked feature. It is a disease of childhood and is predisposed to by general ill-health, rickets and adenoids. The onset is that of an ordinary cold, with slight feverishness, hoarseness and a frequent cough, and during the evening or night the respiration becomes embarrassed. There is inspiratory stridor, recession of the epigastrium and lower ribs and, in some cases, an alarming degree of asphyxia. The symptoms tend to subside towards morning and, though they may recur on the next few nights, it is usually with diminishing severity. The condition should be distinguished from laryngismus stridulus, in which there is no hoarseness or other symptoms between the attacks. The evanescence of the symptoms serves to distinguish it from cedematous laryngitis and from diphtheria, in which the attacks become increasingly severe. The general health requires attention, and adenoids, if present, must be removed after recovery has taken place.

ŒDEMATOUS LARYNGITIS

Synonym.—Œdema of the Larynx.

Ætiology.—Œdema of the larynx is not a disease but a pathological condition due to a variety of causes. Non-inflammatory œdema may be mentioned here for the sake of completeness; it occurs, though rarely, as part of the general anasarca of renal and cardiac disease. Angio-neurotic œdema sometimes occurs in the larynx, in which event it produces rapid and sometimes fatal dyspnoea (see p. 1074). The swelling which occasionally results from administration of potassium iodide in susceptible subjects may be placed in the same category.

Inflammatory œdema seldom results in adults from a simple catarrh, but it may do so in children; it more often occurs as part of an acute septic infection of the pharynx, trachea and bronchi, "acute fulminating laryngo-tracheo-bronchitis." Œdema may follow various forms of traumatism, the drinking of corrosive poisons, inhalation of irritating vapours, such as the poison gases of warfare, the lodgment of foreign bodies, or rough or unduly prolonged bronchoscopy. Scalding, from attempts to drink from a kettle-spout, is a common cause among children. In other cases it is a sequela of typhoid fever, pneumonia, scarlet fever or small-pox, and is a local complication of syphilitic, tuberculous, cancerous or traumatic ulceration.

Symptoms.—If part of a septic pharyngo-laryngitis, the general symptoms are severe. The chief local symptom is dyspnoea with inspiratory stridor and the associated symptoms of asphyxiation; there is hoarseness or aphonia, local discomfort and tenderness, and sometimes dysphagia. The aryteno-epiglottidean folds are enormously swollen, appearing as pale or purple translucent flask-shaped masses; if the epiglottis be œdematous it forms a sausage-shaped swelling of the same appearance. The mucosa of the vocal cords is too adherent to permit much swelling, and "œdema of the glottis" is therefore a misnomer. The subglottic region is lax and may become swollen; indeed, the œdema may be confined to this region and then appears as a red swelling below each vocal cord. In children œdema may be inferred from the steadily increasing dyspnoea without the rapid increase and decrease typical of spasmodic laryngitis.

Treatment.—In slight cases, the swelling may be reduced by sucking ice and by the application of an ice-bag to the neck; the latter is inadmissible in young children. A spray of adrenaline, 1 in 1000, may be used. Hypodermic injections of pilocarpine, gr. $\frac{1}{2}$, are recommended and, for the œdema produced by iodides, large doses of bicarbonate of soda. Free scarification of the œdematous tissues at the upper aperture should be practised without undue delay; in adults this should be done with a laryngeal lancet under guidance of the mirror, but in children it is best performed with a curved bistoury guarded to near the point with strapping and passed down along the left forefinger as a guide. If this does not give quick relief, or if the dyspnoea be severe, tracheotomy should be performed without delay. Intubation is not suitable for cases of œdema of the upper aperture of the larynx, though it may be employed for subglottic cases, provided that skilled attention be immediately available should the tube be coughed out. Angio-neurotic œdema should be treated by a spray of adrenaline, and the same

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drug, or a colloidal preparation of calcium, may be injected hypodermically ; tracheotomy may here also be required.

MEMBRANOUS LARYNGITIS

The formation of false membrane in the larynx is nearly always part of an attack of diphtheria, which is discussed elsewhere, but by the term "membranous laryngitis" is implied a formation of membrane of non-diphtheritic origin. Apart from traumatic cases, due to irritating chemicals and scalds, inflammation of the larynx accompanied by membrane may be caused by streptococcal infection. The affection occurs especially in children between the ages of 2 and 8 years. The first symptom is hoarseness, soon followed by a brassy cough and the signs of dyspnoea ; the patient is restless and the temperature rises rapidly to 103° or 104°. In some cases, however, the disease takes a subacute form, the attacks of dyspnoea being worse at night and abating towards morning. The diagnosis from diphtheria is only possible by bacteriological examination, and pending the report the case should be treated with antitoxin ; but it may be noted that the pharynx is nearly always involved in diphtheria, whereas in membranous laryngitis the disease is often primary in the larynx. The prognosis is grave, worse than that of diphtheria since the introduction of antitoxin. An emetic dose of ipecacuanha should be given, and a steam-tent and hot fomentations to the neck are advisable. Calomel treatment is recommended ; 1 or 2 grains every 3 hours until the bowels have acted freely, and subsequently 1 grain three or four times a day ; the sulphanilamide group of drugs will doubtless be of value in this, as in other streptococcal infections. Tracheotomy or intubation must not be delayed when there is serious dyspnoea.

CROUP

Croup is an expression which dates from a period when the diagnosis of diseases of the throat was less exact than now but, as it is still in occasional use, it may well receive a definition in this place. The term can be used to cover any respiratory obstruction or obstructive dyspnoea, especially in children, but is usually limited to acute affections and therefore does not ordinarily include such conditions as multiple papillomata, congenital web or cicatricial stenosis. Croup may thus be caused by various inflammatory affections such as cedematous laryngitis, membranous laryngitis (both diphtheritic and non-diphtheritic), spasmodic laryngitis and retropharyngeal abscess, or by such reflex disturbances as laryngismus stridulus and spasm of the glottis, and is in fact descriptive of a symptom rather than of a disease.

CHRONIC LARYNGITIS

Ætiology.—The causation is similar to that of acute catarrhal laryngitis ; indeed, chronic laryngitis is often the result of recurrent or persistent acute catarrh. The principal factors which predispose to chronicity are nasal

obstructions and discharges, dusty occupations and lack of fresh air, over-use of the voice and faulty voice-production, and the abuse of alcohol or tobacco ; consumptives are particularly liable to non-specific catarrhal laryngitis, and oral sepsis must not be omitted. Almost any cause of general ill-health may be included among the predisposing causes, such as gout, rheumatism, anæmia, and gastro-intestinal, cardiac and hepatic disorders.

Symptoms.—The only constant symptom is impairment of the voice, which is hoarse, easily tired or even, though rarely, completely aphonic ; it is sometimes weakest when tired in the evening, but is often at its worst on rising in the morning or after a rest. There is frequently a sensation of aching, dryness, tickling or of a lump in the throat, and there is usually some cough, but little expectoration unless the trachea and bronchi are involved.

The objective appearances vary with the severity of the affection. The larynx generally is of a deeper red than usual, and the vocal cords have lost their normal pearly lustre and are pink or grey ; they are usually somewhat thickened at the edges, and enlarged vessels may be visible on their surface ; the vocal processes are often prominent and may be reddened or show up white against the hyperæmic cord. Strings of sticky secretion may stretch between the cords, or a little globule of mucus may form on the centre of the cord during phonation ; adduction is frequently imperfect. When the epiglottis is reddened, its yellow edge stands out clearly and enlarged vessels are visible ; the ventricular bands are often swollen so as to hide the outer part of the cords. The mucous membrane in the inter-arytenoid space, thickened and relaxed, is seen to be thrown into folds on adduction of the cords, and may form a mass large enough to prevent their approximation.

Atrophic rhinitis usually produces a form of inflammation, *laryngitis sicca*, in which small brown scabs adhere to the cords and posterior commissure, but occasionally the disease itself spreads to the larynx, which is covered by large greenish or brownish-black fetid crusts ; more rarely still the crusts extend into the trachea and cause severe dyspnoea, which may prove fatal.

Pachydermia laryngis is a somewhat rare variety of chronic laryngitis, occurring principally in middle-aged men. It is frequently ascribed to alcoholism, perhaps on insufficient grounds, to syphilis and to tubercle ; the diagnosis between pachydermia and these two diseases is, however, often a matter of difficulty. The characteristic epithelial thickenings are probably of the nature of corns, resulting from frequent cough and continued irritation. There is hoarseness of a rough raucous character, but no particular discomfort. The epithelial thickening is pink or whitish and occupies the posterior or cartilaginous region of the glottis from the vocal processes backwards to the posterior commissure. A circumscribed swelling appears on each vocal process, with a small cup or depression at the apex ; the approximation of the cords is better than would be expected, because the prominence on one vocal process fits into the depression at the other. The epithelium of the inter-arytenoid space is thrown into ridges, which fill up the angles between the arytenoid and the posterior commissure, but leave a depression in the middle line. These firm, opaque, symmetrical swellings, without ulceration, are distinguishable from the soft irregular granulations

of a tuberculous lesion, and the cup-shaped swelling on the vocal process, even when more marked on one side, should not be mistaken for an early epithelioma.

Treatment.—The detection and correction of the aetiological factors are the most important part of treatment. Any constitutional disturbance, such as anæmia, rheumatism, gout or dyspepsia, should receive attention. Over-indulgence in tobacco or alcohol, lack of ventilation and exposure to dust must be considered, and with teachers the black-board chalk is a common source of irritation.

Incorrect voice-production is a factor of great importance especially, but by no means exclusively, among those who use the voice largely in their occupations; in such, a course of lessons in voice-production often works wonders. In a large proportion of cases the primary cause of the laryngitis is to be found in the nose, therefore any source of nasal obstruction, catarrh or suppuration must be carefully looked for and treated; any concomitant pharyngitis should also receive attention.

Locally, treatment must begin with rest of the voice, which should be absolute in the case of professional voice-users. Where there is much secretion a saline lotion may be used in a spray—sodium bicarbonate, borax, sodium chloride, 10 grains of each, thymol water, 120 minims, glycerin, 60 minims, water to 1 ounce. Oily solutions are usually preferred, such as menthol, 5 grains, camphor, 2 grains, chlorbutol, 5 grains, or oil of eucalyptus, pine or gaultheria in similar proportions, to an ounce of liquid paraffin. The direct application of pigments is not often called for, and is to be recommended only when pachydermatous changes are present; in such cases the cautious application of a solution of nitrate of silver on a cotton-wool mop once a week may be tried, beginning with 5 grains to the ounce and increasing the strength gradually to 50 or more grains. Dundas Grant advises an alcoholic solution of salicylic acid, beginning with 1 per cent. and increasing to 6 or 8 per cent.

Internally, small doses of potassium iodide, 2 or 3 grains, or the yellow proto-iodide of mercury, $\frac{1}{18}$ grain, three times a day over long periods, is of value.

CONGENITAL LARYNGEAL STRIDOR

In this condition there is an exaggeration of the infantile shape of the upper aperture of the larynx; the epiglottis is sharply folded laterally, the ary-epiglottic folds are almost in contact, and the opening is thus reduced to a narrow vertical slit. As these parts are very flaccid in infancy, they become sucked together during inspiration and, by their vibration, produce the characteristic stridor. This stridor is noticed very soon after birth, it is inspiratory, of a peculiar purring or even musical character, and is most marked during active breathing and crying. The voice is unaffected, and there is remarkably little sign of dyspnoea or distress. These characteristics distinguish the condition from other forms of obstruction found in infants, such as laryngeal webs or papillomata, or "thymic asthma." It tends to disappear during the second year of life, but the prognosis must be guarded in early infancy, for an attack of bronchitis is more than ordinarily dangerous and kills a proportion of these patients.

SYPHILIS

CONGENITAL SYPHILIS

The early, or secondary, form appears in the first few months of life and is rarely recognised, but it may be suspected when the cry is hoarse in an infant with active syphilitic lesions.

Tertiary lesions are rare, and usually make their appearance about puberty, less often during the second dentition. The disease takes the form of diffuse infiltration with or without ulceration; the swelling may produce obstruction, and rarely cicatricial stenosis may ensue. The symptoms are stridor with hoarseness, and tracheotomy may be required.

ACQUIRED SYPHILIS

Symptoms.—Secondary lesions are superficial, cause no symptoms but hoarseness, and seldom come under observation. The commonest manifestation is an erythema which differs from that of catarrhal laryngitis by being more uneven and patchy in its distribution, and may affect one cord, leaving the other normal. Mucous patches are occasionally found on the cords or on any part of the larynx, appearing as superficial erosions with a smooth greyish base and a sharply defined hyperæmic margin. The fauces are nearly always affected at the same time.

Of tertiary lesions, the superficial serpiginous ulcer is occasionally seen with the same characters with which it more commonly appears on the fauces. Diffuse infiltration may attack any part of the larynx, but chiefly, in contra-distinction to tuberculosis, the anterior regions, such as the epiglottis and the front parts of the vocal cords. Subglottic infiltration is fairly frequent and abduction of the cords often limited, so that stenosis is much commoner than in tuberculous disease. The typical circumscribed gumma is distinctly rare; it is single, unilateral, and attacks especially the epiglottis and arytenoids, and usually breaks down rapidly to form a deep excavated ulcer, which may result in perichondritis, exfoliation of cartilage, and, ultimately, in severe cicatricial stenosis. The subjective symptoms are hoarseness, of a peculiar rough "raucous" character, and sometimes dyspnoea with stridor; pain is in general not a prominent symptom, but a gumma on the upper aperture may cause severe dysphagia.

Diagnosis.—From tuberculous disease the diagnosis is discussed under that heading (see p. 1107). From epithelioma a gumma is distinguished by its more rapid evolution; the edge of an epithelioma is thick and everted and its base nodular, whereas these characters are less marked in syphilitic ulceration, the margin of which is hyperæmic and frequently sharply cut; while other parts of the larynx or fauces often show syphilitic lesions. The hard infiltration of secondarily involved glands is characteristic of malignant disease.

Treatment.—General treatment is urgently called for to prevent perichondritis and stenosis. Local treatment is not often required. Tracheotomy should be performed when decided dyspnoea is present; it seems to aid the recovery of the larynx, and the tube can often be omitted in a short

time, when anti-syphilitic medication has removed the obstructing lesion. Necrosed pieces of cartilage must be removed by internal or external operation, and insufflations of orthoform are indicated when dysphagia is present.

LUPUS

Lupus in the larynx is comparatively rare, and is probably always secondary to the disease in the nasal passages.

Symptoms.—The lesions begin on the epiglottis and slowly spread along the aryteno-epiglottic folds; the interior of the larynx is less often attacked and the cords usually escape. The infiltration is composed of tiny red nodules, which develop the typical "apple-jelly" centre and break down to form multiple coalescent shallow ulcers, the smooth base covered by a scanty secretion and with indefinite uninfamed margins. Cicatricial contraction goes on during the progress of the affection, and the epiglottis, if not destroyed, is usually much deformed; but the scars are less thick, and the contraction less severe than in syphilis, and marked stenosis is less common.

Treatment.—The disease shows a decided tendency to spontaneous cure, and in many cases of cutaneous lupus the scars of healed disease can be seen in the larynx. Open-air treatment, as carried out in a sanatorium, with good food, moderate exercise, and cod-liver oil suffices to cure most cases. Arsenic, in large doses, appears to have a specific effect, starting with 5 minims of liquor arsenicalis 3 times a day and increasing the dose gradually to 15 or more minims. Local treatment should be reserved for those cases which general measures fail to cure. If the lesions are confined to the epiglottis, this may be removed; for more diffuse infiltration repeated galvanocautery puncture gives the best results, but over-zealous application will promote stenosis. Good results have been reported from the use of radium, applied externally to the neck in the form of plates.

TUBERCULOSIS

Ætiology.—Primary tuberculosis of the larynx is so rare as to be of no practical importance; in the overwhelming majority of cases the disease is secondary to pulmonary tuberculosis, of which it is a common and important complication. It is probably caused by infection from the sputum, is two or three times commoner in men than in women, and is most frequent between the ages of 20 and 40. St Clair Thomson finds that the difference in sex-incidence is occupational, and that women working in office and factory are as susceptible as men.

Symptoms.—The disease attacks, in order of frequency, the vocal cords, arytenoid region, inter-arytenoid space, ventricular bands and epiglottis; in general the lumen is invaded before the upper aperture, and the posterior rather than the anterior parts of the larynx. The typical infiltration is finely nodular, pallid and soft in appearance; ulcers are shallow, with a smooth speckly base and pale ill-defined margin. On the vocal cord the disease chiefly attacks the posterior half and especially the vocal process, where ulceration readily reaches the underlying cartilage and may produce a deep

triangular excavation. Thickening in the inter-arytenoid region is common; infiltration of the arytenoids results in typical pale semi-translucent flask-shaped swellings, while the epiglottis appears as a firmer red sausage-shaped mass.

Of subjective symptoms, the hoarseness is very characteristic, the voice being weak and effortless and very different from the raucous voice of syphilis. Cough and expectoration are mostly due to the pulmonary disease and not in any considerable degree to the larynx. Pain on swallowing is common and often very intense; there may also be actual obstruction to deglutition and, in a late stage, entry of food into the larynx. Dyspnoea is rare.

Diagnosis.—Although signs of pulmonary tuberculosis are helpful in diagnosis, it is obvious that any kind of laryngeal disease may occur in a consumptive patient.

From simple laryngitis.—In the earliest stage of invasion tuberculous laryngitis may exactly resemble catarrh, but redness of one cord only is certainly not due to catarrh, and the latter quickly improves under treatment. The swollen arytenoids of cedematous laryngitis are less pale and more transparent, while the affection is acute and the entire larynx inflamed. Inter-arytenoid infiltration resembles pachydermia, but the latter is opaquely white, firm and symmetrical.

From lupus, typical tuberculosis differs completely. The former is painless, affects first the epiglottis and upper aperture, is never accompanied by cedema, and tends to cicatrization. But there is a chronic "lupoid" form of tuberculous laryngitis which attacks the epiglottis and is very similar to lupus.

From syphilis.—The tuberculous ulcer has an ill-defined margin without surrounding hyperæmia; the base has a yellow speckled appearance, and on healing there is little scarring or contraction. The superficial syphilitic ulcer has a well-defined hyperæmic margin, with a smooth, flat base; the deep ulcer is "crateriform," with thickened punched-out edge, and, on healing, leaves a dense scar and marked deformity. In general, syphilitic lesions attack the anterior half of the larynx, tuberculous the posterior; the former look firm and dense, the latter soft, translucent and ill-defined.

From neoplasms.—Only the rare tuberculomata resemble innocent tumours. Occasionally tuberculosis attacks one vocal cord in an elderly patient, and may then easily be mistaken for epithelioma (see p. 1111).

Prognosis.—Any tuberculous lesion of the larynx renders the prognosis of a case of pulmonary tuberculosis much more serious. A considerable number of the superficial lesions become healed; but it is doubtful if any cases of extensive infiltration recover, with the exception of a few rare instances where it is confined to the epiglottis and can be entirely removed.

Treatment.—Tuberculous laryngitis is but a complication of pulmonary tuberculosis, and by far the most important part of the treatment is that of the general infection. For the laryngeal lesions the most valuable remedy is complete silence, but it is a severe and depressing measure and should not be insisted on unless there is a prospect of cure; the pain and irritation

in advanced cases are, however, often relieved by vocal rest. Any concomitant catarrh should receive attention; an oily spray containing menthol and chlorbutol (7 grains of each in an ounce of liquid paraffin) is valuable, and irritable cough should be relieved by a simple lozenge, or, if severe, by heroin, $\frac{1}{12}$ gr. or less in a lozenge or linctus. Attempts to cure by active local treatment must only be made when the pulmonary lesions are quiescent or progressing towards arrest, the general health good, and the local lesions not very extensive. Of these methods the galvano-cautery is the most generally useful, and may be employed to the surface of superficial ulcers, or as multiple puncture of infiltrated areas. Chemical caustics may be applied to ulcerated surfaces, especially on the cords and posterior commissure; lactic acid, 50 to 80 per cent., may be used, or Lake's mixture of lactic acid 50 per cent., formalin 7 per cent., and phenol 10 per cent. Ulcers covered with sprouting granulations may be curetted, and occasionally infiltration of the epiglottis or arytenoid may be removed with punch-forceps.

In advanced cases the dysphagia is so distressing that its relief is of great importance. For this purpose the most valuable drug is orthocaine (orthoform), which may be combined in equal proportions with benzocaine (anæsthesine); it is an insoluble non-toxic powder and is used as an insufflation in doses of 3 to 5 grains half an hour before meals; patients readily learn to inhale it into the throat through a glass tube. Cocaine and morphine should be reserved to the last stages. When the dysphagia is due to infiltration of the epiglottis, the greatest relief is afforded by its removal under cocaine with special punch-forceps; and when the pain is caused by a tense swollen arytenoid, the removal of a piece with punch-forceps relieves tension and gives similar relief. Injection of alcohol into the superior laryngeal nerve is a useful method of alleviating pain in cases of extensive disease. Tracheotomy is seldom required, and tuberculous infection of the wound is common after this operation.

TUMOURS

INNOCENT TUMOURS

Singer's nodules are inflammatory epithelial thickenings, and may be considered as a form of pachydermia. They are found in teachers and speakers, rather than in singers, and are caused by faulty voice-production and, especially, by forcing the voice when the cords are inflamed. The appearance is that of a minute pink or whitish nodule on the edge or upper surface of the vocal cord, surrounded by a varying amount of injection; there is usually a nodule symmetrically placed on each cord, but it is frequently larger on one side than on the other. The place where the growth occurs, and which is also the "seat of election" for other innocent tumours, is at the junction of the anterior and middle thirds of the glottis, that is, in the centre of the true vocal cord, for the posterior third of the glottis is cartilaginous. It is here that the cord, if swollen, comes into contact with its fellow on phonation.

Fibromata occur usually on the vocal cord on the same site as the singer's nodule, of which they are in some cases probably a development; or at the

anterior commissure. They are pedunculated, smooth and round, white, pink, or brown from extravasation of blood, and vary from the size of a pin's head to that of a bean. These growths not infrequently become œdematous, when they appear translucent like a nasal polypus, and have been incorrectly described as *myxomata*.

Papillomata are reddish, papillary, pedunculated growths, and occur anywhere on the larynx, but, when single, generally occupy the "seat of election" on the vocal cord, and are seldom found on the posterior half of the glottis. Multiple papillomata occur especially in childhood and, as they present special characteristics, will be considered separately later.

Cysts.—A fibroma may degenerate with the formation of a cystic space in its interior. Apart from this, cysts, which may reach a large size, are found as a rarity on the upper aperture of the larynx, especially on the anterior surface of the epiglottis. They are thin-walled and translucent, with ramifying vessels running over the surface.

Angiomata occur, though rarely, on any part of the larynx either as a flat patch or a raised purple mass resembling a blackberry.

All innocent neoplasms are rather uncommon; in addition to those already mentioned, *lipomata*, *chondromata* and *thyroid-gland tumours* have been observed.

Symptoms.—A tiny growth situated on the vocal cords, or in such a situation as to prevent their approximation, causes hoarseness and a variable amount of aching and discomfort, but even a large tumour elsewhere may cause no symptoms to attract attention. Dyspnœa results in rare cases when a neoplasm is large enough to block the air-way, but it is astonishing how slight a disturbance may be produced by a large tumour if it has grown slowly; inspiration is more difficult than expiration, except when the tumour lies below the glottis. Angiomata cause hæmorrhage, which may be very severe.

Diagnosis.—This is usually easy on inspection, but a growth on the anterior commissure, or one that drops down below the cords, may be readily overlooked. The rare tuberculoma may so exactly imitate an innocent neoplasm as to be only recognisable under the microscope. The important matter of diagnosis from a malignant neoplasm will be referred to under the latter disease.

Treatment.—Tumours situated away from the glottis and causing no symptoms should be left alone. Cysts are treated by making a large hole with punch-forceps or the cautery, for they refill after simple incision. Angiomata, especially if diffuse, are best left untouched, unless bleeding calls for interference, in which case it can usually be checked by the cautery at dull-red heat; diathermy puncture with a fine terminal through a direct laryngeal spatula, or by suspension-laryngoscopy, is a preferable method; if repeated hæmorrhage becomes a danger, the angioma can be excised by an external operation, but is usually more widely spread than appears on laryngoscopic examination. Singer's nodules, if quite small and sessile, frequently disappear under prolonged rest of the voice; the smaller nodules may be lightly touched with the galvano-cautery; larger ones should be removed with forceps. Fibromata and papillomata are removed with forceps; it causes less disturbance to the patient if this be done under cocaine anæsthesia by the "indirect method," *i.e.* under guidance with the laryngeal mirror

provided that the operator has acquired the necessary skill, but since the introduction of the "direct method" they are usually removed through the tube-spatula. After the little operation, absolute rest of the voice should be enjoined for a few days; in cases of singer's nodule a longer rest is necessary, with training in voice-production and avoidance of dust and conditions of vocal strain.

Multiple papillomata.—This rare but serious condition occurs almost exclusively in children and generally attracts attention between the ages of 2 and 4. The warts may be very numerous, fill the lumen with a cauliflower-like mass, and extend to the subglottic region, down the trachea and sometimes on to the pharynx. The first symptom is hoarseness, and long-continued hoarseness in a child should suggest the possibility of papilloma; dyspnoea ensues later and becomes gradually more severe until tracheotomy is necessary. The growths may disappear after tracheotomy, or spontaneously, or after an acute illness, and tend to vanish or cease to recur about puberty. The growths should be removed through the tube-spatula, or by suspension-laryngoscopy, under general anæsthesia; a preliminary tracheotomy is advisable if there is much dyspnoea; several sittings may be required, and the operation must be repeated as often as the growths recur.

MALIGNANT TUMOURS

Ætiology and Pathology.—Epithelioma is by far the commonest malignant growth in the larynx, but spheroidal-cell carcinoma and sarcoma also occur. Malignant disease of the larynx proper is rare below the age of 40, and is seldom seen in women; but there is a post-cricoid epithelioma, originating on the mucous membrane of the pharynx, which is relatively common in women, and has been known to occur at such an early age as 23. Secondary or metastatic growths are practically unknown in the larynx; on the other hand, owing to the fact that the laryngeal lymphatics do not anastomose freely with other systems, cancers confined to the lumen of the larynx rarely become disseminated and do not readily infect the glands. Therefore, Krishaber's classification into *intrinsic* and *extrinsic* is valuable for treatment and prognosis; the former are those situated within the cavity of the larynx, while the latter affect the upper aperture, epiglottis, arytenoids, and the outer walls.

Symptoms.—Unfortunately, intrinsic malignant tumours do not cause severe symptoms at an early stage and, particularly in hospital practice, frequently do not present themselves for treatment until they have become extrinsic by extension; hence the importance of a laryngeal examination in all patients over 40 with hoarseness which does not rapidly yield to treatment. Hoarseness is the most general, and usually the only, early symptom; owing to the deep infiltration characteristic of malignant disease, it is often more marked than the size of the tumour would appear to warrant. Cough is not a common symptom. Pain is absent in the early stages of intrinsic cancer, but is severe in the later stages and in the extrinsic forms; it radiates to the ear and side of the head, and is made worse by swallowing, speaking and coughing. The later symptoms include involvement of the glands, fetor of the breath, salivation, hæmorrhage, dyspnoea, dysphagia and general cachexia; often the patient dies of septic pneumonia.

On a vocal cord, epithelioma may appear as a definite tumour resembling a papilloma or angioma, or it may begin as a diffuse infiltration, or merely as a localised area of thickening and congestion. On the ventricular band or posterior commissure it usually shows itself as an irregular papillary dusky-red swelling; cancer of the epiglottis ulcerates early and appears as a dirty white or reddish tumefaction. Often the only sign of a post-cricoid growth is a swollen and fixed arytenoid, or merely a pool of saliva in the pyriform fossa; sometimes the upper edge of the growth is visible, but it may be necessary to pass a tube-spatula, or to pull the larynx forward with a probe passed down to the glottis, before it can be seen.

Diagnosis.—In the earliest stage the diagnosis obviously is a matter of extreme importance and sometimes one of great difficulty. The unilateral character of the infiltration is ordinarily sufficient to exclude a simple inflammatory lesion; though pachydermia may be more marked on one side, the lesions are bilateral and the smooth cup-shaped swelling on the vocal process is characteristic. The difficulties of diagnosis are generally between an innocent neoplasm on the one hand, and a tuberculous or syphilitic infiltration on the other. A papilloma or a fibroma on a vocal cord should be regarded with suspicion in a man over 40, and especially if, after removal, the site fails to heal promptly. An innocent neoplasm has a fine pedicle, moves freely in the air-current on phonation, there is no tumefaction at its site of origin, and it is found at or in front of the middle of the vocal cord. A malignant growth may occur in any situation; it is less movable and pedunculated, the cord in the neighbourhood is frequently swollen and may show a leash of tiny vessels running to the tumour; a white spiky appearance of the papillæ is suggestive of malignancy; a sluggish delayed movement of the cord is an important sign, insisted on by Semon, but its absence by no means excludes malignancy, for it only occurs when the growth has infiltrated the muscles, and its presence increases the gravity of the prognosis. Epithelioma beginning as a flat infiltration may resemble a tuberculous or syphilitic lesion, but usually other signs of these diseases are present to aid the diagnosis; in the latter the Wassermann reaction and, still more, the effect of vigorous anti-syphilitic treatment will clear up the doubt. There is a form of senile tuberculosis in which infiltration of one vocal cord may closely imitate epithelioma, more especially as the pulmonary signs are merely those of bronchitis and tubercle bacilli are very scanty in the sputum. Sometimes a piece may be removed for examination, but frequently the growth is too sessile to permit it; the piece removed must not be very small, and a negative finding is of little value, for the tip of a malignant growth cannot always be distinguished microscopically from a papilloma. Biopsy is more important since the adoption of Broders' histological method of estimating the malignancy and radio-sensitivity of tumours, but it should not be performed in cases too advanced for treatment, for it stimulates the growth and does harm.

Extrinsic epithelioma is most likely to be mistaken for tertiary syphilitic ulceration. A gumma grows and ulcerates more quickly, its edge is smoother and sharper, and its base often covered by a yellow slough. Pain has little diagnostic value, for a gumma of the upper aperture may cause severe dysphagia. Palpation with the finger is of great service, for the firm hard feel of an epithelioma is very characteristic.

Treatment.—Malignant disease cannot be removed with certainty by

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the natural passages. Thyrotomy, or laryngo-fissure, is the operation of choice for removal of intrinsic cancer ; it consists essentially in wide excision of the disease from the interior of the larynx after splitting the thyroid cartilage in the middle line. The results in suitable cases are probably better than those of removal of malignant disease in any other part of the body ; there is now practically no immediate mortality in skilled hands, statistics show from 70 to 80 per cent. of permanent cures, and a useful husky voice remains. When, however, the growth has spread at all extensively to the arytenoid or to the subglottic region, laryngo-fissure is no longer possible and a very different picture is presented. By modern methods of laryngectomy the trachea is fixed in the lower part of the neck, aspiration of septic matter is avoided, and the mortality has been much reduced, while the results as regards freedom from recurrence have been greatly improved. The operation leaves the patient in a condition very different from that after thyrotomy ; as he breathes through the tracheal opening in the neck he cannot cough or strain and has no natural voice. Nevertheless he can often produce a fairly audible whisper by means of the air in the pharynx, or with the aid of a tube leading from the tracheotomy wound into the mouth or nose, and a number of patients succeed in passing a surprisingly happy and useful existence. Subhyoid pharyngotomy and lateral pharyngotomy are operations designed to obtain access to cancers at the upper aperture of the larynx ; the latter operation gives very good results in strictly limited tumours of this region. The technique of therapy by means of X-rays and radium has now been put on a reliable basis by determination of the appropriate dosage and by adequate screening ; in many cases of malignant tumours of the larynx, great improvement can be obtained by both these agencies, and a considerable proportion of cures has been effected. A valuable method, in intrinsic cancer, is the insertion of radium tubes through a window cut in the thyroid ala. Great care and judgment are required in the selection of cases for these various operations, and very many come under observation too late. In these much can be done by palliative treatment ; careful attention to the hygiene of the mouth and teeth is of great importance, together with mild antiseptic laryngeal sprays containing hydrogen peroxide, listerine, sanitas or phenol. For dysphagia, insufflations of powdered orthocaine, with or without benzocaine, is the most reliable remedy ; cocaine is disappointing, for its effect is very transient and the resulting numbness interferes with swallowing ; the judicious administration of morphine or heroin is eventually necessary, but the local application of these drugs is useless. Palliative tracheotomy or gastrotomy may be called for, and curettage or partial removal by knife or diathermy gives relief in some cases of cancer at the upper aperture.

PARALYSIS

Paralysis of a vocal cord is a frequent symptom of various diseases of the thorax and of the nervous system, and the laryngoscope is therefore of great value to the physician as an aid to diagnosis ; this is more especially the case in that the common early form, abductor paralysis, causes no symptoms, and can only be recognised by laryngoscopic examination.

The original function of the laryngeal muscles is that of a sphincter to

prevent the entrance of fluid into the lungs, and this sphincter, or adductor, is the only muscle present in the larynx of the most primitive air-breathing animals; the abductors are a later addition, to assist the entry of air. Accordingly, in lesions of the nerve path, the abductor muscles are affected first and the primitive adductors are more resistant. But the function of phonation, much more recently acquired, is associated with adduction, and is under direct control of the will. Functional disturbances, therefore, always cause adductor paralysis, while organic lesions first affect the movement of abduction.

ORGANIC PARALYSIS

The crico-thyroid muscle is supplied by the superior laryngeal nerve, and, when this is injured, the affected cord remains slack on phonation, but, owing to the short course of the nerve, isolated paralysis of this muscle is extremely rare; it results from surgical or suicidal wounds, or by pressure from glands, but most often occurs after diphtheria. In lesions of the vagus above the origin of this branch the signs of this paralysis are obscured by that of the other muscles of the cord. The recurrent laryngeal nerves supply all the other muscles. In any progressive lesion of the nerve-path the muscles become paralysed in a definite order, the enunciation of which is known as Semon's law; the abductors are first affected, then the tensors or thyro-arytenoidei, and finally the adductors.

In *abductor paralysis* the affected cord lies in the middle line; during phonation the sound cord adducts to meet it and the larynx appears normal, but on inspiration it is drawn outwards and backwards and appears longer than its paralysed fellow, which remains unmoved. As would be expected from the course of the recurrent nerves, the left cord is far more often affected than the right. The voice is unaltered, but, although the glottic aperture is reduced by half, there is usually dyspnoea only on exertion, except in children, in whom the narrowing of the naturally small glottis may produce sufficient obstruction to necessitate a tracheotomy.

When the thyro-arytenoid fails, the edge of the cord is concave on phonation, the cord appears narrower than its fellow, and the voice gradually becomes husky. Finally, when *total recurrent paralysis* has occurred, the cord assumes the "cadaveric position" between the middle line and the normal position of rest. On phonation, the healthy arytenoid crosses the middle line and pushes the paralysed cartilage aside; sometimes the latter drops forward and exposes its broad posterior surface, which may be mistaken for a swelling. As the cords are still able to approximate, the voice is not necessarily lost, but is hoarse and easily tired, with a characteristic breathy quality from waste of air, or a diphonic character, due to unequal vibration of the two cords.

In cases of *bilateral abductor paralysis* the cords lie together near the middle line. The voice is good, but the inability to take a full breath gives the speech a peculiar character; dyspnoea is a marked symptom accompanied by inspiratory stridor and severe paroxysmal exacerbations. When the disease progresses to complete *bilateral recurrent paralysis*, both cords remain in the cadaveric position, the dyspnoea becomes less severe but the voice is reduced to a whisper.

Diagnosis.—The diagnosis is almost entirely a matter of accurate

inspection. Obliquity of the laryngoscopic image, due to faulty position of the mirror, may cause confusion. In nervous subjects the cords are sometimes adducted on inspiration, but they will abduct naturally during the involuntary inspiration which follows a prolonged phonation. The only condition which really imitates paralysis is the fixation of the arytenoid cartilage which results from disease in or around the joint; its complete immobility with the presence of swelling or scarring often aids the diagnosis, but in old-standing cases of paralysis secondary fixation frequently occurs.

Ætiology.—The ætiology is of importance, for it is on our know-

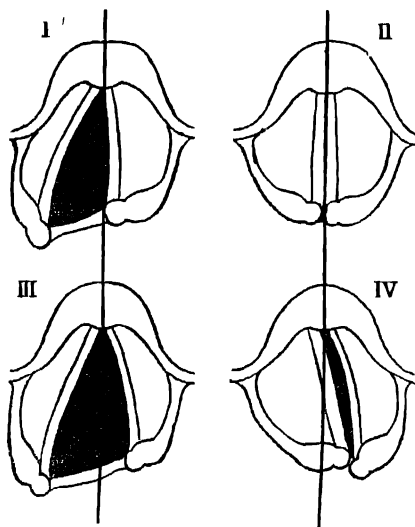


FIG. 97.—Organic Paralysis: I. Abductor paralysis of left cord on inspiration; II. Abductor paralysis of left cord on phonation; III. Total paralysis of left cord on inspiration; IV. Total paralysis of left cord on phonation. (*Lancet*.)

ledge of their causation that the diagnostic value of these lesions depends. The movements of the cords are represented bilaterally in the cortex cerebri, and stimulation of either centre produces movement (adduction) of both cords, from which it follows that no unilateral lesion above the bulbar nuclei can paralyse the larynx, and clinically we find that it is never affected in cases of hemiplegia. The bulbar centres lie in the floor of the fourth ventricle, and here a lesion of one centre causes paralysis of the cord on the same side which, in a gradually progressive lesion, affects first the abductor muscle. Thence the nerve fibres pass in the roots of the bulbar-accessory to the vagus and recurrent laryngeal nerve; the cause of the paralysis may, therefore, be situated (1) in the medulla, (2) at the base of the skull, (3) in the vagus, or (4) in the recurrent laryngeal nerve.

Paralyses of bulbar origin are often, but by no means always, bilateral. In lesions here and at the base of the skull neighbouring nerves are liable to be involved; thus, paralysis of a cord and of the same side of the palate may coexist (syndrome of Avellis), or paralysis of cord, palate, trapezius and sternomastoid from involvement of the spinal accessory roots, or persistent frequency of the pulse due to damage of the cardio-inhibitory centre or nerves. *Tabes dorsalis* is the most frequent cause of paralysis of central origin; it may affect one or both cords and may be associated with anæsthesia, paræsthesia or the spasmodic attacks called "laryngeal crises." In general paralysis of the insane laryngeal palsy is not uncommon. It is the rule in bulbar paralysis, and is usually bilateral, but appears late in the disease.

Syphilitic nuclear disease, pachymeningitis and gummata at the base of the brain are frequent causes, and here the ocular muscles, especially the external rectus, are often attacked.

Peripheral causes usually act by compression of the recurrent nerve, the most frequent being aneurysm, enlarged glands, tuberculous or malignant, and cancer of the œsophagus. Other causes are thyroid tumours, usually but not necessarily malignant, mediastinal tumours, cancer of the lung, pleurisy, and pulmonary tuberculosis in which the nerve, usually the right, may be involved in a lesion at the apex of the lung or by tuberculous bronchial or tracheal glands. Neuritis is a cause of laryngeal paralysis; it may be produced by the toxins of the infectious fevers, usually diphtheria, or by organic poisons, especially lead, and more rarely arsenic and alcohol. Finally, the condition is sometimes the result of traumatism, more especially surgical operations on the thyroid gland and œsophagus.

Prognosis.—Paralysis of one cord is not in itself dangerous to life; but when the cause is undiscovered the prognosis must be guarded, for this paralysis may be for a long time the only sign of serious disease; on the other hand, the recurrent nerve may be involved in some non-progressive lesion, such as a fibrotic bronchial gland, and such cases have been under observation for 20 or 30 years without change.

Treatment.—This depends on the cause. In most cases it is but a symptom of disease elsewhere and does not call for special treatment. In traumatic cases, however, the nerve may sometimes be found and sutured; afterwards, and in cases due to neuritis, strychnine and the local application of the faradic current by means of an intra-laryngeal electrode are indicated. Tracheotomy is advisable in bilateral abductor paralysis, but a plug may usually be worn in the tube, to be removed at night and whenever dyspnoea threatens.

FUNCTIONAL PARALYSIS (FUNCTIONAL APHONIA)

Ætiology.—Functional aphonia is a common manifestation of hysteria, and has been a very frequent symptom of war-neurosis or "shell-shock," but it should be clearly stated that the majority of cases are not purely hysterical. Anything which increases the effort of phonation, such as debility or laryngeal catarrh, predisposes to this affection, which is characteristic rather of feeble neuro-muscular tone than of hysteria; this explains how some women lose the voice completely with every slight cold, while other patients can produce a loud if hoarse voice with severe laryngeal disease.

Symptoms.—Paralysis of the adductors presents a totally different clinical picture from the organic paralysis. It is always bilateral; the larynx appears normal while at rest, but, on attempts at phonation, it is seen that the cords do not adduct into the position necessary for the production of the voice. Very commonly the thyro-arytenoidei are the only muscles which fail to act; the cartilaginous glottis is then properly closed, but an elliptical chink is left between the cords. If the crico-arytenoidei laterales are paretic, the entire glottis remains open to a variable extent, and, very rarely, the arytenoideus is affected alone, when a triangular aperture is left behind the vocal processes. The paralysis is

hardly ever complete; indeed a considerable amount of movement is usually seen, though insufficient to produce phonation. In purely hysterical cases the onset and recovery are sudden, the cough is usually not aphonic and the voice when regained is not hoarse. In some hysterical patients there is also inability to whisper.

Treatment.—In patients suffering from debility the cause should be

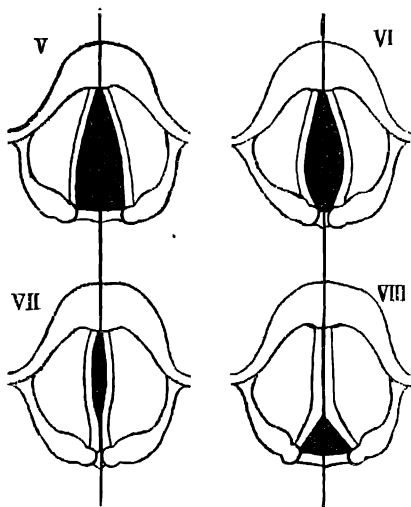


FIG. 98.—Functional Paralysis; all during attempted phonation; V. Paresis of all the adductors; VI. Arytenoides still active; VII. Paresis of internal tensors; VIII. Paresis of arytenoides. (*Lancet*.)

is no serious disease, but that he is not that he can produce a good voice quite or tongue-depressor is in position. With a little elementary instruction in voice-production this is usually successful, the patient's confidence is restored and the voice does not again fail; in obstinate cases some perseverance in lessons on production is required. These methods have been extraordinarily successful with shell-shocked soldiers, but it must be confessed that such certain and rapid results are not always obtainable in nervous women.

found and treated; chronic phthisis is such a common cause of functional aphonia that it should always be thought of in this connection. There is frequently a slight degree of laryngitis and in some of these cases the failure of adduction is "myopathic," or due to inflammation of the muscles; in these the local condition must receive appropriate treatment. When the larynx is normal the voice can nearly always be temporarily restored by any powerful local stimulation, such as the intralaryngeal application of chloride of zinc, or any similar drug, or of the faradic current; but the aphonia usually recurs again, and succeeding applications are less effective, so that the most difficult cases to cure are those who have had much local treatment. Far better results are obtained by moral suasion, explaining to the patient that there

using his muscles correctly, and easily when the laryngeal mirror or tongue-depressor is in position. With a little elementary instruction in voice-production this is usually successful, the patient's confidence is restored and the voice does not again fail; in obstinate cases some perseverance in lessons on production is required. These methods have been extraordinarily successful with shell-shocked soldiers, but it must be confessed that such certain and rapid results are not always obtainable in nervous women.

SPASMODIC AFFECTIONS

SPASM OF THE GLOTTIS

Spasm of the laryngeal muscles produces adduction of the cords, for, though the abductors are probably affected, they are overpowered by the stronger adductor muscles.

Ætiology.—(1) In the majority of cases the spasm is a reflex set up by local irritation : foreign bodies, including the laryngoscopic mirror, irritating gases ; inflammation, ulceration or tumours in or near the larynx, children being much more liable than adults to spasm from local irritation. (2) Spasm is also caused by irritation of the recurrent laryngeal nerves by enlarged glands, mediastinal tumours and, especially, by aneurysm. (3) Central nervous lesions, especially tabes. (4) Functional disturbances, frequently hysterical, often associated with globus hystericus, and sometimes excited by sexual disturbances.

Symptoms.—The attacks vary much in different subjects in severity and duration. The patient is usually aware of its onset, and clutches some support or rushes to the window. The respirations are rapid and shallow, with loud inspiratory stridor, and, in the height of a severe attack, are completely arrested with all the signs of asphyxia. The subjective sensations include a horrible feeling of anxiety, but consciousness is not lost. Many cases are less acute but persist longer, even for several hours.

Prognosis.—The attacks are practically never fatal, unless a foreign body or tumour be present.

Treatment.—During the attack amyl nitrite or chloroform may be inhaled, and ampoules of these drugs should be kept on hand. Between the attacks sources of irritation should be sought for and removed, the upper air-passages brought to a healthy condition, and the general health and mode of life should receive attention. Administration of bromides may be required when the attacks recur frequently.

LARYNGISMUS STRIDULUS

Ætiology.—This is a condition, clinically similar to glottic spasm, occurring in children. It is far commoner than the spasm of adults, and it has been suggested that the asphyxial attacks of laryngismus are caused by collapse of the soft and yielding cartilaginous framework of the larynx, and not solely by spasm of the muscles. It is commonest between the ages of 6 months and 2 years, but may persist later ; it occurs in ill-nourished, unhealthy children, usually in association with rickets, and practically always in association with adenoids.

Symptoms.—The onset is sudden and usually at night. The child wakes gasping for breath, and a series of short noisy inspirations is followed by complete cessation of breathing and terminated by a long, crowing inspiration. There are retraction of the lower ribs and epigastrium, cyanosis and great terror and distress and, in severe cases, carpo-pedal contractions, convulsions and evacuation of urine and fæces. When the attack is over the child is perfectly normal and there is no hoarseness. Slighter and less typical attacks often occur.

Diagnosis.—This is easy if the symptoms are carefully noted ; the sudden attack of dyspnoea, with complete absence of symptoms in the intervals, is quite distinctive.

Prognosis.—The prognosis is somewhat grave in severe cases ; an infant rarely dies in an attack, but is often worn out and succumbs to collapse of the lungs.

Treatment.—During the attacks the face and chest may be freely

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sponged with cold water, and the inhalation of amyl nitrite from a capsule broken in a handkerchief may be tried. The quickest relief can usually be obtained by drawing the tongue forward with a finger passed into the mouth to its base, a manœuvre easily performed by the mother or nurse. The attacks are so short and sharp that there is no time for the hot bath or administration of bromides frequently recommended.

Prevention involves general tonic treatment, fresh air, wholesome food and correction of digestive disturbances. The removal of adenoids is of great importance, even if not large enough to be definitely obstructive, as is the treatment of naso-pharyngeal catarrh with the usual saline lotion which, in small children, may be dropped into the nostrils from a pipette. Bromides are to be avoided if possible as depressing, but 10 to 30 drops of liquid extract of *grindelia* may be given 3 or 4 times a day in milk or sweetened water as recommended by Eustace Smith.

CICATRICAL STENOSIS

Ætiology.—Suicidal and other wounds, gunshot injuries and scalds may produce cicatricial narrowing of the lumen of the glottis. After thyrotomy a web may form across the anterior commissure. In the haste of an emergency tracheotomy, the wound has often been made too high and the cricoid cartilage cut through; in these cases it generally happens that, after the subsidence of the acute condition, dyspnœa follows every attempt to remove the tube, and a stenosis is found to have resulted from swelling and narrowing in the subglottic region. Similarly, a proportion of cases intubated for diphtheria are unable to breathe without the tube, by reason of a subglottic stenosis. Lupus and tuberculosis of the larynx can produce cicatricial stenosis, and especially when the cautery has been extensively employed in treatment. Syphilis is the most fruitful cause of this condition, and the great difficulty of obtaining a cure at this stage is a powerful reason for early and thorough treatment of syphilis of the larynx. Leprosy and scleroma cause stenosis, but are rarely seen in this country. The perichondritis which is an occasional complication of enteric fever, small-pox and diphtheria commonly ends in severe stenosis.

Symptoms and Diagnosis.—The principal symptom is naturally dyspnœa, but in chronic cases it is remarkable how great may be the narrowing before dyspnœa becomes severe. The obstruction and the stridor are most marked on inspiration, in contra-distinction to tracheal stenosis where the stridor is both inspiratory and expiratory. The larynx moves downwards with each inspiration; this "respiratory excursion" of the larynx is a further diagnostic sign of laryngeal obstruction, but is not always present. The patient sits upright, with the head thrown back; whereas in tracheal obstruction he bends the neck forward to relax the trachea.

Treatment.—In all cases with decided dyspnœa tracheotomy should be first performed, and in syphilitic cases it is important that all active disease should be arrested by thorough treatment before any attempt at dilatation be begun, and the stenosis itself will often be greatly improved by such treatment. The administration of iodides is dangerous in these cases, for the increased secretion is pent up behind the stenosis and may overwhelm

the lungs. Difficulty in dispensing with the tube after tracheotomy is sometimes due to nervousness on the part of the child, and can then be surmounted by using a fenestrated tube which is plugged occasionally and by encouraging the patient to breathe through the mouth by blowing soap-bubbles or sounding a whistle. When the tracheotomy wound is too high, a low tracheotomy should be performed and the original wound allowed to close; this is often sufficient to overcome the difficulty.

The successful treatment of severe cicatricial stenosis demands the greatest skill and perseverance on the part of the surgeon as well as the patient co-operation of the sufferer. The whole circumstances of the case should be carefully considered before advising difficult and prolonged treatment. Adult patients can live active lives with a permanent tracheotomy opening, which is not so serious a disability as it is generally considered to be. If the stenosis be not too extreme, a fenestrated tube may be worn which can be kept plugged during the day, so that the patient may have the use of speech and respiration by the natural passages. In children and young people a permanent tracheotomy is more harmful, but the prospect of cure by dilatation is better; the best method of dilatation is by the use of intubation tubes.

HAROLD S. BARWELL.

DISEASES OF THE TRACHEA

INFLAMMATION OR TRACHEITIS

ACUTE TRACHEITIS

Acute tracheitis may occur from any condition leading to irritation of the mucous membrane of the trachea. When it occurs as a result of bacterial or chemical agency, the whole of the upper air-passages are usually involved in greater or less degree, and the clinical manifestations are not confined to the trachea. In some cases, however, the stress of the resultant reaction falls upon this tube, and the condition therefore requires separate consideration.

Ætiology.—1. *Microbic invasion.*—This is the commonest cause. The bacteria usually found associated with tracheitis are the so-called catarrhal organisms, such as the *Micrococcus catarrhalis*, the pneumococcus, the Freidländer pneumo-bacillus and Pfeiffer's *H. influenzae*. It is probable that the primary organism in many cases is of filter-passing type. Frequently a member of the streptococcus group may be found, either alone or in association with one or more of those just mentioned. As with catarrhal inflammation of other parts of the upper air-passages, damp, cold or foggy climatic conditions predispose to tracheitis. It is more common in young and middle-aged adults than in infancy or in old age. Mouth-breathers are more liable to this condition. Exposure to sudden changes of temperature may be a factor in its onset.

Tracheitis may also occur as part of the clinical picture in some of the acute specific diseases, such as enteric fever, diphtheria, whooping-cough

and measles. It is often a troublesome and distressing association or sequel of true influenza.

2. *Chemical agencies.*—Irritating or poisonous fumes and vapours may lead to a very acute form of tracheitis. It may, therefore, occur in certain occupations, unless adequate precautions are taken. The use of "poison gases" in warfare has drawn widespread attention to this form of the condition, since tracheitis was an almost constant result of certain forms of "gassing." The chief chemical irritants used in the War of 1914-1918 were chlorine, phosgene and yperite, or dichloroethyl sulphide, commonly known as yellow cross or mustard gas. Of these the last was perhaps the most irritant to the trachea, and fatal cases invariably showed tracheal lesions. Direct inhalation of steam may also induce an acute tracheitis.

3. *Mechanical causes.*—The presence of a foreign body, or the invasion of the trachea by extension from malignant growth in adjacent structures may lead to a local or even to a general tracheitis. It is noteworthy, however, that the trachea is frequently spared in occupations involving the respiration of dusty air, which leads to deposits in the lungs and bronchial glands with resulting pneumonokonioses. Although a coal miner's lungs are black, yet his trachea may be practically normal.

Pathology.—The changes found in the trachea vary from simple catarrhal inflammation to intense destructive changes with ulceration, and in some cases croupous or membranous exudate. In the catarrhal forms, the mucous membrane shows changes similar to those in bronchitis. It is at first swollen, red and dry, the vessels running across the trachea being engorged and clearly visible. Then, owing to increased activity of the mucous glands, excessive mucoid secretion occurs and the mucous membrane becomes moist, after which resolution may take place, or the process may proceed to a mucopurulent stage, when the fluid on the membrane coheres to form yellowish or green tenacious pellets. Occasionally numerous red blood cells are extruded and the tracheal exudate becomes streaked, tinged or uniformly pinkish.

In some inflammations, such as those induced by poison gases or inhaled steam, the mucous membrane may be intensely engorged and actual destruction may occur, involving even the deeper structures and the cartilages, so that greyish yellow sloughs result, which on separation leave ulcers. In diphtheria the characteristic false membrane composed of necrosing fibrin, leucocytes and bacilli may be found loosely attached to the mucous membrane, as in other localisations of this process. It may be primary or secondary to faucial or laryngeal diphtheria, either by direct extension or through diphtheritic infection of a tracheotomy wound.

In influenza the pink appearance of the trachea is of such constancy in fatal cases that it has come to be regarded as one of the most characteristic post-mortem changes found in this disease. The bright injection generally involves the lower half of the trachea, but it may occur along the whole length of this tube.

In whooping-cough the inflammatory reaction is usually less acute.

In typhoid fever small ulcers may occasionally be found in the trachea similar to those occurring more commonly in the larynx.

Symptoms.—Acute catarrhal tracheitis usually begins more or less acutely, like the common "cold," of which it is to be regarded as one form, with malaise, slight headache, and a mild degree of fever, the temperature

being usually between 99° and 100° F., rarely 101° F. The patient soon experiences a sensation of irritation behind the sternum, rapidly leading to a harsh, dry cough of noisy character. The cough aggravates the retro-sternal discomfort, which develops into a sensation of rawness or soreness, making the cough very painful and distressing. If the larynx is involved at the same time, the voice becomes hoarse and sometimes lost, or reduced to a raucous whisper. In tracheitis alone the voice is usually unaffected. After from 12 to 24 hours the condition passes into the mucoid stage. The cough becomes looser and less painful, and small pellets of tenacious mucus are coughed up, usually greyish or black in town-dwellers, whitish in those in rural conditions; in either case, the mucus may be streaked with blood or even tinged a uniform pink colour; in the more acute forms it sometimes becomes yellow and more purulent. In the mucoid stage, the retrosternal soreness becomes less, the constitutional symptoms abate, while the temperature subsides and becomes subnormal. The patient often feels weak and out of health for some days, and is sometimes left with a noisy morning cough and tracheal irritation which may last for days or weeks. The aspect of the patient shows nothing characteristic. There is the general appearance of fever, malaise and discomfort. The rise of temperature and increase in pulse-rate are usually moderate. In the early stages physical examination of the chest shows no abnormality, but when exudation occurs a coarse wheeze may be audible over the trachea, particularly when the patient takes a deep breath or just before a cough occurs.

Diagnosis.—The association of catarrhal symptoms with a dry, harsh cough and retrosternal soreness, without signs of bronchitis, is almost pathognomonic. In some cases the diagnosis can be established with the laryngoscope or by endoscopy, but in most the discomfort which these examinations entail is unnecessary.

Prognosis.—This is almost invariably good, except in debilitated subjects or in those with cardiac or renal disease, in whom the process may spread to the larynx, bronchi or lungs. The usual course is from 2 days to a week, though cough and expectoration may persist for days or weeks. The condition may become chronic. To some extent the prognosis depends upon the care and treatment in the initial stage. Cases that are neglected are liable to become chronic.

Treatment.—The prophylactic and remedial treatment of acute tracheitis is practically identical with that of acute bronchitis of the larger tubes. Even in mild cases the patient should go to bed, though this may be necessary only for 1 or 2 days; but he should keep to his room till his temperature has become normal. There may be less need for expectorants than in bronchitis, and a simple saline diaphoretic mixture, with the addition later of tinct. ipecac. and tinct. opii camphorata, may be all that is necessary. Sedative inhalations, such as vapor benzoini, are useful, and counter-irritation to the sternal region is comforting and grateful to the patient. When a chronic noisy cough develops, a mixture containing small doses of apomorphine and tinct. chloroform. et morphin. co. often gives relief, or codeine linctus B.P.C.

When tracheitis occurs as part of some specific disease, such as diphtheria or influenza, the treatment should be that appropriate to the primary disease.

In "gassing," every effort should be made to relieve the distressing and

painful symptoms, and for this purpose morphine, either alone or in combination with atropine and strychnine, may be required. Various inhalations may be tried, and useless cough should be checked by sedative mixtures or by a linctus of heroin, morphine or codeine.

CHRONIC TRACHEITIS

Ætiology.—Chronic tracheitis may follow an acute attack, or it may develop insidiously in patients suffering from chronic laryngitis or bronchitis. Inhalation of cigarette smoke is a not infrequent cause. It is also sometimes a sequel of chronic rhinitis, especially of the atrophic form or ozæna. A certain degree of chronic tracheitis accompanies the specific lesions of syphilis and tuberculosis, which are described below.

Pathology.—Various degrees of chronic inflammatory lesions may be found. In chronic catarrhal tracheitis, the vessels are distended or engorged, and the mucous membrane of the trachea becomes thickened and more or less covered with mucoid or muco-purulent secretion, the histological changes being those of chronic catarrhal inflammation, namely, shedding of the ciliated epithelial cells, overactivity of the mucous glands, and sometimes thickening and induration of the submucous tissues from proliferative changes. A condition of perichondritis of the tracheal cartilages may, in this case, be observed, and this may result in a mammillated appearance in the internal aspect of the trachea. In ozæna, crusts similar to those in the nose and pharynx may form on the tracheal mucosa.

Symptoms.—The symptoms of chronic tracheitis are similar to those of the acute form. There is a sense of discomfort and irritation about the trachea and a chronic, almost dry cough, often worse in the morning. There is generally some scanty, sticky expectoration, mucoid or muco-purulent, darkened by carbon particles and occasionally blood-tinged.

There are practically no physical signs of this condition, except that the tracheal changes can be observed by the laryngoscope or by endoscopy of the trachea.

Diagnosis.—This is concerned chiefly with its differentiation from chronic changes in the trachea due to syphilis, tuberculosis or leprosy and to the effects of new growths. It must largely be made by endoscopic examination.

Prognosis.—The prognosis depends upon the cause. When this can be removed, as by treatment of predisposing conditions in the nose and throat, the outlook is good. When the tracheitis is due to other conditions, such as syphilis and tuberculosis, it depends upon the situation and extent of the other lesions and upon the treatment adopted.

Treatment.—This is, in its main features, similar to that of acute tracheitis, but climatic treatment may be of great importance. The patient may perhaps spend the winter months in a warm or equable and clear climate with great advantage. Vaccine treatment may also yield good results. When other conditions are concerned, such as ozæna, syphilis or tuberculosis, the treatment appropriate to them should be employed as well.

CYSTS AND TUMOURS

These are rare conditions, but require careful consideration.

CYSTS

Owing to weakening of the wall of the trachea, local bulging may occur, giving rise to a cystic, air-containing swelling in the neck, in direct communication with the lumen of the trachea. Such cysts are known as "tracheoceles" or "aeroceles." They are resonant to percussion and can often be temporarily reduced by pressure.

Small retention cysts may occur in the posterior wall of the trachea, from obstruction of the ducts of the mucous glands as they pass through the trachealis muscle. They are of pathological interest only, and do not give rise to symptoms.

SIMPLE TUMOURS

The most important is papilloma. It occurs chiefly in children and is usually pedunculated. When it grows in polypoid form it may lead to obstruction of the trachea low down, in which case tracheotomy may fail to give relief, and death results unless the tumour can be removed by endoscopic methods.

Other innocent tumours occur, but are rare. They include enchondrosis from localised overgrowth of cartilage, multiple enchondromata, and osteoma from ossification of a pre-existing enchondroma. Lipoma and aberrant thyroid tumours may occur, but are very rare.

Symptoms.—These tumours produce varying degrees of tracheal obstruction, and can usually only be recognised by endoscopy. Treatment is considered under that of tracheal obstruction.

MALIGNANT TUMOURS

A few cases of primary carcinoma of the trachea have been recorded. Secondary growths are not common, but the trachea is often involved and infiltrated by primary carcinoma in adjacent structures, such as the oesophagus, the thyroid, the larynx, or by the extension of secondary deposits in the cervical or mediastinal glands.

Primary sarcoma of the trachea is also very rare. The growth is usually smooth and not pedunculated. Secondary deposits of sarcoma in the trachea may occur from sarcoma of distant organs, such as the kidney; or it may be invaded directly by sarcoma originating in the thymus or other mediastinal structures, and especially by lympho-sarcoma of the mediastinal glands.

Symptoms.—The tracheal symptoms and signs are usually those of obstruction, accompanied by pain. When the primary growth is in the oesophagus, antecedent dysphagia and sometimes laryngeal paralysis reveal the origin of the tracheal symptoms when they occur. In this case copious frothy mucoid expectoration is frequent, and when ulceration develops with perforation, food particles may enter the trachea, excite cough and soon lead to inhalation broncho-pneumonia or gangrene. When the growth is near the bifurcation, urgent dyspnoea is the rule, and spasmodic attacks may

occur, causing extreme distress. In most cases of tracheal growth the characteristic clanging brassy cough (gander cough) of tracheal obstruction can be heard. The trachea may be pushed to one side and its lumen distorted and obstructed by growth in the cervical glands. In mediastinal new-growth invading the trachea, the pressure signs and symptoms characteristic of that disease usually render the explanation of the tracheal symptoms apparent.

Course.—This is generally rapidly progressive.

Diagnosis.—Intratracheal growths have to be differentiated from other causes of tracheal obstruction, and the diagnosis is considered in detail under that condition. Endoscopy affords valuable confirmation if it is practicable or desirable. In œsophageal and mediastinal new-growths invading the trachea, X-ray examination may assist in diagnosis.

Prognosis.—This is hopeless, death occurring from asphyxia or from some complication or by asthenia.

Treatment.—Treatment can be palliative and symptomatic only. In obstruction, it may be possible in rare cases to give temporary relief by a low tracheotomy, but as a rule this is impossible, owing to the presence of obstruction below any point where the trachea is accessible.

THE INFECTIVE GRANULOMATA

SYPHILIS

. The trachea may be affected in both the congenital and acquired forms.

In congenital syphilis, a progressive cicatrisation may occur, leading to stenosis. In acquired syphilis, during the secondary stage, the mucous membrane of the trachea may become generally hyperæmic, or small raised mucous patches may develop locally. In the tertiary period, gummata may occur in the trachea, the commonest site being towards the lower end. Degenerative processes, leading to necrosis and softening, eventually result in ulceration, sometimes with local sloughing of parts of the tracheal rings. In the process of cicatrisation a progressive stenosis may develop.

Symptoms.—Symptoms are those of chronic tracheitis and tracheal irritation in both the secondary and tertiary manifestations, but in the latter, signs of tracheal stenosis may develop when scarring and healing are in progress. Laryngeal involvement occurring at the same time tends to distract attention from the tracheal lesions or to obscure them.

Diagnosis.—The diagnosis of syphilis of the trachea depends upon a careful study of the history of the case, indications of tracheal irritation, laryngoscopic or endoscopic examination, the coexistence of other manifestations of syphilis, and in their absence, a positive Wassermann reaction.

Prognosis.—If the condition is recognised early, excellent results may be obtained by treatment, but it is obvious that where deep destructive changes have resulted, medicinal measures can only palliate.

Treatment.—Anti-syphilitic treatment should be administered vigorously, and after a course of neoarsphenamine, mercury or bismuth preparations should be given. Inunction seems sometimes of special value in such cases. In cases of stenosis of the trachea from cicatrisation, dilatation of the stricture by means of bougies introduced through an endoscope may be practicable and afford useful help.

TUBERCULOSIS

Tuberculosis of the trachea is occasionally found post mortem in advanced cases of pulmonary tuberculosis, usually in those with extensive laryngeal involvement. Primary tracheal tuberculosis is unknown. The rarity even of secondary lesions in this tube is probably to be explained by the ciliated epithelium preventing lodgment of the bacilli.

Pathology.—Tuberculous lesions may occur at any part of the trachea, but they are more frequent in the lower part and on the posterior wall. When they occur they are usually numerous. There may be some general hyperæmia, or small tubercles, varying in size from a pin's head to a split pea, may be visible. Later, superficial ulceration occurs, forming irregular punched-out ulcers. Occasionally, the process may extend deeper, and erosion of the cartilages may occur, with the formation of sinuses and even fistulous communication with the œsophagus.

Symptoms.—Since tracheal tuberculosis is usually a late manifestation of advanced disease, its clinical indications are slight and are usually obscured by the more obvious laryngeal and pulmonary symptoms and signs, though if the process extends deeply and produces sinuses and fistulous tracks, it may become apparent. The actual tracheal symptoms are those of cough and retrosternal soreness.

Diagnosis.—This condition has to be distinguished from other chronic tracheal lesions, and a diagnosis can only be made from a careful review of the history, the general evidence of tuberculous disease and by the tracheal involvement which may be visible by endoscopy.

Treatment.—This must, from the nature of things, be largely palliative, and is in effect practically identical with that of laryngeal tuberculosis, notably intratracheal insufflation with orthoform and benzocaine (anæsthesine).

LEPROSY

In some cases of this disease, granulomatous lesions occur in the trachea, and these may eventually give rise to tracheal stenosis, owing to the contraction of new-formed fibrous tissue. The diagnosis can only be made from the occurrence of tracheal symptoms in a case with established lesions of leprosy in other parts.

The treatment is symptomatic.

SCLEROMA

Although in most cases this condition affects the nose only, scleromatous lesions may be found in the trachea as a pathological curiosity. The disease in any form is rare in England, and occurs chiefly in Poland and Austria. The nodules of granulomatous tissue in the trachea may cause partial obstruction mechanically, or, on contraction, lead to actual stenosis.

TRACHEAL OBSTRUCTION

Obstruction to the lumen of the trachea may be produced by foreign bodies, by conditions originating in the trachea, and by pressure from without.

FOREIGN BODIES IN THE TRACHEA

The commonest route by which foreign bodies enter the trachea is through the mouth and larynx, in the acts of breathing, laughing, yawning, sighing, or before and after coughing, when food or some foreign substance is in the mouth. A piece of bone, a stud, button, false teeth, chewing gum, peas, articles of food, nuts, grains of wheat, beads or blades of grass are among the substances which may gain entrance to the trachea in this manner. Surgical operations in the mouth and throat may lead to the inhalation of a tooth, a piece of tonsil or a mass of adenoid tissue. Material vomited from the stomach, such as food, blood clot or intestinal worms, may be inhaled into the trachea. A large blood clot in hæmoptysis may temporarily obstruct it. Foreign bodies may also gain access through the tracheal wall, such as small projectiles in wounds of the neck, a piece of new growth, or tuberculous glands by ulceration through the wall.

Unless it becomes impacted, or is too large to enter one of the two main bronchi, a foreign body rarely remains long in the trachea. It either causes death with dramatic rapidity, is coughed out again, or passes down into one or other of the large bronchi or their secondary divisions, where it produces results which are described in the section on diseases of the bronchi.

Symptoms.—These depend upon the mode of entry, the size of the foreign body, and the degree of obstruction to the air current which it induces, but in general the tracheal symptoms are less urgent than those of laryngeal obstruction, and less serious than those of obstruction of one or other main bronchus. There may be intense dyspnoea, with great discomfort and alarm during the actual passage through the larynx of a small foreign body, especially if it is temporarily arrested there; but when it enters the trachea there is an almost instantaneous cessation of the acute distress, though some degree of dyspnoea may persist. The type of dyspnoea is inspiratory in the main, though a minor degree of expiratory difficulty may be apparent if the foreign body is of considerable size. There may be a definite stridor with both phases of respiration, but it is more pronounced in inspiration. If the foreign body remains loose in the trachea, which may occur if it is rounded and too large to engage in one of the main bronchial divisions, a sound of vibratory character may be heard on auscultation of the trachea, sometimes described as the *bruit de grelottement*. This may be produced by friction of the foreign body against the tracheal wall, or more commonly by the air passing over it during respiration. A paroxysmal cough may occur, caused by the foreign body irritating the sensitive posterior wall of the trachea, and during such an attack the foreign body may be forced up to the larynx, obstruct it, or cause reflex spasm with intense dyspnoea and cyanosis and a risk of suffocation, unless it drops back, is coughed out, or removed. When sudden rupture of caseous material into the trachea occurs, the lumen may be blocked and death take place rapidly.

Course.—A foreign body impacted in the trachea may give rise to septic inflammation of its walls, with subsequent cicatrisation after removal, or it may lead to secondary infective processes in the lungs, such as purulent bronchitis and broncho-pneumonia.

Diagnosis.—The history of disappearance of some object from the mouth during coughing, breathing or laughing should give rise to suspicion of an

inhaled foreign body, and this may be confirmed by seeing the object directly by endoscopy, or indirectly by means of the X-rays.

Prognosis.—This depends in the main on the nature of the foreign body, and the time elapsing before its removal. An irregular, rough or soft foreign body is more likely to induce septic complications than a smooth, hard substance. Apart from rapidly fatal results, the prognosis is better with intratracheal foreign bodies than with those reaching the bronchi. If removal is effected within 24 to 36 hours, recovery is usually rapid and complete.

Treatment.—Treatment consists in rapid removal with as little damage to the trachea and larynx as possible. This may be effected by means of forceps passed through a bronchoscope, or rarely by tracheotomy alone, when the foreign body may be coughed out through the opening or be easily removed by forceps. Inversion of the patient in the hope that gravity may assist the expiratory efforts of cough is dangerous and should only be attempted after tracheotomy has been performed. Where rupture of a caseous gland or softening new-growth occurs into the trachea, an immediate tracheotomy may be necessary.

OBSTRUCTION FROM CICATRISATION OF THE TRACHEAL WALLS

Ætiology.—This may result from any condition leading to ulceration of the tracheal walls, with subsequent healing, such as a syphilitic gumma, or less commonly other granulomata, such as tubercle, leprosy or scleroma. Another cause is cicatrization from wounds of the trachea, accidental, suicidal or after tracheotomy, when the incision has been made too near the cricoid, or when the wound has become infected or the tube left in too long. Scarring from damage to the trachea by the inhalation of boiling or caustic liquids or even by inhaled gases may lead to stenosis.

Pathology.—The deformity of the trachea and the obstruction of its lumen depend upon the situation and the extent of the cicatricial contraction of its walls. It may be local, producing an hour-glass constriction, or involve a long extent of the tube. Occasionally, especially in syphilitic lesions, stenosis may occur at two different levels.

Symptoms.—These depend upon the degree of stenosis, the rapidity with which it develops, and the condition of the larynx, bronchi and lungs. When the stenosis is produced gradually, as in cicatrization, a degree of obstruction may result, greater than would be compatible with life if suddenly induced. In the early stages of a progressive stenosis, slight dyspnoea may be present on exertion, and during sleep a faint stridor may be audible, disappearing when the patient is awake. As the contraction progresses, the dyspnoea becomes more marked, and a definite and persistent stridor develops, at first inspiratory only, though expiration may become both noisy and obstructed. The patient may experience a sensation of obstruction referred to the neck or under the sternum, accompanied by pain and irritation, leading to cough, which may be dry, noisy and metallic, or accompanied by more or less frothy sputum, if the primary condition is associated with widespread tracheitis. The voice may lose tone and volume, and the patient talk more quietly than normal and with some evident effort. In advancing stenosis, sudden and alarming attacks of dyspnoea may occur,

leading to cyanosis and threatening suffocation. These attacks are usually due to an accumulation of mucus at the site of the stenosis. The patient in advancing degrees of obstruction cannot lie down, and generally sits leaning forward with chin depressed. It may be noted that the extraordinary muscles of respiration contract forcibly, and yet the laryngeal excursions may be small or hardly noticeable, in contrast with those of laryngeal obstruction in which they are maximal. This distinguishing sign was first pointed out by Gerhardt, and is of value, but unfortunately it is not absolute and cannot, therefore, be regarded as pathognomonic. On auscultation over the trachea, a noisy roar may be audible, of maximum intensity near to the obstruction, whereas the breath-sounds over both lungs may be deficient, although the stridor may be conducted bilaterally.

Course.—The course of cicatricial stenosis is usually progressive, unless arrested by treatment, and the dyspnoeic attacks become more frequent and alarming.

Diagnosis.—Tracheal obstruction from cicatrization has to be distinguished from laryngeal obstruction, in which the symptoms are usually more acute and more urgent. Gerhardt's sign described above may also be suggestive. It has also to be differentiated from obstruction due to pressure from without (*vide infra*). The only reliable method of distinction is by direct inspection with the bronchoscope.

Prognosis.—Early syphilitic stenosis may be arrested by appropriate anti-syphilitic treatment. Obstruction due to other granulomatous conditions varies with the severity and extent of the primary lesions. Caseous material or degenerated growth ulcerating into the trachea is usually immediately fatal, or leads to death within a few days from pulmonary complications.

Treatment.—Rest, avoidance of exertion, smoking and alcohol should be advised. The patient's fears should be allayed and symptomatic treatment ordered, such as sedative inhalations or a linctus to check useless cough. In syphilitic stenosis vigorous anti-syphilitic treatment with neoarsphenamine,¹ mercury or bismuth preparations should be given. A low tracheotomy may be necessary for an intractable stricture high up in the trachea. In some cases where an ordinary tracheotomy cannot be performed below the stricture, it may be possible to insert Koenig's long tracheotomy tube through an opening in the trachea made above it. In other cases, dilatation of a fibrous stricture by bougies passed through an endoscope may be feasible.

OBSTRUCTION FROM EXTERNAL PRESSURE

Pressure on the trachea may occur in the neck or in the mediastinum.

Causes of pressure in the neck.—Strangulation, throttling or garotting leads to death by occlusion of the trachea and suffocation. Enlargement of both lobes of the thyroid body may cause lateral compression of the trachea, until eventually its lumen is reduced to a narrow slit—the so-called "scabbard trachea." Irregular or unilateral enlargements on the other hand cause deviation of the trachea, with kinking of its lumen. Other less common

¹ According to Mr. Harold Barwell, administration of potassium iodide is very dangerous, as it increases the secretion which is pent up behind the stenosis. It may be given in combination with belladonna, but it is better withheld until the severity of the condition has been relieved by neoarsphenamine.

causes of compression of the trachea are enlargement of the cervical glands from tuberculosis, malignant disease, Hodgkin's disease or leukaemia. The trachea may be pressed on from behind by a foreign body impacted in the oesophagus, or by a bony tumour arising from the vertebrae.

Causes of pressure in the mediastinum.—An aneurysm of the aortic arch may press directly upon the trachea at, or near, the bifurcation and cause obstruction. Similarly deep pressure may be caused by a retrosternal goitre, a persistent and enlarged thymus, or a thymic abscess, mediastinal glands enlarged from any cause, usually malignant disease, a dermoid cyst or a bony tumour originating in the sternum.

Symptoms.—The symptoms are in the main identical with those of stenosis of the trachea from intrinsic causes, with the special symptoms due to the primary external condition superadded.

Diagnosis.—This may be simple and obvious, as in those cases due to pressure from tumours in the neck, whereas, in those due to mediastinal pressure, it is usually only possible after a careful survey of all the symptoms, and is in brief identical with that of aneurysm or mediastinal new-growth, to which reference should be made. In some cases X-ray examination may give valuable information.

Prognosis.—This is good in obstruction due to causes in the neck other than malignant disease, but it is almost uniformly bad, indeed hopeless, in obstruction due to mediastinal causes, with the exception of abscess and some thymic conditions.

Treatment.—The treatment is that of the primary condition. In goitre and tuberculous glands, in simple tumours, cysts and some thymic conditions, operation may be possible and may effect complete cure. In those due to mediastinal pressure, especially from aneurysm or new-growth, treatment, in most cases, can be only palliative or symptomatic, and directed to the relief of pain, dyspnoea, cough and distress.

INJURY

Direct violence to the trachea has been known to cause rupture when the chin is raised upwards and the trachea is, therefore, extended.

R. A. YOUNG.

G. E. BEAUMONT.

DISEASES OF THE BRONCHI

BRONCHITIS

Inflammation of the bronchi, or bronchitis, is one of the commonest maladies and may be induced by a variety of causes. These, in the main, fall into three groups: bacterial, chemical and mechanical, similar to the causes of tracheitis, which is, indeed, in many cases, a concomitant or antecedent of bronchitis, so that tracheo-bronchitis would be a more accurate designation of the majority of cases. At the same time it should be recognised

that the trachea may be alone or predominantly affected, while, on the other hand, in many cases of bronchitis of the smaller tubes, the trachea may escape, or be only slightly involved.

Bronchitis is so varied in its extent and in the form and severity of its manifestations that a satisfactory classification is somewhat difficult to formulate. We propose to consider the clinical manifestations of bronchitis according to the following classification :

1. ACUTE FORMS—(a) Catarrhal bronchitis, (1) of the larger tubes, (2) of the smaller tubes ; (b) suppurative ; (c) secondary bronchitis ; (d) bronchitis due to mechanical and chemical agencies ; (e) fibrinous.

2. CHRONIC FORMS—(a) Catarrhal, (b) suppurative, (c) secondary, (d) due to mechanical agencies, and (e) fibrinous.

1. ACUTE BRONCHITIS

ACUTE CATARRHAL BRONCHITIS OF THE LARGER TUBES

Synonyms.—This condition is often called Bronchial Catarrh, or Acute Tracheo-bronchitis.

Ætiology.—*Predisposing causes.*—Climate and latitude undoubtedly play an important part. Catarrhal bronchitis is rare in polar and arctic regions and near the equator, but is very prevalent in damp and foggy climates. In England, attacks are common in late autumn, winter and early spring. It is probable that some degree of hereditary predisposition occurs, since "weakness of the chest" is common in some families. Owing chiefly to greater exposure, the disease occurs more frequently in men than in women. It is most common at the extremes of life, infancy and old age, but it is not infrequent at any age. Fatigue and privation play their part, and exposure to cold, wet or fog so frequently seems to initiate the attack that it is often regarded as the exciting cause. Scoliosis, kypho-scoliosis and other malformations or deformities of the chest predispose to bronchitis, and some of them are induced or aggravated by bronchitis early in life. Chronic cardiac and renal disease both render their subjects more liable to bronchitis, as do also conditions of the nose and pharynx which lead to mouth-breathing, in consequence of the inhalation of air which is unwarmed and unfiltered by the nose. In childhood, dentition seems to be a frequent predisposing condition.

The exciting cause is usually one of the catarrh-producing organisms, and one or more of the following may be found in the sputum: the pneumococcus, Friedländer's pneumo-bacillus, streptococci, *Micrococcus catarrhalis*, staphylococci, *M. tetragenus*, and filter-passing organisms. It may also be caused by the *Spirochæta brônchialis*.

Pathology.—The changes induced in the bronchi are similar to those in the nasal mucosa in coryza and in the trachea in tracheitis. Three stages may be described: An initial dry stage, when there is active hyperæmia of the bronchial mucosa, with exudation into the submucous layer, causing temporary diminution of the bronchial secretion from occlusion of the mucous ducts. The second or mucoid stage is associated with copious discharge of mucoid secretion, owing to increased activity of the mucous

glands, this secretion being mixed with shed ciliated epithelial cells and scanty leucocytes. Sometimes in acute cases a few red blood corpuscles are present. The third stage is that of resolution, though not infrequently a muco-purulent stage occurs, when the sputum becomes less copious and greenish in colour from large numbers of pus cells.

In fatal cases the lung tissue may appear slightly distended and red, while the bases may be sodden from cedema. On section, the bronchi appear injected and the mucosa is swollen. On squeezing the lung, beads of mucoid fluid or muco-pus exude from the cut ends of the bronchi. There is no consolidation and the lung tissue floats in water.

Symptoms.—An attack of acute bronchitis generally begins suddenly, with malaise, aching in the limbs, and a sense of oppression in the chest. If the trachea is also involved, there is the characteristic feeling of rawness under the sternum. The temperature rises, varying from 99° to 100° F. in mild cases to 103° F. in more severe ones. The cough is at first dry, irritating and ineffective, but in a few hours it becomes looser. The sputum in the early stage is scanty, tenacious and sometimes streaked with blood; it then becomes copious, mucoid and frothy in character, and is found to contain mucus, shed epithelial cells, leucocytes and red blood corpuscles. Later it lessens in quantity and may become thick, yellow and muco-purulent. With the onset of expectoration there is generally an abatement in the symptoms, the rawness under the sternum disappears, and the feeling of pain or soreness about the pectoral muscles and the costal attachments of the diaphragm lessens. The febrile reaction may last only 3 or 4 days, but the cough and expectoration may go on for 10 days or longer, gradually diminishing, until they are present only night and morning, and then cease completely. *SIGNS*

In the early stage the patient is flushed and the breathing may be slightly increased in rate, but it is rarely or never laboured, unless emphysema co-exists. Vocal fremitus is unaltered, but rhonchal fremitus may sometimes be felt over one lung or both. The chief physical signs are discovered only on auscultation. The breath-sounds may be harsher and higher-pitched, particularly in infants and children, but they remain vesicular, and expiration may be prolonged. The voice conduction is unaltered. As a rule rhonchi, either sonorous or sibilant, according to the size of the bronchus in which they are produced, are audible over both lungs, and during the mucoid stage bubbling râles may be heard, especially at the bases.

Complications and Sequelæ.—Bronchitis may go on to broncho-pneumonia or may be followed by lobar pneumonia, fibroid induration or bronchiectasis. It may lead to chronic bronchitis, or be followed by active tuberculosis. Occasionally acute interstitial emphysema may result from violent coughing.

Course.—This is variable. The patient may be convalescent in from 7 to 14 days, but cough, expectoration and a condition of debility may continue for several weeks, though, in this case, the possibility of pulmonary tuberculosis should always be considered.

Diagnosis.—The diagnosis of bronchitis is usually easy, owing to the characteristic rhonchi, but it is important to differentiate primary bronchitis from bronchitis occurring as a secondary condition in acute specific fevers and other diseases.

Prognosis.—Bronchitis of the larger tubes is rarely fatal, except when it occurs in infants or the aged, or as a complication of advanced cardiac or renal disease.

Treatment.—**PROPHYLACTIC.**—This consists in the avoidance of stuffy, ill-ventilated rooms and places of entertainment when catarrhal infections are rife. In mouth-breathers, steps should be taken to deal with the conditions of the naso-pharynx inducing this habit, and instruction in normal breathing given. In dusty occupations, suitable measures should be taken to minimise the irritant particles in the air, as is now done in most factories and workshops. Where poisonous gases have to be encountered, some form of efficient gas-mask should be utilised.

Prophylactic inoculation by vaccines, either from stock mixtures such as are now available, or from autogenous cultures, is now being extensively used, and with some success. An autogenous vaccine is usually to be preferred, if possible. The dose given depends upon the organism and varies from 1 to 50 or 100 millions. Two or three doses at intervals of 7 to 10 to 14 days are usually given in the case of the stock vaccines, whereas with the autogenous a course of 6 to 12 gradually increasing doses is given at intervals of about a week.

CURATIVE.—No matter how mild the attack may be at the onset, the patient should be kept in bed. This may only be necessary for 1 or 2 days, but he should keep to his room till his temperature has returned to normal. The Turkish bath taken by some patients at the first onset is unwise and should be discouraged. The room temperature should be kept at 60° to 65° F. While the temperature is raised the diet should be the ordinary, simple, liquid diet suitable to febrile conditions, namely, milk, weak tea, cocoa and simple gruels, broths or one of the many invalid foods. The patient is often thirsty, and warm or hot demulcent drinks, such as toast water, fruit juices in hot water, and linseed tea sometimes afford great comfort. The air of the bedroom may be moistened by means of a steam kettle in the dry stage, but the use of a steam tent is to be avoided. Local applications over the sternum, acting as counter-irritants, seem to give some relief to the distressing soreness so often complained of. A mustard leaf or one of the medicated wools is the most easy to apply, but a linseed poultice, kaolin poultice or a liniment, such as camphorated oil or the acetic turpentine liniment, may be ordered. Some patients find a cold or hot compress to the neck comforting. Medicated inhalations may be used, either in a special inhaler or in a domestic substitute, such as a jug. At first vapor benzoini—60 minims to the pint of water at 160° F.—is the most comforting, but in later stages vapor pini (olei pini, min. 10; mag. carb. levis, gr. 10; aquam ad min. 120)—120 minims to the pint—or a dry inhalation of creosote, terebene and spirits of chloroform may be useful. It is often wise to start treatment with an aperient, unless this is contra-indicated. In the dry stage a simple saline diaphoretic mixture may be given, with tinct. ipecacuanhæ or vin. antimoniale in small doses. One-drop doses of tincture of aconite are also sometimes given. When expectoration starts it may be encouraged by saline and stimulating expectorants, such as ammonium chloride or carbonate, combined with squills and flavoured with syrup of tolu or of Virginian prune. For the first night it may be well to give 10 grains of Dover's powder to relieve discomfort and secure sleep.

During convalescence the patient should take care to avoid chill and should be given a more liberal diet. A mixture of strychnine and phosphoric acid may be given for a few days, and a linctus or lozenge containing small doses of diamorphine (heroin) or other sedative, to lessen the ineffective cough, which not infrequently occurs, especially at night. Convalescence is usually shortened by a few days' stay at the coast, especially the south.

When bronchitis occurs as a part of some specific disease, such as diphtheria or influenza, the treatment should be that appropriate to the particular disease.

ACUTE CATARRHAL BRONCHITIS OF THE SMALLER TUBES

Synonym.—Capillary Bronchitis.

It is open to question whether this condition exists as a separate entity. When the finer bronchi and bronchioles are inflamed the alveoli invariably become involved, since very little swelling of the bronchiolar walls is sufficient to occlude the lumen of the tube, with the inevitable production of an area of lobular collapse. The transition from this condition to actual lobular pneumonia is a very small one. In any case, the causes, the symptoms and the treatment of capillary bronchitis and broncho-pneumonia are identical. (See Secondary Broncho-pneumonia.)

ACUTE SUPPURATIVE BRONCHITIS

Synonyms.—Sometimes called Acute Purulent Bronchitis, or Suffocative Catarrh.

This condition was brought into prominence during the Great War. In 1916 and 1917 it appeared in epidemic form amongst the British troops in England and France. Although it was then regarded by some observers as a new disease, it is more probable that it was, in reality, an epidemic form of a condition usually rare and sporadic and previously termed "suffocative catarrh." That name has unfortunately been applied loosely to a number of conditions associated with acute dyspnoea.

Ætiology.—*Predisposing causes.*—The exceptionally severe winter of 1916-17, together with conditions of overcrowding in huts and billets, were undoubtedly concerned in the epidemic just mentioned. The condition affects young adults chiefly, and is much more common in men. Over-exertion, fatigue and debility predispose to it, but the disease may occur in robust and healthy persons. A history of chill may be given, but often no obvious cause can be discovered.

Exciting cause.—The organisms usually found are the pneumococcus and Pfeiffer's *H. influenzae*, the latter being reported in 90 per cent. in some series of cases. The *Micrococcus catarrhalis* is also sometimes present.

Pathology.—A very intense inflammation occurs in the medium-sized and small bronchi, leading to an exudate rich in leucocytes. The inflammatory process may extend to the alveoli, which then contain a fibrinous fluid, with entangled red cells. The condition occurs in both lungs and is usually almost universal, no portion being spared. Post mortem the lungs are heavy and red in colour. On section the bronchi are found to contain a thick yellow purulent fluid. Small areas of collapse and sometimes of broncho-pneumonic consolidation are seen, and there is usually oedema of the

bases. Plastic pleurisy is not infrequent, and the glands at the root of the lung are enlarged.

Symptoms.—The onset is usually abrupt, often in young people apparently in robust health. A definite chill may occur, or only coryza and general malaise, with aching of the muscles. The temperature rises quickly and may reach 104° F. early in the disease. A cough soon develops and extreme dyspnoea is a characteristic feature. Expectoration starts early, often on the second or third day. At first it may be streaked with blood, but it soon becomes yellowish green and nummular; it consists of almost pure pus; there is often as much as 5 or 6 ounces in 24 hours. In most instances there is great prostration. In grave cases the patient becomes unconscious and loses control of the sphincters.

There is intense cyanosis, the face, lips and ears being purple. Respiration is rapid, 30 or 40 per minute, and the accessory muscles are often in full action. Palpation and percussion may not show any abnormality though slight dullness is sometimes present at the bases. At first no signs may be discovered on auscultation, but soon the breath-sounds become largely obscured by medium-sized bubbling râles, often audible from apex to base, both front and back. The pulse is frequent, the right heart may dilate and the heart-sounds become weak.

Complications and Sequelæ.—In severe cases recurrent bronchitis, broncho-pneumonia, fibroid disease or emphysema may follow.

Course.—In favourable cases complete restoration to health results. In severe cases the course is rapid, the patient becomes comatose from toxæmia, expectoration ceases and death occurs from exhaustion in 2 or 3 days from the onset. In other cases the disease may last for weeks and proceed to recovery or death.

Diagnosis.—The early occurrence of marked dyspnoea and cyanosis, the expectoration of copious pus, and the widespread râles without dullness are very suggestive of acute suppurative bronchitis. The disease must be differentiated from other conditions described as acute suffocative catarrh that are associated with extreme dyspnoea and cyanosis.

Acute pulmonary oedema is usually afebrile, and the sputum is albuminous, frothy and copious. The condition leading to it, such as cardiac or renal disease, may be apparent.

Capillary bronchitis or broncho-pneumonia may give rise to difficulty, but in these conditions the sputum is scanty, tenacious, sometimes rusty, and but rarely purulent; moreover, cyanosis and dyspnoea develop late and depend upon the extent of the disease and the condition of the right side of the heart.

Pneumonia of the wandering type may simulate this condition, but the character of the signs, with dullness and tubular breathing, and the rusty sputum, usually render diagnosis easy.

Prognosis.—This is very grave. The mortality is high, often as much as 50 per cent. Cases extending to 3 weeks or more with swinging temperatures usually recover.

Treatment.—Cases of this disease should be isolated. If there are indications of an epidemic spread, prophylactic inoculations with a vaccine made from the special strain of pneumococci concerned may be useful in limiting it. In any future epidemic, a trial should be made of sulphapyridine

(M. & B. 693). The steam tent and the inhalation of medicated vapours, such as vapor benzoini, may give a little relief to the dyspnoea. Oxygen should be administered either by means of an oxygen tent, the double nasal catheter or the B.L.B. mask. Venesection may give temporary relief, but produces no lasting effect. Ammonium carbonate and potassium iodide are generally recommended. Stimulants, such as brandy and strychnine, should be given freely, and hypodermic injections of nikethamide (coramine) or leptazol (cardiazol) may be given.

SECONDARY BRONCHITIS

Ætiology.—Bronchitis, usually of catarrhal type—indistinguishable as regards symptoms and signs from primary acute catarrhal bronchitis—occurs as a definite part of many acute infectious diseases and as a complication in others. Among these may be mentioned measles, whooping-cough, influenza, the enteric group, small-pox, diphtheria, malaria and plague. Acute nephritis of infective origin is often accompanied by acute bronchitis. Other conditions associated with bronchitis are pulmonary tuberculosis, glanders, secondary syphilis, pleurisy and gunshot wounds of the chest.

Diagnosis.—Bronchitis is easy to recognise, but it is important not to overlook the fact that it may not be the primary condition. In all cases of bronchitis in the early stages, the possibility of a primary acute specific infection should be borne in mind. The diagnosis is also of importance in regard to treatment—for example, in malaria, nephritis and syphilis, in which treatment directed to the primary condition may be more helpful than the ordinary treatment of catarrhal bronchitis.

BRONCHITIS DUE TO MECHANICAL AND CHEMICAL AGENCIES

Ætiology.—*Mechanical.*—Attacks of acute bronchitis may be caused by the inhalation of dust-laden air. In occupations where the worker is liable to inspire fine particles of carbon, silica, steel, iron, asbestos or kaolin, acute bronchitis may result, but more often these conditions lead to chronic bronchitis and pneumokoniosis. Pressure on a bronchus by aneurysm or new-growth, or irritation by the presence of a foreign body, may induce local acute bronchitis. The symptoms and signs are practically identical with those of the catarrhal form and need no special description.

Chemical.—Acute bronchitis may follow the inhalation of chemical irritants, either as a result of occupation, accidents, attempts at suicide, or the use of poison gases in warfare. Special attention has been drawn to this subject by the large number of cases of “gassing” dealt with in the War of 1914–1918. Death not infrequently occurred, much acute suffering was caused, and some permanent damage has resulted in many cases which recovered. “Mustard gas” produces its chief effects upon the skin, the eyes and the bronchi. A fibrinous exudate forms on the mucosa as a false membrane, which separates as a slough. The suffocative gases chlorine and phosgene affect the alveoli primarily and more intensely. Chlorine inhaled in a concentration of 1 in 10,000 causes a rapid alveolar flooding with a serous and highly albuminous fluid, and if the victim does not die at once he is liable to suffer from an acute bronchitis. A condition called bronchiolitis fibrosa

obliterans may occur as a sequel. It is often associated with asthmatic dyspnœa.

Symptoms.—These are similar to those of acute catarrhal bronchitis, but there is great pain, distress and almost constant cough, often with copious expectoration.

The treatment is referred to under the heading of Tracheitis, and is, in the main, symptomatic and directed to the relief of pain, useless cough and distress. If there is cyanosis, oxygen should be given continuously if necessary by double nasal catheter or B.L.B. mask.

ACUTE FIBRINOUS BRONCHITIS

Synonym.—Acute Plastic Bronchitis.

Definition.—A comparatively rare acute disease in which there is inflammation of the bronchi, with the formation of casts. These may be hollow or solid, and are coughed up in the expectoration.

Ætiology.—The cause of the disease is unknown. It is more common in males, and is met with both in children and in adults. It may begin as a primary catarrhal bronchitis, or develop as a complication of enteric fever, measles or pulmonary tuberculosis. Such organisms as the pneumococcus or a streptococcus may be found in the casts.

Pathology.—The casts may involve the main bronchi only, or more frequently the smaller ones and the bronchioles. They are greyish white, solid or tubular, and when large, bear the impress upon their exterior of the bronchial walls in which they have been enclosed. Thus, when a cast extends up to the lower portion of the trachea, the indentations made by the tracheal rings may be seen impressed upon it. The fine terminations generally show a spiral moulding. Chemically, they consist of fibrin or of fibrin and mucin. Post mortem the casts may be seen in some places *in situ*; in other areas the bronchi from which they have been expelled may be recognised. The bronchial mucous membrane is at times acutely inflamed, red in colour, with the lining epithelial cells desquamating, or it may appear pale and unaffected. There is usually a certain degree of emphysema, and there may be collapse of lung tissue beyond the site of obstruction.

Symptoms.—The disease generally begins somewhat abruptly with a cough and malaise. In the course of a few days the patient becomes considerably worse, dyspnœa develops and a certain degree of pyrexia, but the temperature is often not more than 99° or 100° F. The dyspnœa becomes more intense, and is the prominent and all-important symptom. The face is seen to be cyanosed, the *alæ nasi* and the accessory respiratory muscles are in violent action, sometimes with retraction of the intercostal spaces. There may be diminished movement of the chest, either bilateral or unilateral. If there is unilateral pulmonary collapse the heart may be slightly displaced towards the same side. Vocal fremitus may be normal or locally diminished. The percussion note is somewhat hyper-resonant over the anterior chest-wall, but behind there may be some degree of dullness over one or other lobes. If the bronchi are unilaterally affected there may be dullness limited to one lower lobe, with diminution of air entry and no adventitious sounds. Vocal resonance over the affected area is lessened. There is usually some diffuse bronchitis, as indicated by the presence of rhonchi or râles. Marked stridor

is sometimes heard with respiration. A special sign, the "bruit de drapeau," has been described when the cast lies free in the bronchial lumen. It is a dry clicking sound, caused by the flapping of the cast against the wall of the bronchus as the air passes over it. The ordinary sputum does not show any peculiarities. It may, however, show Curschmann's spirals, Charcot-Leyden crystals and eosinophile cells, and it may be absent until the crisis occurs. This consists in the expectoration of the cast after a violent fit of coughing. The cast may be stained with blood, or there is sometimes actual hæmoptysis. The peculiar nature of the expectoration often escapes notice, unless it is examined by floating in water, when a large intact cast is revealed. The dyspnœa ceases immediately after the cast has been expelled.

Complications and Sequelæ.—Emphysema may occur as the result of the violent coughing, or the disease may become chronic, recurring at intervals of varying duration. The most serious complication is laryngeal obstruction, caused by the cast becoming impacted between the vocal cords.

Course.—The disease is generally self-limited, terminating with the separation and expectoration of the cast. The acute stage does not, as a rule, continue for more than 12 to 24 hours.

Diagnosis.—The stridor and respiratory obstruction are suggestive of œdema of the glottis, but auscultation will show that the site of the lesion is lower down the respiratory tract. Asthma, and all causes of laryngeal and tracheal obstruction, must be excluded. The dyspnœa and the presence of signs localised to one lobe may suggest an active lobar collapse, or a lobar pneumonia, but the dyspnœa is more intense than is met with in either of these conditions. Casts are expectorated in diphtheria, pneumonia, chronic disease of the heart, pulmonary tuberculosis and hæmoptysis. The casts of acute fibrinous bronchitis are firmer than those found in these affections, and are expectorated in long pieces, showing the many branches and bifurcations of the bronchial tree.

Prognosis.—The immediate outlook is fair. Death may occur in the first attack, or recurrences may take place, which lead to an increasing degree of emphysema, with its usual results. The ultimate prognosis is, therefore, not good.

Treatment.—The patient should be kept in bed and treated as a case of acute bronchitis. Inhalations of medicated vapours often afford relief. Potassium iodide is believed to expedite the separation of the cast. Intratracheal injections of olive oil or lime water have been recommended, as the casts tend to dissolve in the latter. Tracheotomy instruments should always be at hand in case of laryngeal impaction.

2. CHRONIC BRONCHITIS

Chronic bronchitis is perhaps even more difficult to classify than the acute varieties, each one of which may have its counterpart in chronic form, so that the same classification may be followed. At the same time it must be admitted that, especially in the catarrhal forms, the clinical manifestations are somewhat varied.

CHRONIC CATARRHAL BRONCHITIS

Ætiology.—The causes are practically identical with those of the acute form, of which it is in most cases a sequel.

1. This affection may commence at any age, although it is more common in
2. middle life and with advancing years. Men are more frequently affected
3. than women. It seems also to have a special incidence in some families.
4. It is more common in damp and foggy climates, and is favoured by urban conditions and by dusty occupations. It starts each winter with a more or less acute catarrhal attack, but each year the summer intermission becomes shorter, until the bronchitis persists throughout the year. It tends to produce
5. emphysema and is aggravated in turn by this condition. It is especially
6. favoured by cardiovascular lesions, such as valvular defects and arterial
7. disease; also by gout, chronic nephritis, syphilis and alcoholism. Con-
8. ditions associated with chronic cough predispose to it, notably emphysema, asthma, arrested pulmonary tuberculosis, mouth-breathing and cigarette-smoke inhaling.

The bacteria found are practically identical with those in acute bronchitis, the commonest being the pneumococcus, Friedländer's pneumobacillus, *Micrococcus catarrhalis*, streptococci and staphylococci. Mixtures of two or more of these may be present. A rarer cause is bronchial spirochætosis, from infection with the *S. bronchialis*.

Pathology.—The bronchi show chronic inflammatory changes of a catarrhal nature. The walls are thickened from chronic hyperæmia and also from productive changes in the connective tissues. The mucous glands may be hypertrophied or atrophied, and there may be widespread desquamation of the ciliated epithelial lining of the bronchi. In long-standing cases there is usually some peribronchitis, leading to cylindrical bronchiectasis and distortion of the bronchi by fibrosis. There is almost invariably a greater or less degree of emphysema, which may be generalised or only marginal. Post mortem, the lungs are generally red and somewhat engorged, but if much emphysema has resulted they may be paler than normal. On squeezing the lung after section, pus or muco-pus exudes from the cut bronchi, and there is usually some evidence of œdema at the bases.

Symptoms.—A patient with chronic bronchitis complains of his "chest." By this he means that he suffers from cough, expectoration and shortness of breath on exertion. The cough varies greatly in its severity. During the warm weather the patient may be completely free, and yet suffer for years from a winter cough. It may occur frequently throughout the day and in attacks at night, or only in the mornings and evenings.

The expectoration varies considerably in quality and quantity, so much so that the old classifications of chronic bronchitis were based on this factor. Thus, there may be practically no sputum or only small tenacious pellets, the "crachats perlés" of Laennec; on the other hand, there may be a profuse expectoration resembling unboiled white of egg diluted with water, constituting the form described as "pituitous catarrh" or "bronchorrhœa serosa." Usually the sputum is mucous or muco-purulent and contains greyish-black particles mixed with a frothy fluid. The dyspnoea is largely due to the accompanying emphysema, and so indicates the degree of chronicity of the disease. At first the patient may only notice that he gets out of breath on going upstairs or on mounting slopes, but later even walking on the level causes dyspnoea.

Slight rises of temperature occur in the acute exacerbations of the catarrhal process. Slight cyanosis is frequently observed, especially after

exercise, when the accessory respiratory muscles are called into play. Sometimes rhonchal fremitus is felt. Movement of the chest is restricted by emphysema, and the percussion note then becomes hyper-resonant. On auscultation, expiration is prolonged and sonorous or sibilant rhonchi are heard all over the lungs, with bubbling râles if there is thin secretion in the smaller bronchi. On the other hand, rhonchi may be scanty or only occasionally heard. Voice conduction is unaffected. The fingers may be slightly clubbed, and further evidence of venous obstruction apparent in the dilated venules on the cheeks or along the costal attachments of the diaphragm.

Complications and Sequelæ.—The following changes may occur in the lungs—peribronchial fibrosis, bronchiectasis and emphysema. Asthma or attacks of bronchial spasm sometimes form a complicating factor in chronic bronchitis, especially in the cases of so-called bronchorrhœa. The increased cardiac strain may lead to right-sided dilatation, with basal pulmonary congestion, ascites and œdema of the legs. Late in the disease, as the result of the cyanosis, a peculiar form of confusional delirium is met with, which is worse at night.

Course.—The disease once firmly established, unless relieved by suitable climatic treatment, remains chronic and becomes progressively more severe as further damage is wrought in the lungs with each hibernal exacerbation. As the emphysema develops, a vicious circle is initiated, the aerating power of the lungs diminishes, and finally cardiac failure ensues.

Diagnosis.—Chronic bronchitis must be distinguished from pulmonary tuberculosis, bronchitis secondary to heart failure, and from bronchiectasis. In tuberculosis with bronchitis there is generally wasting, and often flattening of the chest-wall, owing to fibrosis of the lungs. In all cases where the summer intermission of the symptoms fails suddenly rather than lessens gradually, tuberculosis should be suspected. The diagnosis is clinched by the presence of tubercle bacilli in the sputum. In bronchitis secondary to heart failure, in addition to the cardiac signs, the râles in the lungs are chiefly basal and the rhonchi are not so universally distributed. In bronchiectasis the signs are usually characteristic and often limited to one lobe. The X-rays may afford useful aid in diagnosis.

Prognosis.—The immediate prognosis is good, the ultimate is bad. Much depends upon the patient's social condition and opportunities for treatment, especially in respect to climate. The expectation of life of a patient suffering from chronic bronchitis is considerably shortened.

Treatment.—Those subject to chronic bronchitis should live in a warm, equable and dry climate. In England the south-western districts are best, but it is advisable to winter farther afield if possible, either on the Riviera, the north coast of Africa, or in Madeira. High altitudes should be avoided if emphysema is present or if there are cardiac complications. Exposure to wet and chill is dangerous. The question of occupation is often difficult. Much time should be spent out of doors, provided that the patient is not exposed to the inclemencies of the elements; and, further, the work undertaken must not involve severe muscular efforts, or the inhalation of dusty or irritant particles.

In England it is difficult to find an outdoor occupation conforming with these desiderata, consequently light indoor work in a good atmosphere should be advised. Clothing should be warm but light, and afford special

protection to the chest without overloading, as some patients are liable to do. Excesses in diet are to be avoided, also alcohol and heavy smoking. The general nutrition should be well maintained, and many patients, especially those of spare habit, seem to derive great benefit from cod-liver oil during the winter months.

If cough is troublesome and expectoration tenacious or scanty, various combinations of expectorant remedies are useful, such as ammonium carbonate or chloride, tinct. ipecacuanhæ, preparations of squills or senega, with tolu, liquorice or Virginian prune as flavouring agents. A simple saline mixture such as R. Sodii bicarb., grs. 10; sodii chlorid., grs. 3; sp. chlorof., min. 5; aquam anisi ad fl. oz. 1, taken with an equal quantity of hot water in the morning or at night, may help to "clear the tubes" and give the patient a spell of freedom from cough. In older patients the ether and ammonia mixture may be given, and in cases with bronchial spasm potassium iodide with anti-spasmodics, such as stramonium, lobelia, belladonna or grindelia, may be of great value. Various antiseptic drugs, such as turpentine min. 10, terebene min. 5-10, creosote min. 3 in capsules or perles, have been recommended, and the elixir thymi et diamorphin. B.P.C. min. 60. Sedative lozenges, such as compound liquorice, heroin or codeine, are often useful in checking useless cough. Intercurrent attacks of acute bronchitis must be treated on the principles described under that condition and the patient kept indoors or in bed, as may be necessary. When an advanced degree of emphysema coexists the treatment appropriate to that condition should be applied. Sometimes benefit may follow the use of the compressed air chamber. When failure of the right heart ensues, with visceral engorgement, the treatment must be modified suitably as described under emphysema. Liniments applied to the chest-wall, especially those containing camphor, turpentine or belladonna, are soothing and afford relief. Care should be taken that any tendency to constipation is checked. In some cases, especially when the predominant organism is the *Micrococcus catarrhalis* or Friedländer's pneumo-bacillus, an autogenous vaccine prepared from the sputum ameliorates the symptoms when given in small doses. This should be considered especially in cases unable to undergo suitable climatic treatment.

CHRONIC SUPPURATIVE BRONCHITIS

Synonym.—Fetid Bronchitis.

Ætiology.—This condition is not sharply defined and is not a specific and separate nosological entity, but it is a convenient group to include cases with fetid purulent sputum. In some forms of chronic bronchitis the secretion may from time to time accumulate in the bronchi and prove offensive on expectoration. In some instances this condition becomes chronic and the expectoration is fetid up to the time of death.

Pathology.—There is chronic inflammation of the bronchi, with marked peribronchial thickening. The bronchial secretion becomes purulent, and ulceration of the bronchial wall or dilatation of the lumen may occur. Post mortem, the lungs are soft, and on section some broncho-pneumonic areas, with œdema of the bases, may be seen. Pus of an offensive nature exudes from the cut ends of the bronchi.

Symptoms.—These resemble those found in chronic bronchitis, with,

in addition, the unpleasant characteristics of the sputum, in which Dittrich's plugs may be found. These are small, yellowish bodies, with an intensely offensive odour, composed of compact secretion.

Complications and Sequelæ.—Ulceration of the bronchial walls, abscess or gangrene of the lung, and areas of broncho-pneumonia may develop. As with bronchiectasis, pyæmia sometimes ensues, with the formation of secondary abscesses in the brain.

Course.—The disease is progressive, but in the early stages there may be long remissions in which the sputum is not offensive although the bronchitis persists.

Diagnosis.—The sputum is offensive in abscess and gangrene of the lung, bronchiectasis and interlobar empyema. X-ray examination of the chest is of great value in revealing these conditions, and lipiodol or neo-hydriol investigation will usually serve to distinguish between them.

Prognosis.—As the disease becomes firmly established the patient's strength is gradually undermined from the absorption of toxins, and death ensues in the course of a few years, either from exhaustion, toxæmia or pyæmia.

Treatment.—An endeavour should be made to lessen or prevent the offensive character of the sputum. For this purpose creosote or garlic may be administered, as in bronchiectasis. If sputum is copious, postural drainage by means of a Nelson bed may be useful. Creosote vapour baths are also of great value. Apart from this, the treatment is as for chronic bronchitis.

CHRONIC SECONDARY BRONCHITIS

Chronic bronchitis is a common association of chronic cardiac and renal disease, and possibly also of gout. Its clinical characters do not need special description. It is only necessary to emphasise, as in the acute forms, the importance of recognising that the bronchitis is not the essential condition, and that treatment must be directed especially to the primary disease.

CHRONIC BRONCHITIS FROM MECHANICAL AND CHEMICAL AGENCIES

This usually proceeds to interstitial changes in the lung, and these results may be studied more conveniently under the heading of the pneumokonioses.

CHRONIC FIBRINOUS BRONCHITIS

Acute fibrinous bronchitis has been described above. In certain cases of chronic catarrhal bronchitis a fibrinous exudate may occur from time to time, with the formation of intrabronchial casts. There is then cough and dyspnoea, which abate with the expectoration of the cast. It therefore very closely resembles acute fibrinous bronchitis, and the treatment indicated is that described above.

TUMOURS OF THE BRONCHI

Tumours arising in the bronchi may be (a) simple or (b) malignant.

(a) *Simple tumours.*—The following varieties occur: Adenoma, lipoma, myxoma, papilloma and chondroma. Any of these may lead to bronchial

obstruction and, in consequence, to either collapse or bronchiectasis. Adenoma is of sufficient frequency and importance to require separate description.

(b) *Malignant tumours*.—Primary carcinoma or sarcoma may originate in the bronchi. In carcinoma the growth is usually of the columnar type, and arises from the lining epithelium of the bronchi or from that in the mucous glands. Oat-celled tumours also occur, and occasionally squamous-celled carcinoma. In some instances secondary deposits of carcinoma may follow very closely the paths of the main bronchi. Sarcoma may originate in the connective tissue of the bronchial walls.

Although the majority of primary malignant tumours within the lung originate in the bronchi, either from the lining epithelium or from the cells of the mucous glands, their pathological effects and clinical manifestations are in the main pulmonary, and it is therefore more convenient to describe them as tumours in the lung (see pp. 1219–1222).

ADENOMA OF BRONCHUS

Ætiology.—Adenoma of the bronchus occurs about equally in the two sexes, and usually in adults below the age of 40.

Pathology.—The tumour is at first small and of polypoid form, as a rule arising in a main bronchus, but not infrequently in the branch to the lower lobe. It is about twice as common on the right side as on the left. The bulbous end is generally directed towards the trachea. The surface is usually smooth and shiny, but may be nodular. An erroneous diagnosis of carcinoma was not uncommon in the past owing to differences of staining of certain of the constituent cells and their irregular distribution in the connective tissues. Metastases, however, are unknown. An adenoma often projects through the bronchial wall, giving it a dumb-bell or cottage-loaf conformation.

Clinical Features.—Often the earliest symptom is hæmoptysis, and this may be slight or profuse, since adenomata are very vascular and bleed easily. In other cases the tumour causes bronchial obstruction with resultant cough and wheezing, proceeding later to pulmonary collapse or bronchiectasis. Dry pleurisy may be an early result of infection, and at times pleural effusion or empyema may conceal the underlying cause.

Diagnosis.—Other causes of hæmoptysis must be considered, such as pulmonary tuberculosis, mitral stenosis, dry bronchiectasis or bronchial carcinoma. Pulmonary collapse may suggest an unresolved pneumonia. In cases of pleural effusion or empyema the diagnosis is liable to be overlooked. The injection of lipiodol or neo-hydriol, or tomography, will often reveal a blocked or deformed bronchus, but the diagnosis can only be established by microscopical examination of a portion of the tumour removed through a bronchoscope.

Prognosis.—This varies with the stage at which the diagnosis is established. If the condition is recognised early, and treated before the growth has extended outwards through the bronchial wall and before bronchial obstruction and septic infection have occurred, the outlook is favourable.

Treatment.—When the adenoma is recognised before secondary effects have developed, piecemeal removal through a bronchoscope, followed by radon application is often completely successful. There are, however, the

risks of hæmorrhage, primary and secondary, and of local recurrence, especially when the growth has extended outside the bronchus. Deep X-ray therapy has been recommended. If secondary bronchiectasis and fibrosis have occurred, lobectomy, or in rare cases pneumonectomy, may be necessary.

THE INFECTIVE GRANULOMATA

SYPHILIS.—During the secondary stage, a generalised hyperæmia of the bronchial mucous membrane may occur, giving rise to slight bronchial catarrh with the usual symptoms and signs, a condition that has been called syphilitic bronchitis. It is frequently beneficially influenced by anti-syphilitic treatment. In the tertiary stage, gummata may form in or near the large bronchi. They tend rather to fibrosis and contraction than to softening and ulceration, although the latter processes may occur. Contraction may lead to bronchial stenosis, with the symptoms and signs described below, or to extensive peribronchial inflammation and bronchiectasis. If the gummata extend into the lung, as may happen in rare instances, destructive lesions with cough, expectoration and hæmorrhage may result. This condition is more fully described in the section on pulmonary syphilis (see p. 1218).

TUBERCULOSIS of the bronchi occurs as part of pulmonary tuberculosis and does not require separate description.

LEPROSY.—The bronchi may be involved in this disease, with the production of cellular infiltration and even nodule formation. At first, these lesions may produce bronchitis, and they are progressive, leading to cough, expectoration, wasting and asthenia. The general clinical picture may simulate chronic pulmonary tuberculosis, from which it is distinguished by the presence of leprous lesions elsewhere, and the absence of tubercle bacilli from the sputum.

BRONCHIAL STENOSIS AND OBSTRUCTION

Obstruction of the main bronchi or of their subdivisions within the lungs may arise from causes within the bronchi or from conditions outside them, and these require separate consideration. It is important to emphasise the fact that in both conditions the symptoms differ according to whether the obstruction is sudden and complete, in which case collapse of the corresponding lung is the rule, or whether it is partial and more gradual, when bronchiectasis usually results. Obstruction of the smaller bronchi may result from spasm as in asthma (see p. 1148) or from disease as in small-tube and capillary bronchitis (see p. 1133).

(a) INTERNAL CAUSES

These are most conveniently considered in two groups—(1) Foreign bodies ; (2) those due to disease or cicatrisation of the bronchial walls.

(1) FOREIGN BODIES IN THE BRONCHI

These usually gain access through the larynx and trachea by inhalation. Any inhaled foreign body that is small enough to pass down the trachea may

reach a main bronchus, more commonly the right, or if it is small it may pass into one of the secondary bronchi. It may at once become impacted, or be moved by cough, but unless it is expelled in this way, it is sooner or later drawn into the smallest bronchus that will receive it, and there becomes impacted.

The recorded varieties of foreign body thus reaching the bronchi are very numerous, but among the more common are pieces of bone, beads, pins, coins, ear-rings, studs, pencils, fruit stones, grains, grasses, beans, nuts, teeth and pieces of tonsil or adenoid growths after tonsillectomy. Even a living fish has been inhaled into a bronchus. Foreign bodies may reach the bronchi through a tracheotomy wound, or a gland may ulcerate into the lumen of a bronchus. Broncholiths and pneumoliths, calcareous particles originating in the bronchi and lungs respectively, may be inhaled into a bronchus instead of being expectorated.

Pathology.—The pathological changes resulting from a foreign body in a bronchus depend upon the nature of the foreign body, the duration of its stay, the size of the bronchus obstructed by it, and the degree of obstruction induced. If the foreign body is smooth and comparatively little septic, and if it be removed within 24 hours or so, complete recovery after a very mild local inflammatory reaction may be expected. If, on the other hand, the foreign body is rough, or soft and laden with septic organisms, acute pneumonic processes, often septic in character, may develop very rapidly. A soft type of foreign body may swell and completely obstruct the bronchus it reaches, leading to complete collapse of the corresponding lung area, often the whole or half of the lower lobe. If the stay of any foreign body is prolonged to days, weeks, months or longer, irreparable damage almost invariably results. The forms this may take are numerous. Collapse and septic pneumonia have already been mentioned. If the obstruction is partial, septic bronchitis, with stagnation of the bronchial exudate and pus behind the obstruction, leads in turn to peribronchitis, bronchiectasis and fibroid induration of the corresponding lung area. In other cases gangrene of the lung results. Not infrequently an empyema may occur and the foreign body may be found in the empyema cavity. Suppuration round a foreign body may lead to localised intrapulmonary suppuration or abscess. Simple bronchial obstruction, uncomplicated by sepsis, may lead to bronchiectasis, owing to the resultant lowering of intrapleural pressure.

Symptoms.—During the passage of the foreign body through the larynx and trachea urgent symptoms may occur which leave no doubt as to what has happened; but this is not invariable, and the patient may not be sure whether he has inhaled or swallowed it. In any case, after a bronchus has been reached, there may be a latent period which engenders a sense of false security and leads to delay in treatment. In most cases pain, discomfort and cough develop rapidly. The cough may lead to the expulsion of the foreign body, or may cause dyspnoea if it forces it up to the larynx. The cough soon becomes noisy, often paroxysmal, and if local septic changes are set up expectoration occurs, sometimes mucoid and copious, at others muco-purulent. Hæmoptysis is not uncommon. Pain may be absent, but is often severe. The temperature is generally normal for the first few hours, but soon rises, especially if bronchitis, pneumonia or broncho-pneumonia

develops. The further symptoms are those of the reactive changes and complications which ensue.

The physical signs naturally depend upon the bronchus affected and upon the degree of obstruction. They are at first those of deficient air entry. The affected side may show less movement, and there may be some recession of the lower intercostal spaces in young people. If a large bronchus is involved and collapse results, there is some displacement of the heart to the affected side. Vocal fremitus may be diminished or absent, the percussion note impaired, and the breath-sounds and voice-sounds weak or absent over the whole or part of one lung, almost invariably the lower lobe. When bronchiectasis, empyema or other conditions develop, their characteristic signs become apparent.

Complications and Sequelæ.—These have been enumerated in describing the pathological results. Sometimes septic meningitis or cerebral abscess develops.

Course.—Spontaneous relief may occur in two ways, either by the foreign body being coughed up, as may happen within a few hours or days or after an interval of months or years, or the foreign body may track through the lungs and pleura, and be discharged in an abscess bursting through the chest wall. In both cases, if an interval of more than days occurs, irrecoverable damage may have resulted. Apart from these occurrences and from successful treatment the course is very variable. Death may occur quickly from some of the septic complications, or after a longer or shorter interval from bronchiectasis, gangrene or cerebral abscess.

Diagnosis.—The history of disappearance of some article from the mouth in the act of laughing, breathing, yawning, coughing or sighing, should always arouse suspicion of an inhaled foreign body. If signs indicating bronchial obstruction are found, the diagnosis is almost certain. In every suspicious case radiograms of the chest should be taken in two different directions, in case the shadow may be merged in that of the scapula or of the ribs. The possibility of a foreign body should always be borne in mind in cases of unilateral basic bronchiectasis, especially if no obvious cause can be found. When such unilateral lung signs develop after an anæsthetic, or after operations on the mouth or naso-pharynx, the possibility of some inhaled material should always be remembered.

Prognosis.—This is grave unless the foreign body is removed within 36 hours, owing to the various dangerous complications that may ensue. Excluding the few cases in which cure occurs by spontaneous discharge of the foreign body, about 50 per cent. of cases left untreated die within 1 or 2 years.

Treatment.—This consists in removal, if practicable, as soon as possible after the diagnosis is established. If the foreign body is in a main bronchus or one of its principal divisions it can usually be removed by means of the bronchoscope and appropriate forceps. In case of failure the question of pneumotomy may have to be considered. If this is decided on, every effort must be made to localise the foreign body by X-ray examination. If intrapulmonary or pleural suppuration has occurred, this must be dealt with surgically, and sometimes the foreign body can be removed at the same time. The medical treatment of the cases consists in that of the various conditions resulting.

(2) OBSTRUCTION OR STENOSIS FROM DISEASE OR CICATRISATION OF THE BRONCHIAL WALL

Ætiology.—Primary bronchial new-growths, including adenoma, columnar-celled carcinoma, oat-celled tumour and squamous-celled carcinoma lead to bronchial obstruction at an early stage. These conditions produce symptoms and signs practically identical with those of new-growths in the lung (see p. 1220).

A plug of mucus or a blood clot may cause temporary obstruction of a large bronchus.

The causes of cicatrisation are those leading to ulceration of the bronchial wall, with subsequent healing, such as syphilitic processes in and around the bronchi, ulceration from injury produced by a foreign body or in its removal, or by the inhalation of severe irritants. The fibroid variety of tuberculosis may also produce it.

Pathology.—The stenosis may occur in one of the main bronchi, or in one passing to a lobe or to part of a lobe. At first partial, it may progress until the lumen is almost completely occluded at one point. The changes occurring in the lung beyond the obstruction vary with its degree. At first there is retention of secretion in the bronchi, and air may be forced past the obstruction in inspiration, but not expelled during expiration, producing emphysema, with gradual bronchial dilatation. When the obstruction is more complete the air is absorbed, the lung tissue gradually becomes fibrotic, and the bronchi dilate further.

Symptoms.—Cough, not infrequently of paroxysmal character, is an early symptom and is usually a continuation of that caused by the primary condition. It may be dry or associated with mucoid sputum, sometimes blood-streaked. The expectoration may cause dyspnoea, by obstructing the narrowed bronchus. If bronchiectasis develops, the sputum usually becomes fetid.

The physical signs are those of collapse of a part of the lung and are progressive. Local limitation of movement and flattening, with displacement of the heart to the affected side, may be apparent on inspection. The vocal fremitus is diminished, the percussion note, impaired at first, may progress to complete dullness when fibrosis develops. The breath-sounds are weak or even absent, and the voice-sounds diminished. In the early stages a bronchial stridor may be audible. Compensatory emphysema of the adjacent healthy lung tissue often develops.

Complications.—These are similar to those in stenosis from a foreign body, notably fibrosis and bronchiectasis.

Course.—Unless the primary condition causing the stenosis is one which can be arrested by treatment, the condition is progressive, and eventually the area of lung beyond the obstruction becomes permanently functionless.

Diagnosis.—Bronchial cicatrisation must be differentiated from obstruction due to extrabronchial causes, such as pressure from new-growths, aneurysm and the other mediastinal conditions mentioned in the section below. The history, the physical signs and examination by X-rays and, if necessary, by the bronchoscope may help in distinguishing. The Wassermann reaction should be investigated in every case where the stenosis is proved to be of intrabronchial origin.

Prognosis.—This varies with the cause. It is most favourable in cases due to syphilis submitted to treatment at an early stage.

Treatment.—Vigorous anti-syphilitic treatment should be employed in cases due to syphilis. In other cases the treatment is to relieve symptoms by appropriate measures.

(b) EXTERNAL CAUSES

These may be subdivided into—(1) *Mediastinal conditions*, chiefly enlargement of the bronchial or mediastinal glands from tuberculosis, Hodgkin's disease or malignant growth, aneurysm of the aorta, mediastinal abscess, pericardial effusion and oesophageal new-growths. (2) *Intrapulmonary causes*, generally primary or secondary new-growths.

Symptoms.—These are practically identical with those just described, but in addition there are those of the condition causing the pressure.

Diagnosis.—This has been discussed in the previous section. The bronchoscope should not be employed where there is any suspicion of an aneurysm.

Prognosis.—This is extremely unfavourable, except in cases due to tuberculous glands and pericardial effusion, and in some cases of mediastinal suppuration.

Treatment.—This can only be palliative in the majority of cases. Useless cough may be checked by a sedative linctus of diamorphine (heroin) or morphine. Dyspnoea when due to spasm may be lessened by inhalations of creosote and spirits of chloroform, or by administration of oxygen. Pain may be relieved by aspirin or other analgesic drugs.

ASTHMA

The term asthma has been loosely employed to denote any form of dyspnoea of expiratory type occurring in paroxysms. For all conditions other than that now to be described some descriptive qualification should be employed to avoid confusion.

Asthma or true spasmodic asthma is a paroxysmal affection, occurring most frequently in patients of neuropathic inheritance. It manifests itself in attacks of severe expiratory dyspnoea due to excessive vagal discharges, set free by peripheral irritation, chemical agencies or cerebral influences.

Ætiology.—Probably no other disease shows such a varied and complex causation, but studies of idiosyncrasy and anaphylaxis have served to explain many of the obscurities.

Predisposing causes.—*Age.*—The first attack may occur at any age, even as early as the period of the first dentition. The majority of cases begin before the age of 25.

Sex.—Asthma is generally stated to be nearly twice as frequent in the male sex as in the female.

Heredity.—Asthma certainly runs in families. The heredity is not always direct, the nervous instability sometimes being evidenced in other generations by migraine, epilepsy or hysteria. The view that hypersensitiveness to certain proteins is inherited is now discredited, and it is believed that an unduly irritable bronchial centre is the factor transmitted by heredity.

Other diseases.—Gout and syphilis are said to predispose to asthma. Bronchitis not infrequently leads to paroxysms in patients with asthmatic tendencies. Tuberculosis of the lung occasionally induces it, but here again it is probably in patients with the asthmatic diathesis.

Climate and locality.—Asthmatics seem very sensitive to both of these, but no general relationship can be proved, as the effects are most variable. Some patients are better in dry, others in damp, foggy climates, and in regard to locality each patient is a law to himself.

Conditions of the nose and naso-pharynx.—Nasal obstruction from swelling of the turbinates, deflection of the septum, spurs and polypi, and conditions of the naso-pharynx, such as adenoids and enlarged tonsils, undoubtedly predispose to asthma, and may also be exciting causes of the actual paroxysm.

Exciting causes.—Chemical substances.—The emanations from certain animals may be the determining cause. The best known of these are the horse and cat, but rabbits, hares, guinea-pigs, deer, dogs and monkeys may have a similar effect. Even human hair appears capable of discharging the paroxysm. The dust from some substances, such as corn, rice or oats, the smell of certain drugs, such as ipecacuanha, and the scent and the pollen of grasses and flowers may act in a similar fashion, as also may articles of diet, and many drugs. This factor in causation has attracted much attention—in this country by Freeman, Coke and Bray, and in America by Walker. It is claimed that at least 50 per cent. of asthmatics show hypersensitiveness to various protein antigens obtainable from animals, grains, bacterial bodies, foods and drugs, and over a hundred are now available for routine testing of these patients. The analogy with the causation of hay fever and paroxysmal sneezing is obvious. This group has been referred to as “allergic” asthma.

Peripheral irritation.—As already mentioned, irritation of the nose, naso-pharynx and bronchi may be asthmogenic in those of asthmatic tendency.

Gastro-intestinal disturbance.—This is well recognised as a cause, and most asthmatics find by experience the penalties of a heavy late meal and of indigestible articles of diet. It is possible that actual metabolic errors may be a factor, as in the so-called “week-end asthma,” due to altered conditions of diet and exercise at this period.

Genito-urinary conditions, particularly in women, notably ovarian or uterine disorders, sometimes act in inducing asthma.

Cutaneous.—Asthmatics are peculiarly liable to urticaria and eczema, although these conditions usually alternate with the asthmatic attacks.

Nervous factors.—Fatigue, emotion and nervous shock may precipitate an attack. This factor cannot be ignored, even in cases due to protein hypersensitiveness, as is shown by a well-known case in which a patient susceptible to roses developed asthma when handed an artificial rose.

Pathology.—Numerous theories have been propounded to explain the asthmatic paroxysm. Among these may be mentioned vascular turgescence of the bronchial mucous membrane, spasm of the bronchial muscle and increased secretion of the mucous glands. Spasm of the diaphragm or of the inspiratory muscles has also been suggested. That bronchial spasm plays the major part seems to have been established by the experiments of Brodie and Dixon, and this view is strongly supported by their observations on the effects of drugs on the bronchial musculature. Muscarine, pilocarpine and physostigmine produce bronchial constriction and asthmatic symptoms

in animals, while atropine, hyoscyamine and chloroform abolish these effects.

There can now be little doubt that the broncho-constrictor fibres of the vagus are the channel by which the impulses discharging the asthmatic paroxysm reach the bronchi, although the possibility that impulses leading to vaso-dilatation and to increased bronchial secretion are also concerned, must be admitted.

ANAPHYLAXIS.—The important part played by extraneous proteins in the genesis of asthma and the obvious analogy between the asthmatic paroxysm and the symptoms of anaphylactic shock have suggested that in many cases, if not in all, asthma is an anaphylactic phenomenon. Evidence is accumulating in support of this view. It has been shown that the lungs of the guinea-pig killed in anaphylactic shock show extreme constriction of the bronchioles. Asthmatics are well known to show anaphylactic tendencies, and especial care in the administration of antitoxic serums is necessary with them. It is of some interest to note that the Eppinger and Hess group of vagotonics show urticaria, dermatographia, eosinophilia and liability to anaphylactic shock, all conditions which occur in asthmatics. It is tempting, therefore, to assume that the foreign protein or toxin produces the asthmatic attack by inducing vagotonicity. Lastly, the observations of Freeman, Coke and the American workers have demonstrated the cutaneous hypersensitiveness of many asthmatics to special foreign proteins. Further research is needed before it can be accepted that anaphylaxis accounts for all cases of asthma, but it is almost certainly an important factor in many.

Symptoms.—The asthmatic paroxysm most commonly commences about 2 a.m. or later, but it may sometimes develop in the daytime. There are often preliminary indications some hours beforehand, constituting the "asthmatic aura." These include restlessness, irritability, mental exaltation, less frequently depression, itching of the nose or chin, flatulence or polyuria. Some attacks are ushered in by coryza. Such warnings are not constant, and the sufferer usually wakes from sleep with a feeling of suffocation. In early attacks great restlessness, anxiety and alarm occur. The difficulty in breathing and the sense of suffocation increase; the patient sits up in bed, or gets up to throw open the window, and fixes his arms to bring into action all possible muscles of respiration. Respiration, although laboured and difficult, is often slow, inspiration being short while expiration is greatly prolonged. Both are accompanied by loud wheezing sounds, audible at a distance from the chest. The patient appears pale, but the lips are dusky and the expression is anxious and distressed. The jugular veins are distended and prominent. The accessory muscles of respiration are seen to be in violent action, notably the sterno-mastoids, scalenes and pectorals. The skin is moist and there may be marked sweating. The chest is much distended, and at each violent attempt at inspiration very little further enlargement occurs, while there is often sucking-in of the supra-clavicular and lower costal regions.

Percussion reveals marked hyper-resonance and encroachment on the cardiac and hepatic dullness. On auscultation inspiration is short and high-pitched, expiration very prolonged, and both are obscured by abundant sonorous and sibilant rhonchi, and later by bubbling râles at the bases. The

pulse is small, quick and sometimes irregular. There is usually marked epigastric pulsation. A differential blood count during an attack may show an eosinophilia of as much as 35 per cent. Cough does not develop until late in the paroxysm, and is quickly followed in many cases by the expectoration of small pellets, called "perles" by Laennec, and often likened to boiled sago or tapioca. These were carefully studied by Curschmann, and when examined on glass on a black background, prove to consist of a central highly refractive mucinoid coil, with masses and threads of mucin wrapped spirally around it. Microscopically leucocytes, mostly eosinophils, may be seen entangled in the mucus. The sputum frequently contains Charcot-Leyden crystals, which are now accepted as spermin phosphate. With the onset of expectoration the dyspnoea quickly lessens, and the attack subsides. The patient often passes a large quantity of pale urine and then may sleep until morning, awaking in apparent comfort. More frequently he appears pale, tired and anxious.

Course, Complications and Sequelæ.—Such an attack may last from a few minutes to several hours, and may remit and then return. When the spasm is very severe and prolonged into hours, with little or no remission, the condition is often termed "status asthmaticus." The patient may be extremely ill, and death may occur unless the attack remits spontaneously or as a result of treatment. More often the attacks recur at the same time each night for a considerable period extending to weeks, and then pass off, after which the patient may enjoy a period of freedom of weeks or months. The intermissions may become shorter with successive attacks, and increasing emphysema may develop. This in turn leads to secondary bronchitis, which persists, together with some degree of permanent oedema of the bases. Later still the cardio-vascular changes incidental to emphysema occur as sequels, namely, engorgement of the right heart, tricuspid regurgitation, venous stasis, ascites and oedema. Chronic asthmatics frequently present a characteristic appearance. Of thin build, with sallow complexion, anxious expression and nervous manner, they often have a long neck, high straight shoulders, and a forward stoop. Asthma necessarily imposes limitations upon those who suffer from it at all severely, although many asthmatics lead active, useful lives in spite of their disease.

Diagnosis.—This involves the differentiation from other forms of dyspnoea, particularly those of spasmodic expiratory type. The chief forms of paroxysmal expiratory dyspnoea are:

1. *Bronchial asthma or spasmodic dyspnoea complicating chronic bronchitis and emphysema.*—This condition is sometimes a late result of true asthma, but may occur independently. The dyspnoea is more persistent and is more definitely related to the bronchitic attacks, being therefore more common in the winter.

2. *Cardiac dyspnoea or cardiac asthma.*—This, like true asthma, is usually nocturnal, but the signs of failure of compensation in association with valvular or myocardial disease usually make the nature of the dyspnoea clear.

3. *Uræmic dyspnoea or renal asthma.*—This is also not infrequently nocturnal and may be almost indistinguishable from true asthma. Examination of the urine, the urea and non-protein nitrogen content of the blood, usually enable the distinction to be made with certainty. Cardio-vascular changes with high blood pressure are frequently but not invariably present.

4. *Hay asthma* is probably only a severe form of hay fever and is to be regarded as a variety of true asthma.

5. *Pulmonary tuberculosis may be associated with asthmatic dyspnœa.*—The differentiation may not be easy during the attack, but the persistence of apical signs in the interval may give a clue. It is a wise precaution to examine the sputum for tubercle bacilli in all cases of asthma. A low blood pressure in an asthmatic should also arouse suspicion of tuberculosis.

The dyspnœa of laryngeal or tracheal obstruction and of mediastinal pressure can usually be recognised by the fact that it is chiefly of inspiratory type, and may be associated with stridor, instead of wheezing. In all cases of doubt the chest should be examined with the X-rays to exclude aneurysm or new-growth.

Prognosis.—When the disease starts in childhood or in early adult life it may stop spontaneously or be relieved permanently when some causal condition is discovered and treated. During a severe attack the aspect of the patient may be so alarming that a fatal issue may seem imminent, yet death rarely occurs. In chronic cases, the ultimate prognosis is made more serious by the complicating emphysema and bronchitis, and in spite of popular belief, the asthmatic has less than the normal expectation of life.

Treatment.—(a) *During the attack.*—The list of anti-spasmodic drugs and measures employed is a long one, and it is impossible to foretell which will be efficacious, for asthmatics vary as widely in their response to drugs as they do in regard to asthmogenic causes. Drugs may be administered for this purpose by inhalation, by nasal sprays, by the mouth or by hypodermic injection. Adrenaline hydrochloride, in doses of 2 to 5 minims of a 1 in 1000 solution hypodermically, may act with dramatic efficacy if administered sufficiently early, but it should be given cautiously to elderly asthmatics. It may also be combined with pituitary extract, as in the special preparations evatmine, pitrenalin and asthmolysin. In status asthmaticus, the procedure suggested by Sir Arthur Hurst may give relief. A syringe of 1 c.c. capacity is filled with adrenaline solution 1 in 1000. This is slowly injected over a period of several minutes to half an hour or until the spasm relaxes. Ephedrine hydrochloride, in tablets of gr. $\frac{1}{4}$ to $\frac{1}{2}$, has proved itself a useful substitute for adrenaline in some cases and can be given by the mouth. Pseudo-ephedrine in doses of $\frac{1}{2}$ to 1 grain is often helpful where ephedrine fails. Ephetonin, a synthetic preparation of similar character, is also sometimes employed. Adrenaline often proves helpful as a nasal spray, especially in combination with chloretone. A weak solution of cocaine and atropine in an oily excipient has been much employed as a nasal spray, but it is not devoid of risk if used indiscriminately. The fumes of burning nitre paper, or of a powder composed of tobacco, stramonium and nitre, sometimes help to relieve the distress, but they should be avoided in cases with bronchitic complications. Smoking a cigarette or a cigar may be helpful in patients who do not smoke habitually; others are helped by cigarettes containing stramonium. Inhalations of amyl nitrite, ethyl iodide or chloroform may be tried in some cases. Various drugs have been employed, of which potassium iodide and bicarbonate with tincture of stramonium, hyoscyamus, lobelia or belladonna are the most useful. Twenty minims of liquid extract of grindelia every 20 minutes for three doses have been found useful in some cases. Other drugs which have been recommended are chloral hydrate, phenacetin and the other coal

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tar antipyretic drugs, elixir of caffeine tri-odide (eupnine) in 60 minim doses, and an emulsion of benzyl benzoate, 120 minims every 2 hours. Other measures include drinking a cup of strong coffee, the application of a mustard leaf over the sternum, and placing the feet in hot water and mustard. In very severe cases, if all else fails, it may be necessary to inject morphine or diamorphine (heroin), but this should only be done after careful consideration, owing to the danger of inducing habit particularly with heroin. It is especially dangerous in cases of status asthmaticus.

(b) *Between the attacks.*—The greatest care should be taken to discover and deal with any predisposing or exciting cause. The patient should live in that locality which his experience shows to be most suitable for him, and at present no rules can be formulated in advising on this matter.

Diet requires careful consideration. Any article of diet to which the asthmatic is susceptible should be entirely eliminated, and only the lightest of meals should be taken after midday. Dextrose has proved to be helpful in some cases of asthma in childhood. It is recommended to give 3 teaspoonfuls in lemonade or orange juice 3 times a day, with extra sugar and sweets at meals. Alkalis may also be given at the same time. Fatigue, overwork and emotional stress are to be avoided. Care should be taken to see that the bowels act efficiently. The general health should be maintained by every possible means. Arsenic may be given by the mouth or intravenously or intramuscularly as sodium cacodylate (gr. $\frac{1}{2}$ in min. 15 sterile water). When the patient is having a series of attacks, iodide of potassium with one or more of the anti-spasmodic group of drugs such as stramonium, lobelia, belladonna and grindelia, may be given regularly with great benefit. Any local source of irritation in the nose or naso-pharynx should be dealt with adequately. Sometimes touching the nasal septum with the galvano-cautery may alone be efficacious. In cases complicated by bronchitis, the sputum should be examined bacteriologically, and a vaccine may be made from the predominating organisms, but small doses and very gradual increments should be employed, since asthmatic patients are frequently hypersensitive to vaccines. If these prove to be *Micrococcus catarrhalis*, or Friedländer's pneumobacillus, great benefit may result, but the patient should be told that the vaccine can only help the asthma by lessening the accompanying catarrh. Some cases associated with marked emphysema obtain considerable relief from compressed air baths, at first on alternate days, then daily, the course extending to 1 or 2 months.

Careful investigation of the question of protein hypersensitiveness should be undertaken, and the method of testing by means of the cutaneous application of various antigens is worth a trial. For this purpose, the particular protein antigen, or a series of such antigens, may be applied to the skin of the forearm in the form of powder, solution or paste, and superficial scarification is then effected by means of a sterile needle or scalpel. A positive reaction is shown by the development of an urticarial wheal surrounded by a hyperæmic area. A control scarification with normal saline or a paste free from protein should be made at the same time. A positive result may be expected in about 50 per cent. of asthmatic patients. If such a condition is established to one or more such substances, they should be avoided if possible; if not, the methods of desensitisation may be tried, but the results are frequently disappointing. The specific antigen

may be employed in very minute doses by injection, starting, for example, with 1 minim of a 100:1000 solution and gradually increasing. Peptone given by the mouth or by hypodermic injection is sometimes employed as a shock desensitising agent.

A variety of "shock" treatment which has given good results is the intramuscular injection of sulphur oil (*huile soufrée*), 0.03 gramme in 1 c.c. This is given in doses up to 1 c.c. once or twice weekly over a period of weeks or months.

A gold salt, allochrysin, has been given intramuscularly in doses of 0.05 gramme, followed in a week by 0.1 gramme, and then, if tolerated, up to 0.2 gramme at weekly intervals till a total amount of 2 grammes has been given.

Breathing exercises of expiratory type such as those recommended by the Asthma Research Council are often of great value.

BRONCHIECTASIS

Definition.—Bronchiectasis is a condition of permanent dilatation of one or more bronchi. When it occurs in the finer divisions it is sometimes described as bronchiolectasis.

Ætiology.—Bronchiectasis is invariably secondary, and may result from disease of the bronchi, the lung parenchyma or the pleura. Even the rare congenital cases are probably consequent on malformation, atelectasis or intra-uterine disease.

1. The bronchial conditions which may progress to dilatation are bronchitis, and any affection leading to partial bronchial obstruction, such as inhaled foreign body, tumour (simple or malignant), stenosis from cicatrization and external pressure from new growth or aneurysm. Localised pulmonary collapse thus induced seems to be the commonest antecedent condition. In children, measles and whooping-cough are not uncommon causes, especially when they follow one another in rapid succession, although either alone, if severe, may lead to it.

2. Conditions of the lung parenchyma which may cause bronchiectasis are unresolved pneumonia, broncho-pneumonia, collapse, syphilis and tuberculosis. Syphilis is rare and usually acts by leading to bronchial obstruction or stenosis. Fibroid tuberculosis is a common cause, but the clinical manifestations are as a rule masked by the primary condition. The pulmonary complications of influenza are not infrequently followed by bronchiectasis.

3. The pleural conditions which are followed by bronchiectasis are those which lead to pleural adhesion and those which are associated with pulmonary fibrosis, notably chronic pleural thickening, or empyema leading to prolonged or permanent collapse of the lung.

In a lesion with such diverse antecedents the age relations are necessarily indefinite. It may occur at any age, but is commonest in the third and fourth decades. It frequently commences in childhood, although the characteristic clinical manifestations may not develop until adult life.

Sex.—In most recorded statistics there is a striking preponderance in the male.

Social state.—It is noteworthy that bronchiectasis in its more severe form is more common in the poor than in the well-to-do.

Pathology.—Four factors in the pathogenesis of bronchial dilatation have to be considered. (1) The most important is the localised collapse which leads to secondary bronchial dilatation. (2) Weakening of the bronchial walls. Most of the conditions preceding bronchiectasis tend to induce severe bronchitis and peribronchitis, and thus render the walls more yielding. Where stagnation of secretion occurs, septic and putrefactive organisms develop, producing tryptic ferments which may act injuriously upon the lining membrane. The importance of the infective factor has been stressed by Moll. (3) Increased pressure on the walls thus weakened is the determining factor. This is generally expiratory in origin and due to the strain of cough. The actual pressure of secretion accumulating behind an obstruction may promote yielding of the bronchial walls. In cases of bronchiectasis following on collapse of the lung the force of inspiration has been regarded as contributory, but this is doubtful and in any case is less important than the expiratory strain of cough. (4) The fourth possibility is the traction exerted upon the walls of the bronchi by contracting connective tissue in the surrounding fibroid lung. This obviously postulates the existence of pleural adhesion, which is not invariably present. While this must be admitted as a possible contributory factor, its importance is certainly less than that of the preceding ones.

Congenital bronchiectasis is a pathological rarity and may be confused with congenital cystic disease of the lung (see p. 1222). It is usually unilateral, and the bronchi involved are of small size, although in some cases the lung may show a large central cavity, with smaller spaces around it. Bronchiol-ectasis is also more of pathological than of clinical interest. It occurs chiefly in children, as the result of acute broncho-pneumonic processes. It is said sometimes to follow influenza and possibly tuberculosis. The lung has a peculiar spongy appearance, to which the name "honeycomb" has been applied.

Bronchiectasis of the larger tubes may be either cylindrical or saccular. In the former condition several of the bronchi are more or less uniformly dilated, and when opened out they appear like the fingers of a glove. Sometimes the dilatations are fusiform, at others they show a beaded arrangement, described as moniliform. These forms of dilatation are usually associated with emphysema and chronic bronchitis. Saccular bronchiectasis is generally localised and may be found in any part of the lung, but is most common in the lower lobes and near the base. This is partly due to the fact that the antecedent processes fall with special stress on the bases of the lungs, and partly to the influence of gravity in leading to retention of secretion in these parts. Although it may be unilateral in origin, it often spreads and may involve both bases or even all the lobes. There may be one large saccular cavity, or a series of smaller globular dilatations involving the whole of the walls of one or more bronchi. The cavities are usually filled with a fetid secretion, to be described under expectoration. When this is removed away the walls are found to be thin, smooth and formed of a single layer of mucous membrane. In places this may have ulcerated, owing to the caustic action of the secretion, and the lung tissue is thus exposed. If such a cavity may then form, and an aneurysm sometimes develops, as in a saccular cavity. The openings of the smaller bronchi, derived from the bronchus, can often be recognised in its walls. In doubtful cases the

histological demonstration of cartilage and muscle in the walls establishes the bronchial origin of a cavity. The surrounding lung tissue is usually airless and fibroid, and sometimes is almost of leathery consistence. Occasionally, however, it is emphysematous, congested or pneumonic. In the great majority of cases there is a dense pleural adhesion over the area of lung involved.

Other morbid conditions found post mortem include lardaceous disease, gangrene of the lung, empyema, pyo-pneumothorax, suppurative pericarditis and cerebral or spinal cord abscess. Owing to the obstruction of the pulmonary circulation which may result, engorgement and dilatation of the right side of the heart, tricuspid regurgitation and the results of systemic venous stasis are often found.

Symptoms.—The onset is usually insidious, the symptoms developing during the course, or as a sequel, of one of the acute or chronic affections mentioned above. In some few cases, however, they develop rapidly in patients previously in good health. This is particularly the case where bronchiectasis results from an inhaled foreign body or after general anæsthesia, and a rapid onset should lead to the suspicion of this. The cough in well-developed cases is somewhat characteristic and occurs in paroxysms. These are frequently induced by change of posture—for example, bending forward or lying down. They occur with special frequency on rising, and are usually associated with the expectoration of large quantities of sputum, due to the overflow of the secretion, accumulated in the cavities during the night, into a sensitive or relatively healthy bronchus, which excites cough reflexly. They also occur on retiring to bed and at long intervals during the day. The sputum frequently amounts to as much as 20 or 30 ounces in the 24 hours. It is generally extremely fetid, although in the earlier stages it is not invariable. The patient's breath is often also malodorous, and the stench may pervade the room or even the house in which he lives, although it is not persistent. The patient is himself much distressed by the unpleasant character of the sputum, of which he is, as a rule, acutely conscious. On standing in a glass vessel it can be seen to settle into three layers—a surface scum of light frothy mucus, an intermediate stratum of thin, turbid, greenish fluid, and a deep layer of brownish colour consisting of muco-pus, bacteria, anaerobes, spirochaetes and putrefactive products, including foul-smelling organic acids. Fetid yellow bodies called Dittrich's plugs can usually be found in the deep layer. Elastic tissue is only present when erosion of the wall has occurred. Hæmoptysis is not infrequent, and may occasionally be fatal. It may be the first and only symptom in some cases, which are referred to as dry or silent bronchiectasis. Dyspnoea is not, as a rule, apparent unless the condition is widespread, or unless the pulmonary or cardiac complications are present. The general condition of the patient is at first but little affected, and there may be no fever for long periods. As the disease progresses, lassitude, anorexia and some wasting slowly develop, while bouts of fever occur, due to retained secretions or to some complication.

Physical signs vary with the extent and degree of dilatation, and also with the amount of secretion present. In the early stages there is at most slight dullness at one base, with diminished air entry, peculiar sticky, "leathery" râles, and diminished vocal resonance. When bronchiectasis is well developed the signs are almost characteristic. The patient may appear well nourished and of good colour, although on cold days, especially

in children, duskiness or cyanosis is often noticeable. There is well-marked clubbing of the fingers, generally of drum-stick character, and pulmonary osteo-arthropathy, involving many joints, sometimes develops. There may be localised flattening or retraction of the chest wall over the affected area, with diminished movement, and the heart is drawn over to this side. The remaining signs vary with the state of the cavity. If this is full, there is diminished vocal fremitus, dullness and weak or absent breath-sounds and voice-sounds. If the cavity is empty or partly empty, the vocal fremitus is increased, the percussion note is boxy or dull, while the breath-sounds are bronchial or cavernous. Adventitious sounds are then generally audible, the most characteristic being sharp metallic or "leathery" râles. Bronchophony and pectoriloquy are marked, and occasionally the "veiled puff" of Skoda can be heard. Signs of bronchitis are often apparent in the adjacent lung tissues; compensatory emphysema may be demonstrable in the unaffected parts of the lung, and on the opposite side. X-ray examination before and after the injection of lipiodol or neo-hydriol serves to define the extent of the disease and the degree of fibrosis. Tomography may also be useful.

Complications and Sequelæ.—The chief pulmonary complications are septic broncho-pneumonia, gangrene and abscess. The pleura may become involved, giving rise to dry pleurisy, which sometimes progresses to empyema and rarely to pyo-pneumothorax, while in other cases pleural adhesion and contraction result. Septic pericarditis may develop and prove fatal. Septicæmia and pyæmia sometimes occur as terminal results. Cerebral abscess constitutes a serious and somewhat common complication, and may be found in the frontal, parietal or temporal regions, the cerebellum or cord. Occasionally multiple abscesses form. Lardaceous disease sometimes develops, especially in the liver, kidneys and intestines.

Course.—This is progressive, but is often slow unless fever or complications develop, though the morbid process may eventually involve the other lung. The sputum at first may be simply purulent, then becomes unpleasant and finally fetid. The disease may start in childhood and not lead to death until well on in adult life. The course is slower in cases due to bronchitis and fibroid lung conditions than in those due to foreign bodies, new growths or aneurysm.

Diagnosis.—In well-developed basic cases this is, as a rule, easy. The history of cough, influenced by posture and associated with copious sputum, is suggestive, especially when variable physical signs are observed. The development of the characteristic sputum with these signs renders the diagnosis almost certain, and the X-rays usually serve to confirm. Radiological investigation after an intra-tracheal injection of 10 to 20 c.c. of lipiodol or neo-hydriol, through the crico-thyroid membrane or between two rings of the trachea, under local anæsthesia, or with care directly between the vocal cords, has greatly facilitated the diagnosis of bronchiectasis. Franklin has recommended the nasal route for the introduction of the lipiodol. One nostril, the oro-pharynx and the larynx are cocaineised, then a gum-elastic catheter is passed along the floor of the nose into the larynx. Some cocaine is injected down the catheter and then the lipiodol follows. An attempt should be made to direct the lipiodol towards the affected side by turning the patient towards that side. The injection should be carried out in the X-ray room and the patient instructed to restrain cough if possible until the films have

been taken. The pictures obtained are strikingly characteristic and of great value. In cases with less characteristic symptoms and signs the distinction has to be made from chronic bronchitis, especially the fetid variety, pulmonary tuberculosis, gangrene or abscess of the lung, and fetid empyema. The distinction from chronic bronchitis may be difficult, especially in the early stages when the sputum is not fetid, but the paroxysmal cough, the copious expectoration with signs including bronchial breathing and sticky râles at the base, may be strongly suggestive. In fetid bronchitis the fetid sputum is not constant, and the cough and sputum may occur only during exacerbations of the bronchitis. Pulmonary tuberculosis may give rise to difficulty, particularly in cases of apical bronchiectasis. Repeated examinations for tubercle bacilli and also for elastic tissue in the sputum should be made. The history, the mode of spread, and X-ray examination may all assist. It should be remembered that the two conditions may coexist and, this may be suspected in some cases of fibroid tuberculosis with basic excavation. Abscess and gangrene of the lung have a more acute onset and course, but the chronic cavities left by these conditions may give rise to difficulty. In such cases the history may be an important aid in diagnosis. In fetid empyema rupturing through the lung, particularly when of interlobar origin, the patient is generally acutely ill, there may be a history of pleurisy at the onset and possibly some evidence of mediastinal pressure or cardiac displacement. The rare condition of congenital cystic disease of the lung may give rise to some difficulty (see p. 1223).

Prognosis.—This varies with the cause. {If due to aneurysm or growth, the duration is determined by these conditions. Bronchiectasis induced by a foreign body is generally permanent, even when the latter is removed, but it is not progressive. If the foreign body is not removed, complications generally supervene, and the course may be rapid. In bronchiectasis due to bronchial or pulmonary disease the course may extend into years, particularly if treatment is followed strictly, but sooner or later toxæmia and general or local complications supervene, with the result that the duration of life is inevitably considerably shortened.

Treatment.—Prophylactic treatment in cases of chronic bronchitis, delayed resolution in pneumonia and other conditions tending to fibrosis is of the utmost importance. This comprises respiratory exercises, climatic treatment, inhalations and vaccines.

The medical treatment of bronchiectasis consists in measures to promote the general health and well-being of the patient, to secure efficient emptying of the cavity, and to lessen or control the putrefactive processes occurring in it. The first of these involves a careful mode of life, adequate rest and change, a good and digestible diet, and medicines such as cod-liver oil, iron, quinine, strychnine or arsenic. The evacuation of the cavity may be promoted by postural methods, such as bending over the edge of the bed or stooping forwards. This can now be effectively secured by treating the patient on a Nelson bed. By X-ray and iodised oil (lipiodol) the exact position of the cavities in relation to the trachea can be determined, and the patient placed in the position best adapted to secure effective drainage. Expectorants, especially of antiseptic character, may be given, such as creosote, terebene, tar preparations, balsam of tolu or Peru, compound tincture of benzoin or the benzoates. If the sputum is tenacious, or if much bronchitis coexists iodides

and alkalis may be given in an ordinary expectorant mixture. To lessen the fetor, creosote is most frequently given in perles of 3 to 5 minims three times a day after food, or in an emulsion with cod-liver oil. Guaiacol carbonate and other creosote derivatives may also be tried. Syrupus *allii*, in 60 minim doses, is sometimes given, and is of value; but it is not always well tolerated by patients on account of its taste and tendency to repeat. Although these drugs are helpful, the amount of antiseptic reaching the cavity by the blood must necessarily be small. Attempts to secure more direct application by intratracheal injection and by inhalation have been made. The former method is now seldom used.

For inhalation purposes, solutions of volatile antiseptics are employed on a Burney-Yeo mask, such as creosote, terebene, menthol or eucalyptol in spirits of chloroform. The mask may be worn almost continuously or at intervals during the day. The creosote vapour bath is, however, the most satisfactory form of inhalation treatment, and is of great value. This should be given in a concrete-floored room without furniture. The patient is covered with a smock, the eyes are protected by closely-fitting goggles, and the nostrils by cotton wool plugs. A small quantity of creosote is heated in a metal dish, on a tripod over a spirit lamp. When the patient inhales the vapour, which quickly fills the room, violent cough is excited and the cavity is emptied. The ensuing deep inspirations carry down creosote-laden air into the air passages. The baths should be at first given on alternate days and last from 10 to 15 minutes. When the patient becomes accustomed to them, they may be given daily for half an hour or longer. The results are often strikingly beneficial. Vaccines made from the predominant organisms found in the sputum have been given with benefit in some cases. Surgical treatment is now more often employed than formerly. Repeated washing-out through a bronchoscope, at weekly intervals, is often helpful, giving comfort to the patient by diminishing the amount of sputum, and lessening or abolishing its fetor. Induction of artificial pneumothorax is sometimes of value, especially in early cases, in which it may be successful. Unfortunately it is often impracticable, owing to adhesions, and even in cases in which it is carried out, the beneficial effect only persists as a rule while the collapse is maintained. Temporary or permanent paralysis of the phrenic nerve has also proved helpful, especially in localised basal cases and in those secondary to abscess of the lung. Lebeotomy or pneumonectomy is proving a satisfactory and effective method of cure in unilateral cases. The mortality from these operations is rapidly lessening with recent improvements in technique.

INJURY

External trauma applied to the chest-wall may cause rupture of a main bronchus. This is especially liable to occur after severe crushing accidents. One or other of the main bronchi may be completely severed from the trachea. The chief clinical feature presented in such a case is emphysema of the neck and upper portion of the chest-wall. Death usually ensues in 2 to 3 days.

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DISEASES OF THE LUNGS

HYPERÆMIA AND ŒDEMA

Hyperæmia of the lungs may be either active or passive. In the former there is an increased supply of arterial blood through the pulmonary and bronchial arterioles. In passive hyperæmia there is engorgement of the pulmonary venous radicles and capillaries. With both forms there is frequently œdema, due to the exudation of serous fluid into the lung alveoli. The term "congestion" is sometimes employed as an alternative to hyperæmia, but owing to its erroneous popular use it is best avoided.

(a) ACTIVE HYPERÆMIA

Ætiology.—This may occur in association with any acute inflammatory process affecting the bronchi, lungs or pleura. It sometimes results from the inhalation of pulmonary or bronchial irritants, such as poisonous gases or heated air. Severe muscular exertion and exposure to extreme cold are described as causes, but the former at least is doubtful. An important variety is that known as *collateral or fluxionary hyperæmia*, which occurs when there is obstruction to the circulation in the whole or part of one lung, from conditions such as a large or rapidly developing pleural effusion, an extensive and spreading pneumonia, or in association with pneumothorax. This may develop in the sound lung, or in the unaffected parts of that diseased. A primary form of acute hyperæmia, the "*maladie de Woillez*," has been recognised by French authors, but this is generally regarded as a mild or abortive pneumonia.

The clinical manifestations of acute hyperæmia are merged in those of the processes with which it is associated, and therefore do not need separate description.

(b) PASSIVE HYPERÆMIA

Ætiology.—Passive hyperæmia may be produced by (1) conditions impeding the venous return from the lungs; (2) those leading to increased resistance to the passage of blood through the pulmonary capillaries, and (3) failure of the driving power of the right ventricle. The commonest causes of impeded return are left-sided heart lesions causing overfilling of, and increased pressure in, the left auricle. In mitral stenosis it may occur early and sometimes almost acutely, but aortic and myocardial lesions also lead to it, when the left ventricle fails and the mitral valve yields. Direct obstruction of the pulmonary veins sometimes results from external pressure by aneurysm, mediastinal tumour or enlarged bronchial glands, or from obstruction of the lumen by thrombosis. The passage of blood through the pulmonary capillaries may be impeded by emphysema, chronic bronchitis, pulmonary tuberculosis and fibrosis of the lungs. Failure of the right ventricle occurs in the late stages of right-sided heart lesions, with tricuspid regurgitation, and as a late sequel of left-sided failure.

Passive hyperæmia is obviously in the main dependent on mechanical

factors; it is not surprising, therefore, that gravity seems to play a part in the localisation of its effects, which are usually most marked in the bases or most dependent parts of the lungs. In bedridden, enfeebled or old patients, particularly if myocardial weakness or degeneration coexists, this factor becomes of great importance. Not infrequently some degree of œdema of the bases develops, and the condition is then called hypostatic congestion. If such an area becomes infected the resulting process is known as hypostatic pneumonia. Basal hyperæmia and œdema of the hypostatic type also result from toxæmia due to diseases such as enteric fever, from poisoning by drugs such as morphine, and as a terminal event in many cerebral lesions causing increased intracranial pressure.

Pathology.—The pulmonary veins and capillaries are engorged, with the result that the lung is darker in colour and heavier, while the alveolar walls and septa are swollen. If the condition persists for some time, pigment derived from the hæmoglobin of red corpuscles escaping by diapedesis is deposited in the epithelium of the alveoli and in the fibroblasts in the inter-alveolar septa. In long-standing cases the lung is firmer than normal and brownish-red in colour, a condition described as *brown induration*. If any degree of œdema is present, serous fluid is found in the alveoli on post-mortem examination, and on section of the lung frothy serous fluid exudes, which may contain some of the pigmented alveolar cells, constituting what are called “cardiac cells.” Although congested and œdematous lung is heavier than normal, it usually floats in water.

Symptoms.—In slight degrees of hyperæmia these may be absent or negligible. In more advanced cases, they are those resulting from the impeded circulation through the lungs and the deficient aeration which this entails. Dyspnoea is the most prominent symptom, and it is generally a measure of the degree of hyperæmia. It is markedly increased by exertion of any kind, and in extreme degrees it is distressing and eventually alarming. It may be inspiratory or expiratory in type, and in the latter case it is sometimes described as cardiac asthma. In severe cases there is usually orthopnoea. Cough is almost invariably present, and there is usually some expectoration of frothy fluid, which may be blood-stained. The pigmented cells referred to above as “cardiac cells” may be found in it. Cyanosis is common and indicates the degree of anoxæmia. This may be associated with distension of the jugular veins, and there is often obvious distress. As in other forms of cyanosis there is usually some increase in the number of red corpuscles. The vocal fremitus at the bases may be diminished, the percussion note impaired, the breath-sounds weak and accompanied by rhonchi, crepitations or bubbling râles, although these signs are for the most part due to the associated œdema. In addition, the signs of the primary condition in the lungs or heart will be apparent.

Complications.—Pulmonary œdema and infarction are the chief complications.

Course.—If the venous engorgement cannot be removed, it usually tends to become progressively worse, whereas when it results from temporary cardiac embarrassment, recovery is usually complete as soon as the heart function is restored.

Prognosis.—This condition has to be distinguished from (1) chronic bronchitis, in which case there may be some rise of temperature and the

physical signs are more variable and more disseminated; (2) infarction, in which pain and hæmoptysis of sudden onset are the rule.

Prognosis.—This is so entirely dependent upon the nature and degree of the condition responsible for the engorgement that no general rule can be formulated.

Treatment.—In elderly patients, or those likely to be confined to bed for long periods, attention should be directed to the decubitus. This should be changed frequently, and if possible the patient should be permitted to sit up or to get into a chair, and encouraged to take a few deep breaths several times during the day. If the hyperæmia is associated with cyanosis and engorgement of the right heart, bleeding to the extent of 8 to 12 ounces may be helpful. If this is not practicable, the application of 6 leeches over the liver, or dry cupping of the bases of the lungs may be tried. Free purgation and the administration of diuretics, notably injection of mersalyl (salyrgan) or neptal may also help indirectly to relieve the engorgement. In cases associated with cardiac failure, the administration of cardiac tonics, such as digitalis, strophanthus or squills, the injection of strychnine, camphor in oil or nikethamide (coramine) may all be of assistance. Moderate hæmoptysis should not be checked, and cough, if effective, may be promoted by suitable expectorants. In cardiac cases a "régime lactée" or strict milk diet is advocated by some French physicians.

(c) ACUTE OR HYPERACUTE PULMONARY ŒDEMA

In this condition flooding of the alveoli with the serous exudate from the pulmonary capillaries occurs with great rapidity.

Ætiology.—It is more commonly met with after the age of 40 than before, although cases have been recorded in children. It is considerably more frequent in the female than in the male sex. Arterial disease and hypertension are the most common antecedents, but acute or chronic renal disease and pregnancy may all act as predisposing factors. It sometimes occurs in diabetes. The actual exciting cause is often obscure, and probably varies in different cases. A heavy meal, an epileptic fit, or the administration of an anæsthetic may be the immediate cause in those predisposed. In other cases it may be a manifestation of angio-neurotic œdema. Sometimes paracentesis of a pleural effusion is quickly followed by œdema, no doubt as a result of a collateral hyperæmia. It has occurred after "gassing" by chlorine. In diabetes the lipæmic condition which sometimes occurs has been suggested as the determining factor, possibly causing multiple fat embolism. Coronary occlusion and acute left ventricular failure are noteworthy causes. In some cases dissociation of the action of the two ventricles has been supposed to be the cause, the right contracting forcibly while the left is in an enfeebled or asystolic condition. In support of this contention may be added the fact that acute pulmonary œdema has been observed after rupture of the chordæ tendinæ of the mitral valve.

Pathology.—The alveoli are found to be flooded with a thin serous exudate. The lungs are heavier than normal, sodden, and on squeezing exude large quantities of greyish-yellow or pinkish fluid. Frothy fluid of similar character is found in the bronchi and even in the trachea and nasopharynx in hyperacute cases.

Symptoms.—The onset is sudden, and generally occurs when the patient is lying down, hence being most frequently observed at night. The patient awakes with intense dyspnoea, and a sense of suffocation, then frequently rolls or rushes about in the endeavour to breathe, even clutching at the throat. Cyanosis is present, and the aspect is one of anxiety and alarm. Frothy fluid, often pink in colour, may soon stream from mouth and nose, or be brought up in great gulps. The chest movements are hurried, and the accessory respiratory muscles are in violent action. Vocal fremitus is diminished over the lower lobes. The percussion note soon becomes impaired over the lungs, commencing at the bases. The breath-sounds are at first vesicular or harsh with prolonged expiration, then become faint and may be obscured by bubbling râles or crepitations, audible all over the chest. Voice conduction is diminished.

Complications and Sequelæ.—Owing to its acute and rapid course, complications do not occur. Bronchitis may result as a sequela.

Course.—The malady usually lasts only minutes or hours. Unless it remits, or treatment affords relief, the patient rapidly becomes unconscious and death follows, the heart continuing to beat after respirations have ceased.

Diagnosis.—The affection is usually so characteristic that the diagnosis is obvious. In the more protracted cases the dyspnoea and the physical signs are not unlike those of acute suppurative bronchitis or suffocative catarrh and broncho-pneumonia; but in both of these there is some degree of fever and the expectoration is less copious, and when it occurs is usually of purulent or muco-purulent character. The nocturnal onset of œdema may suggest asthma; but the physical signs and the late and scanty expectoration in the latter suffice to distinguish it.

Prognosis.—The prognosis is always very grave; but prompt treatment has saved some cases. Death may occur in less than 10 minutes, or be delayed for 24 or 48 hours. In the angio-neurotic type repeated attacks may occur.

Treatment.—The most successful treatment is the immediate subcutaneous injection of gr. $\frac{1}{4}$ morphine. Good results have also followed the injection of gr. $\frac{1}{100}$ – $\frac{1}{80}$ atropine sulphate hypodermically. These are often given together. Oxygen inhalation by nasal catheter or special mask such as the B.L.B. variety may be used. Prompt venesection has been recommended, and should be tried if possible.

(d) CHRONIC PULMONARY ŒDEMA

This is usually the sequel of chronic passive hyperæmia, and the causes and symptoms are those of that condition. It may also occur in chronic renal disease. In marked degrees of œdema, however, the signs may closely simulate those of pleural effusion, save for the displacement of the cardiac impulse. It is important to remember that some degree of hydrothorax may occur as a complication, and increase the difficulty in diagnosis.

INFARCTION OF THE LUNGS

Infarction of the lungs or “pulmonary apoplexy” results when a branch of the pulmonary artery becomes occluded by embolism or thrombosis.

Ætiology.—*Embolic forms.*—The obstructing plug may originate in any part of the systemic venous system, in the right side of the heart or on its valves or in the pulmonary artery itself. The commonest peripheral cause of embolism is detachment of a thrombus in cases of thrombo-phlebitis. This may occur in the veins of the lower extremity, or in those of the uterus after childbirth. Thrombosis with embolic detachment may also develop in prolonged or wasting diseases, such as enteric fever, tuberculosis and cancer; in acute processes, such as influenza, septicæmia and pyæmia; and in localised septic lesions, such as otitis. Pulmonary embolism is not infrequently observed after abdominal or pelvic operations, and after the radical cure of hernia or hæmorrhoids.

Intracardiac thrombi from the right auricle or ventricle, becoming detached, lead to embolism, and this occurs especially in cases of right-sided heart failure secondary to left-sided valve lesions. Vegetations forming on the tricuspid or pulmonary valves in septic endocarditis on detachment produce pulmonary infarction. Rarer causes are fat embolism after injury to bone or to a fatty liver, the entry of pieces of new-growth or hydatid daughter-cysts into systemic veins, and even air embolism.

The exciting cause of embolism is not infrequently sudden movement or strain leading to detachment of a thrombus or vegetations.

Thrombotic forms.—Thrombosis occurs as a secondary process around pulmonary emboli; but it is probable that some cases of infarction are due to a primary thrombosis. This condition may be produced by some acute or chronic pulmonary disease, such as gangrene, tuberculosis and fibrosis, and by atheroma of the pulmonary artery. Any process leading to chronic venous hyperæmia may also cause it. A rare cause is thrombo-phlebitis migrans.

Pathology.—Although the pulmonary arteries are not strictly speaking end arteries, since there is some degree of anastomosis between them and the bronchial arterioles, yet the result of their obstruction is to produce infarcts comparable with those in other organs. The origin of the blood in the obstructed area has been much discussed. Cohnheim regarded it as the result of regurgitation from the veins, a view subsequently disproved, since the infarct is hæmorrhagic even when the veins are also obstructed. It is now regarded as due to influx from the anastomosing bronchial capillaries into the pulmonary capillaries, and the escape of this blood from the latter owing to their altered nutrition. It is generally accepted that embolism is much more common than thrombosis. It has been suggested that some infarcts are not obstructive, but are the result of hæmorrhage *per rherin* in cases of extreme passive hyperæmia, and that the shape is due to the alveolar distribution of the bronchial area affected. If a large embolus has caused sudden death, it will be found arrested at the bifurcation of a large branch of the pulmonary artery, or even in one of the main divisions of that vessel. In such cases there has not been time for pulmonary changes to occur, and the chief post-mortem condition found is engorgement of the right side of the heart.

In post-mortem examination of cases where smaller emboli have led to infarction, the infarcts are usually found in the lower lobes, more commonly in the right lung. They extend to the surface in the majority of cases, and can be seen before section as slightly raised, dark red areas, with the over-

lying pleura a little roughened from inflammatory exudate. They feel hard and firm, and on section are typically wedge-shaped, with the base on the surface and the apex centrally placed. In the rare deep-seated infarcts a spheroidal form is the rule. When recent, an infarct is dark red in colour, and suggests hæmorrhage with clot formation, hence the term "pulmonary apoplexy." In some cases infarcts have a purplish hue, and are said to resemble the colour of damson cheese; later they change to brownish-red. Infarcted areas sink in water. There may be a single large infarct almost occupying one lobe, sometimes only a small one, or several of varying size and age scattered throughout the lungs. In some cases a fortunate section may reveal the embolus with its ensheathing thrombus, but sometimes a thrombus only is found. Microscopically, the alveoli and finer bronchioles are filled with red blood corpuscles, and there is a sharp delimitation from the healthy lung. If the embolus is infective, suppuration occurs, and abscess or empyema ensues.

Symptoms.—If a large embolus blocks one of the main divisions of the pulmonary artery, there is sudden intense dyspnoea, pain in the chest, distress, cyanosis, and rapid unconsciousness, death resulting in a few minutes from asphyxia. In other cases the patient gives a short cry, and falls unconscious, death occurring almost immediately from syncope. In some cases unconsciousness develops so rapidly, and the respiratory symptoms are so little apparent, that a cerebral vascular lesion may be suspected. On the other hand, life may be maintained for several minutes or even hours, the patient being unconscious or in acute distress and anxiety with urgent dyspnoea, lividity and cyanosis. Respiration is deep and laboured, but fails to give relief to the sense of suffocation. In such cases also, death may result eventually from asphyxia or syncope, or the patient may slowly recover. In less severe forms, such as occur in cardiac and in some post-operative cases, there is sudden pain with difficulty in breathing, followed in a few hours or in a day or two by cough with hæmoptysis or by the expectoration of deeply blood-stained mucus persisting for some days, and slowly clearing up. If the embolus is infective, fever, often of hectic type, results, sometimes delayed for a day or more.

In the severe cases there is cyanosis, distension of the veins of the neck, acute anxiety with exophthalmos and cold, clammy skin. The only physical signs apparent are the deep, laboured breathing, the harsh breath-sounds, and the evidence of cardiac embarrassment with feeble, failing pulse.

In less severe cases the signs are also not characteristic. There are evidences of cyanosis and distress of less urgent character, possibly some limitation of movement on the affected side, increase of vocal fremitus, localised dullness, with weak or absent breath-sounds, and sometimes a pleural rub. In some cases definite bronchial or tubular breath-sounds may be audible. A few fine râles are sometimes present in the adjacent lung areas.

Complications and Sequelæ.—Localised dry pleurisy is almost invariably present. With infective emboli, abscess or gangrene, and later empyema may result. In organisation an infarct leads to a localised area of fibrosis.

Course.—As already described, death may occur from asphyxia or syncope in the course of a few minutes or hours, although recovery occurs in some very severe cases. In the less severe forms, after the initial urgent symptoms

have passed off, recovery is often rapid and uneventful, save for pain, cough and bloodstained expectoration.

Diagnosis.—The dramatic onset, the history and the associated lesions of the veins or heart render diagnosis easy as a rule; but it may be necessary to eliminate other causes of hæmoptysis, notably pulmonary tuberculosis and chronic venous hyperæmia.

Prognosis.—This depends largely upon the initial shock. The prognosis is very grave when the patient rapidly becomes unconscious. As there is less likelihood of sepsis in cases due to cardiac lesions than in those due to localised venous thrombosis, the prognosis is rather better in the former; but, on the other hand, organisation of a clot in a vein may completely remove the source of the emboli, while the source often persists when they are derived from the heart.

Treatment.—The coagulability of the blood may be lowered by the administration of 30 grains of sodium citrate three times daily. This is a wise prophylactic measure in prolonged illness, especially when a milk régime is being enforced. When thrombosis has occurred in a peripheral vein, such as in the leg, the affected limb should be immobilised until organisation of the clot has taken place. Morphine is useful in quieting a patient if there is much mental distress when a pulmonary infarct forms; but usually the patient is collapsed and stimulant measures are indicated. An injection of morphine gr. $\frac{1}{2}$, atropine gr. $\frac{1}{100}$, and strychnine gr. $\frac{1}{30}$ is found of value in some cases. If there is dyspnoea oxygen should be administered. Venesection to the extent of 10 or 12 ounces may be tried in cases where there is marked lividity with a forcibly acting heart. Hæmoptysis, when it occurs, should not be checked. Pain may be relieved by leeches, cupping or by application of iodine. In cases with heart failure the appropriate treatment by cardiac tonics should be administered. A few cases are on record in which immediate surgical aid has been available and the operation of embolectomy has been successful.

COLLAPSE OF THE LUNGS

In collapse of the lungs the alveoli are completely or partly devoid of air. The condition may be congenital, and due to non-expansion of the lung, when it is referred to as atelectasis. On the other hand, collapse may be the result of removal of the air from lung tissue previously expanded, when it is called apneumotosis or acquired collapse. The three terms—collapse, atelectasis and apneumotosis—are, however, used as synonyms by many writers.

ATELECTASIS OR CONGENITAL COLLAPSE

Ætiology.—This condition occurs in still-born and in premature infants, and probably persists to some degree for weeks or even months in weakly children. It may result from immaturity or from weakness of the inspiratory muscles, and from obstruction of the air passages by mucus and meconium. It may be a consequence of disease, such as congenital syphilis or lesions and developmental defects of the nervous system.

Pathology.—Atelectasis is due to failure of the respiratory mechanism

to draw air into the alveoli and expand them, as occurs normally with the first few inspiratory efforts of the newborn infant.

Atelectatic lungs are solid, airless and small. They are usually described as presenting appearances similar to those of adult liver as regards colour and consistence. In partial atelectasis the lung appears mottled, and small expanded areas of pinkish colour may project from the surface. The condition is chiefly of medico-legal and pathological interest.

APNEUMATOSIS OR ACQUIRED COLLAPSE

Collapse of previously expanded lung may be active or passive, the former being due to active shrinking of the lung owing to defects in the inspiratory musculature, the latter to conditions disturbing the pressure relations within the thorax.

1. ACTIVE PULMONARY COLLAPSE.

Synonyms.—Active Lobar Collapse ; Massive Collapse.

Ætiology.—This condition was first described by William Pasteur in 1890 in cases of diphtheria associated with paralysis of the diaphragm. In 1908 he pointed out that it occurred also as a sequel of operations, especially of those upon the abdominal organs, less frequently of those upon the neck and pelvis. It is highly probable that many post-operative lung conditions formerly recorded as pneumonia were in reality due to active collapse. It may also follow after injuries, such as those resulting from falls from a bicycle or a horse. During the War of 1914–1918, when chest wounds were collected in special hospitals, it was found that massive collapse was not infrequently an important complication of penetrating and non-penetrating wounds of the chest. It was also noticed in some cases after severe wounds of the buttocks and pelvis.

Pathology.—The mechanism by which deflation results is obscure, and is the subject of controversy. Pasteur regarded the condition in the diphtheritic cases as due to paralysis of the diaphragm through the phrenic nerves or their nuclei, and in the post-operative and traumatic cases as a consequence of reflex inhibition of this muscle. Briscoe, on experimental, pathological and clinical evidence discards Pasteur's explanation. He maintains that the deflation is caused by an exaggeration of the normal phenomena of breathing in the supine position, in which he states that the crural portion of the diaphragm alone contracts, the costal portion being in abeyance. In the supine position, with quiet breathing, deflation of the lower lobes occurs, and this is promoted by conditions of debility, toxæmia or operation. The clinical manifestations described by Pasteur are regarded by Briscoe as the result of superadded pleurisy, or of inflammation of the crura of the diaphragm.

Boland and Sheret have put forward the suggestion that massive collapse is due to obstruction of the bronchi, followed by removal of the air in the corresponding lung areas by absorption into the blood stream. The obstruction is supposed to be due to increased secretion and the inhibition of the cough reflex.

Post mortem, the lower lobe of one lung is usually found to be deflated and retracted towards the spine. Sometimes the whole of one lung may be affected, or both lower lobes. The collapsed area is bluish-red, firm, does not crepitate and sinks in water. Pleurisy or pneumonic changes may be

seen, and these are regarded by Pasteur as secondary to infection of the deflated lung, the resistance of which is lowered, and by Briscoe as the essential factor in the production of the symptoms. In massive collapse the heart and mediastinum are displaced towards the affected side, and the sound lung is often bulky and distended.

Symptoms.—The symptoms commonly commence within 24 or 48 hours of the injury or operation, although they may rarely be delayed for 5 to 7 days. The onset is generally sudden, with pain in the lower part of the thorax or behind the sternum. Severe dyspnoea quickly follows, and the patient appears dusky, cyanosed and alarmingly ill. Cough, with viscid mucoid expectoration, generally develops in a few hours. The latter may become copious and muco-purulent if pneumonic changes ensue. The pulse and respirations are markedly increased in rate, and the temperature not infrequently rises to 103° F. Occasionally the onset is more gradual with pain and cough, and in some cases of wounds it may give rise to few symptoms and be discovered only on routine examination.

Examination of the chest shows diminished movement on the affected side, and often absence or reversal of epigastric excursion with respiration, whereas the movement on the other side may be exaggerated. In other respects the signs usually simulate those of lobar pneumonia. Over the collapsed lung the vocal fremitus is increased, the percussion note is dull, the breath-sounds are tubular, and bronchophony and whispering pectoriloquy are present; but as a rule there are no adventitious sounds, although occasionally rhonchi and a few fine râles may be heard. In some cases the breath sounds are very weak or almost absent, and voice conduction is diminished. Over the healthy lung, loud and harsh breathing is audible. The displacement of the cardiac impulse towards the collapsed lung is a point of cardinal importance. It is noteworthy that in certain cases of gunshot wounds of the chest the collapse affects the contra-lateral lung.

Complications and Sequelæ.—Bronchitis, lobar pneumonia, or pleurisy may occur as complications. There are usually no sequelæ, except that pleural adhesions may occur.

Course.—The course of the affection is rapid. After periods extending from 2 to 5 days the temperature falls to normal, the symptoms disappear, the lung quickly re-expands, the heart returns to its normal position, and there is complete recovery.

Diagnosis.—The most important conditions from which this malady has to be distinguished are lobar pneumonia, pulmonary embolism, pneumothorax and pleural effusion. The position of the cardiac impulse is often the deciding factor: in collapse it is displaced towards the lung involved, in pleural effusion and pneumothorax it moves away from the affected side, whereas in lobar pneumonia there is usually no cardiac displacement, although there may be dilatation. Labial herpes and blood-stained expectoration are frequently seen in pneumonia, but not in collapse. When in right-sided collapse there is marked distension of the left lung with obliteration of the normal cardiac dullness, the signs superficially resemble those of a left-sided pneumothorax; but with careful examination no such error should be made. The distinction from pulmonary embolism may be difficult at first, but the localisation of the signs, and the blood-stained expectoration, may give useful indications.

Treatment.—**PROPHYLACTIC.**—The administration of morphine and atropine before the anæsthetic, propping up of the patient in bed as soon as possible after it, and the insistence upon periodic deep breaths are useful measures in preventing the onset of lobar collapse. When the "collapse attack" develops oxygen should be administered and pain relieved by local applications, such as leeches or poultices, or by an injection of morphine, atropine and strychnine. Expectorants may be given if the cough is ineffective, and cardiac tonics, such as digitalis, strophanthus and caffeine if there is much cardiac embarrassment. Injections of strychnine or coramine are useful if the patient is collapsed at the onset. Bronchoscopic aspiration has been recommended in cases of post-operative massive collapse.

2. PASSIVE PULMONARY COLLAPSE.

This form of collapse may affect the whole of one lung, or be confined to one lobe or to groups of lobules.

Ætiology.—Total collapse is generally the result of pleural effusion, empyema, pneumothorax or obstruction of a main bronchus. In a large effusion and in pneumothorax collapse is complete, unless the shrinkage is prevented by adhesions. In a smaller effusion, the process may be limited to the lower parts of the lung. Other causes of lobar or partial lobar collapse are conditions leading to complete obstruction of a main bronchial division, particularly new-growth, aneurysm or foreign body. It also occurs in aged or bedridden patients, or in those with enfeebled inspiratory muscles, when prolonged fever has enforced a dorsal decubitus. Abdominal distension from tympanites or ascites can also cause collapse of the bases of the lungs.

Lobular collapse results from any condition impeding the air entry to the smaller bronchi or bronchioles, such as bronchitis, broncho-pneumonia, pulmonary tuberculosis, whooping-cough and diphtheria. Obstruction of the naso-pharynx by enlarged tonsils and adenoids may cause partial collapse, especially in the upper lobes.

Pathology.—The deflation of the lung area may be produced in three ways—(1) By complete obstruction to the air from blocking of a bronchus or bronchiole, the residual air being absorbed; (2) by enfeeblement of the inspiratory mechanism similar to the process in active collapse; and (3) by disturbance of the intrapleural pressure by fluid or air, the lung at first contracting in virtue of its elasticity until the intrapleural pressure becomes equal to that of the atmosphere, when any further accumulation of fluid or air causes positive pressure and compression of the collapsed lung.

Post mortem, in complete or lobar collapse the appearances are similar to those in active collapse. In lobular collapse the deflated areas are contracted and depressed below the level of the healthy lung. They are dark red or slaty in colour, while the adjacent areas are pinkish and often emphysematous. The collapsed areas do not crepitate.

Symptoms.—Total collapse of the lung or of a lobe being usually a secondary process, the symptoms and signs are masked by those of the primary condition, such as pleural effusion, pneumothorax, growth or aneurysm. It can, however, usually be demonstrated by X-ray examination, the collapsed lung being apparent as a fairly dense shadow lying alongside the vertebral bodies. Not infrequently, however, in pleural effusion definite tubular breath sounds, with bronchophony and pectoriloquy, may

be audible in the relatively dull area above the level of the fluid posteriorly, and these signs are due to the collapsed lung. In aortic aneurysm, or less commonly in mediastinal, pulmonary and bronchial neoplasms, distinctive signs due to the local collapse may be apparent. These consist of slightly diminished movement of the corresponding part of the chest wall, with diminution of vocal fremitus and impairment of percussion note or actual dullness. Breath-sounds are weak, as a rule, but may be bronchial or tubular. Voice conduction is increased, and in incomplete collapse crepitations are often audible. The cardiac impulse may be displaced towards the affected side, but this is less apparent than in active collapse, and it is not infrequently displaced to the opposite side by the primary condition. Lobular collapse gives rise to no symptoms which can be differentiated from those of the condition inducing it.

Complications and Course.—The lung usually re-expands wholly or in part when the condition causing collapse has been removed. Thus a lung that has been maintained continuously collapsed by artificial pneumothorax, with repeated refills for as long as 4 years or more, will re-expand when the gas in the pleural cavity is not replaced. In chronic effusion, or in large or neglected empyemata, re-expansion may be incomplete, and some falling in of the chest wall results. Fibroid changes may occur in lung tissue which has been long collapsed.

Diagnosis.—This is frequently a matter of inference, owing to the nature of the primary disease. Valuable help may be afforded by X-ray examination.

Treatment.—No special treatment apart from that of the condition causing the collapse is required. If a pleural effusion is slow to absorb, the necessity for paracentesis or gas replacement, to avoid pleural thickening, may have to be considered.

HÆMOPTYSIS

It should be recognised that hæmoptysis is a symptom, not a disease. It is here considered separately because the accurate diagnosis of its origin is essential to its treatment, which differs widely in different conditions.

Definition.—The term hæmoptysis is arbitrarily restricted to the expectoration of blood, entering the air passages from structures below the larynx or from the larynx itself. When the blood is derived from the naso-pharynx or mouth it is sometimes described as spurious hæmoptysis.

Ætiology.—1. Pulmonary tuberculosis is the commonest cause, the blood being derived from an aneurysm in a pulmonary cavity, or from ulceration of a small vessel, or congestive processes around the early lesions.

2. Chronic venous congestion, particularly in mitral stenosis. These two conditions account for the majority of cases.

3. Inflammatory and destructive diseases of the lungs, air passages or pleura, such as pneumonia, broncho-pneumonia, especially the influenzal variety, abscess, gangrene and bronchiectasis with ulceration of the walls. A latent bronchiectasis without sputum may cause recurrent hæmoptysis (*forme hémoptoïque sèche*). Pneumokoniosis, streptotrichosis and ulceration of the larynx, trachea or bronchi from tuberculosis, gumma or new-growth

may also be associated with hæmoptysis. Breaking down of a caseous or calcareous bronchial gland is a rare cause, as also is rupture of an empyema through a bronchus.

4. Infarction of the lung from embolic or thrombotic obstruction.

5. New-growths of the lung, bronchi or mediastinal glands.

6. An aortic aneurysm may cause hæmoptysis by "weeping" through an eroded bronchus, or by direct rupture, the latter being of course immediately fatal.

7. Traumatic causes.—Injury may cause hæmoptysis, by fractured ribs wounding the lung, by contusion and by breaking down of healed tuberculous lesions. Hæmoptysis occurs frequently in wounds of the chest, both penetrating and non-penetrating. A foreign body, such as a piece of shrapnel, may lie dormant for years, and then cause recurrent hæmoptysis.

8. Certain abnormal blood conditions, chiefly leukæmia, purpura, hæmophilia, scurvy, minor degrees of vitamin C deficiency and occasionally pernicious anæmia. Hæmoptysis occasionally occurs in the malignant specific fevers, especially small-pox and measles.

9. Parasitic causes, such as pulmonary distomatosis and spirochætosis, are common in Asia but rare in Europe. Hydatid disease of the lung may cause repeated slight hæmorrhages.

10. Vicarious menstruation.—Some cases in women have been regarded as vicarious menstruation, and this view dates back to Hippocrates. It is probable, however, that most cases are to be explained as due to leakage from obscure pulmonary lesions.

11. Hæmoptysis occasionally occurs in apparently healthy persons. In some, with high systemic arterial tension, it is probable that the pulmonary arterial pressure is also raised, and the condition may be regarded as analogous to the epistaxis which occurs more commonly in such patients. Sometimes the hæmoptysis is due to leaking from an old arrested tuberculous lesion.

12. Rupture of an hepatic abscess or hydatid cyst through the diaphragm into a bronchus is an occasional cause.

Spurious hæmoptysis is usually due to staining of the saliva or the pharyngeal secretion with blood, generally derived from the gums, which are spongy and congested, often from early pyorrhœa. The condition is common in anæmic girls, and is, as a rule, observed in the morning. Hæmorrhage from an enlarged pharyngeal vein is often suggested as a cause, but is rarely seen. Hæmorrhage after tooth extraction, and staining of the mucus expectorated after epistaxis, are other causes of spurious hæmoptysis.

Pathology.—From the list of causes it might be inferred that the origin of the blood differs in different cases. It may come from the pulmonary or bronchial vessels in pulmonary tuberculosis and other lung or bronchial conditions, and also in chronic venous congestion or infarction. It may come from the thoracic aorta direct, or from some of its branches, in aneurysm and mediastinal new-growth, and from the hepatic vessels in abscess of the liver. In cases due to disease of the trachea and larynx it comes direct from the vessels supplying them.

Post mortem, the larynx, trachea and bronchi may contain clots, or blood-stained froth and mucus, and their walls may be stained in places. Dark reddish areas of lobular distribution, due to inhaled blood, may be seen

in various parts of the lungs, particularly at the bases. Sometimes this may induce bronchitic changes, described as hæmoptoic bronchitis. Careful search in cases of profuse hæmoptysis will usually reveal the source of the hæmorrhage, and in pulmonary tuberculosis this is generally a ruptured aneurysmal dilatation in a cavity or an ulcerated vessel. The aneurysm may be small and escape notice unless many cuts are made into the lung.

Symptoms.—In hæmoptysis, the patient often experiences a tickling in the throat, followed by a gush into the mouth with a salt taste, and on expectoration notices blood. The alarm and anxiety this occasions lead to restlessness and rapid action of the heart. If the bleeding is profuse, cough is frequent, and large clots, together with liquid alkaline blood, may be expectorated to the extent of 20 or 30 ounces in a few hours. The bleeding may cease temporarily, to recur at intervals for several days, until the patient becomes blanched, weak and syncopal, with rapid, weak pulse. In any profuse hæmoptysis, death may occur in a few minutes, either from asphyxia or syncope. In the former case, the blood, at first bright and arterial, is soon dark and frothed, while the patient becomes cyanosed and livid. In slighter degrees of hæmoptysis there may be only streaks, small clots or liquid blood mixed with ordinary sputum. After the actual bleeding has ceased, the sputum may be blood-stained for some days, owing to the expectoration of blood inhaled into other parts of the lungs. This can be recognised by its colour, which varies from dark red to brown, owing to the changes undergone by the blood pigment.

Diagnosis.—This involves two problems—first the differentiation from hæmatemesis and spurious hæmoptysis, and secondly the recognition of the cause of the hæmorrhage. If the patient is seen at the time of the bleeding the first of these is easy. The nature of the blood, and its association with cough and possibly with pulmonary or cardiac signs, are conclusive. When the diagnosis has to be made upon the history given by the patient or by friends it may be difficult, especially in the absence of physical signs.

In hæmatemesis there is frequently gastric pain and faintness before the vomiting, the blood is acid in reaction, dark in colour, even brown from acid hæmatin, and is sometimes mixed with food. The fact that in hæmoptysis blood may be swallowed and subsequently vomited increases the difficulty. Patients often give very dubious answers to questions as to whether the blood was coughed or vomited up. They should then be questioned as to whether sputum was brought up on the following day, and, if so, whether it was blood-stained. In cases of doubt the investigation of the pulmonary and abdominal physical signs, when the patient's condition permits, may decide the diagnosis.

The utmost caution should be exercised to exclude tuberculosis before making a diagnosis of "spurious hæmoptysis." Only when there are no pulmonary symptoms, signs or X-ray indications, and when some obvious cause, such as anæmia or pyorrhœa, is found, is it safe to do so.

While distinguishing between the various causes of hæmoptysis it is well to regard and to treat it as due to pulmonary tuberculosis until some other cause is conclusively established. The sputum should be examined for tubercle bacilli on several occasions, the temperature recorded and the physical signs including X-ray appearances most carefully watched.

The presence of a valvular lesion, especially mitral stenosis with signs of

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pulmonary engorgement, may render the cause of hæmoptysis clear. When tuberculosis and cardiac disease can be excluded, a careful study of the history, the symptoms and signs, may throw light on the diagnosis or suggest some investigation which will serve to establish it, e.g. examination of the sputum for parasites and hydatid hooklets, the cytological examination of the blood and an X-ray examination.

In other cases, as in bronchiectasis, abscess or gangrene, the history, the physical signs and the nature of the sputum are often characteristic.

In the latent or silent form of bronchiectasis (*forme sèche*), the condition may be revealed only by lipiodol injection.

Bronchoscopy may be of great value in revealing the presence of adenoma or carcinoma.

Prognosis.—Apart from hæmoptysis, which is rapidly fatal, due to aneurysm or pulmonary tuberculosis, the immediate prognosis in cases of pulmonary hæmorrhage is not unfavourable, even when it continues for days. The ultimate prognosis depends upon the cause.

Treatment.—This is so entirely dependent upon the cause and origin of the bleeding that reference should be made to the corresponding diseases.

EMPHYSEMA OF THE LUNGS

Emphysema of the lungs, or alveolar-ectasis, is a condition of distension of the alveoli; it is usually progressive and is associated with definite changes in the inter-alveolar walls. The following varieties are generally recognised—(1) Large-lunged or hypertrophic; (2) small-lunged or atrophic; (3) compensatory; (4) acute vesicular; and (5) acute interstitial emphysema. The last named condition has no relation to true emphysema except in name, but will be described in this group for convenience.

1. LARGE-LUNGED OR HYPERTROPHIC EMPHYSEMA (SUBSTANTIVE OR IDIOPATHIC EMPHYSEMA)

This is a chronic affection and is usually bilateral.

Ætiology.—*Predisposing causes.*—It may occur at any age, even in childhood, but is most frequently seen in middle and late adult life. It is commoner in men than in women, probably because they are more exposed to the conditions inducing it. Although not strictly hereditary, it often shows a familial incidence. Certain occupations are credited with being concerned in its production, notably those involving violent or prolonged muscular effort with closed or partially closed glottis, such as blowing wind instruments and lifting heavy weights. Dusty occupations also favour its onset by leading to bronchitis and cough.

The common *exciting cause* seems to be the strain of prolonged and repeated cough, induced by chronic bronchitis, bronchiectasis, asthma, whooping-cough, cigarette smoke inhaling, and other causes of irritation of the upper air passages.

Pathology.—The pathogenesis of emphysema has been much debated and various explanations have been offered. (1) **Primary degeneration theory.** Villemin suggested that the essential lesion was a fatty degenera-

tion of the alveolar walls, while Cohnheim believed that there was a congenital defect of the elastic tissue of the lung. (2) The inspiratory theory, first suggested by Laennec and developed by Gairdner, postulates the force of inspiration as the distending agent. (3) The expiratory theory, first enunciated by Mendelssohn, was independently brought forward and established by Jenner. The distension of the alveoli is regarded as due to the effect of forced expiration and cough. Jenner pointed out the special and early involvement of the apices, the anterior and lower margins of the lungs; in other words, the parts least supported by the thoracic cage. (4) Freund regarded the changes in the lungs as secondary to calcification of the costal cartilages, the chest becoming fixed in the inspiratory position and the lung permanently expanded in consequence. The expiratory explanation is now generally accepted, and emphysema is regarded as the result of increased intra-alveolar tension, due to violent expiratory efforts, acting on walls weakened by congenital defects, by inflammatory processes or by toxic agents, such as alcohol (Nothnagel).

The characteristic conformation of the chest is usually apparent (see Symptoms), the costal cartilages are often calcified, and on opening the thorax post mortem, the lungs bulge instead of retracting, so that the pericardium may be almost completely obscured. They are pale in colour, even in town-dwellers, a condition called albinism of the lung by Virchow. They are soft and pit on pressure, and, as described by Laennec, give the sensation of a down pillow. The surface of the lung under the pleura shows a finely vesicular appearance, due to the distension of the alveoli, the vesicles often being nearly as large as pins' heads. Not infrequently large bullæ or blister-like protuberances, varying in size from a pea to a Spanish olive, occasionally much larger, may be seen projecting from the surface, particularly at the apices and margins. These bullæ when incised show fine fibrous bands crossing them, the remains of inter-alveolar walls and of atrophied blood vessels. It was formerly customary to refer to such cases as bullous or marginal emphysema and to describe those in which the dilatation is less obvious but more widely diffused as general emphysema; but the conditions are so commonly associated together in varying degrees that little is gained by so doing. On section the lungs are pale and dry, except at the bases, where there is frequently some cedema in advanced cases. The bronchi may show some general dilatation, although less commonly than might be expected from the close similarity of the causal factors of emphysema and bronchiectasis. When bronchitis coexists, muco-pus can be squeezed from the cross-sections of these tubes. As pointed out by Fowler, pleural adhesion is relatively uncommon. The infundibula and alveoli are dilated, and the inter-alveolar walls are thin and atrophic, even disappearing wholly or in part. The distension and coalescence of adjacent alveoli result in the formation of bullæ. The calibre of the pulmonary capillaries is diminished by stretching of the alveolar walls, and where atrophy of the inter-alveolar septa occurs the capillaries are destroyed. These two processes result in a considerable diminution in the total aerating surface, and cause the dyspnoea and cyanosis characteristic of the disease. Moreover, the normal anastomoses between the terminal bronchial and pulmonary capillaries increase considerably, and some of the blood in the latter may therefore fail to reach the alveoli and so escape aeration. Atrophic changes in the elastic tissue

have been described. In order to maintain the circulation through the diminished capillary area, the right ventricle hypertrophies and the resultant raised blood pressure sometimes induces atheroma of the pulmonary artery. Emphysema being a progressive lesion, and the defective aeration of the blood perhaps interfering with the nutrition of the heart muscle, cardiac failure eventually ensues, causing tricuspid regurgitation, engorgement of the right auricle, and the visceral effects of venous engorgement, such as "nutmeg" liver. Cabot states that true emphysema is often not found post mortem in cases so diagnosed during life, and prefers to designate the clinical entity here described as "the Barrel Chest."

Symptoms.—Dyspnoea of varying degree is the most characteristic symptom. In uncomplicated cases of moderate extent it is only present on exertion, unless bronchitis coexists. In advanced emphysema, dyspnoea is marked and becomes extreme in the bronchitic or "asthmatic" attacks and in foggy weather. Cyanosis is common, and is to some extent a measure of the degree of emphysema. Varying degrees of polycythæmia may be observed. The patient may walk about with a more extreme degree of cyanosis than in any other condition except congenital heart disease. Clubbing of the fingers of moderate degree is common. Cough is usually due to the associated bronchitis, and is worse in the winter and in foggy weather. It is frequent, noisy and often hacking and paroxysmal. Expectoration is also the result of the bronchial catarrh, and varies from a few grey mucoid pellets to copious muco-pus.

The chest is enlarged, particularly in the antero-posterior diameter, the upper thoracic spine is rounded and kyphotic, the sternum protrudes forward, and the angle of Louis is prominent, the general effect being the so-called barrel-shaped chest. The ribs run forward more horizontally and the intercostal spaces are wider than normal, the chest being as a whole in the inspiratory position. The respiratory movements are much restricted, the patient elevating the rigid thorax with little expansion on taking a deep breath, so that the inspiratory increase at the level of the nipples may be only half to 1 inch, instead of the normal $2\frac{1}{2}$ to 3 for an adult. There is often filling and even bulging of the supra-clavicular hollow, while the neck appears short, the sternomastoids stand out, and the jugular veins are full. A zone of dilated venules, the "emphysematous girdle," is often present along the line of the costal attachment of the diaphragm, but is not pathognomonic. The cardiac impulse is not visible as a rule, and may only be felt with difficulty, but epigastric pulsation is usually apparent. Vocal fremitus is diminished, and the percussion note is hyper-resonant. The superficial cardiac dullness is greatly diminished or even absent, and the lower limit of pulmonary resonance may extend to the costal margin, back and front, the hepatic dullness being encroached on or obliterated.

It is said that in bullous emphysema the breath-sounds are harsh over the outer part of the upper lobes in front, and weak at the bases. In general emphysema the breath-sounds are weak everywhere, inspiration is short, and expiration is greatly prolonged. A loud rumbling, from contraction of the thoracic muscles, may entirely obscure the breath-sounds. A few fine bubbling râles may be heard at the bases or at the sternal margins. If bronchitis is present, scattered rhonchi may be audible. Vocal resonance is generally slightly diminished. The heart-sounds are weak and distant,

and in late stages a tricuspid systolic murmur may develop. The "vital capacity" of the lungs, measured by a spirometer, is often reduced to one-half or less. Examination by the X-rays shows increased extent, and undue transradiancy of the lung tissue. They show the diaphragm lower in position and flattened, and the costophrenic angle widened. The liver is sometimes palpable, possibly from downward displacement by the bulky lung, but more often from enlargement due to passive hyperæmia. The spleen may also be depressed and enlarged.

Complications.—Bronchitis is the commonest, and often constitutes a vicious circle. Asthmatic attacks, so-called "bronchial asthma," are common in later stages; on the other hand, spasmodic asthma may be the cause of the emphysema. Pneumothorax and interstitial emphysema may occur from rupture of the bullæ, although these accidents are surprisingly rare. Pulmonary tuberculosis is an occasional complication of emphysema, which, contrary to popular opinion, is not antagonistic to it, although it may mask and obscure the early stages. Right-sided cardiac failure, with its train of consecutive changes, is a late and often terminal complication.

Course.—Emphysema is progressive, unless the cause is removed or the effects of the disease are mitigated by residence in a warm, dry climate, especially in the winter. Conversely, residence in unsuitable districts, persistence in detrimental employment, and repeated attacks of bronchitis accelerate its course.

Diagnosis.—This is never difficult in advanced cases. The slighter degrees may be more difficult, and the diagnosis is then largely a matter of inference from the association of chronic cough and dyspnoea, with physical signs of hyper-resonance and prolonged expiration.

Confusion may occasionally arise in regard to pneumothorax and pulmonary tuberculosis. Careful record of the symptoms and signs and the investigation of the sputum generally suffice to distinguish these conditions. In doubtful cases the X-rays may assist.

Prognosis.—This depends upon the degree of emphysema and the circumstances of the patient. If progressive, it exerts an increasingly crippling effect, and it certainly shortens life under urban conditions. A "vital capacity" of less than 50 per cent. of the normal is of serious import. The advent of severe bronchitis or of cardiac complications may affect the prognosis gravely.

Treatment.—Emphysema may be arrested but cannot be cured. Attention must be directed to prevention of the causes of chronic cough and increased intra-alveolar tension. In any person with hereditary tendency to emphysema or to winter cough, the questions of occupation and place of residence should be carefully considered. When the disease is established, the patient, if in a position to afford it, should spend the winter in a warm, more equable climate, either abroad or at the south-west coast of England.

Various attempts have been made to increase the respiratory ventilation of the lungs, e.g. by compression of the chest during expiration, by expiring into rarefied air, by breathing compressed air or by expiratory breathing exercises. The patient enters a special iron chamber fitted with a window, and the air pressure is raised during the course of half an hour to $1\frac{1}{2}$ atmospheres. He remains at this pressure for an hour, and is then decompressed to normal during the next half-hour. These baths may be given every other

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day and gradually increased in duration and frequency. This treatment is often helpful in cases of emphysema associated with bronchial spasm or with bronchitis. Cases with marked arterial disease or with much rigidity of the chest-wall are unsuitable for this form of treatment.

The diet should be simple and easily digestible, especially in the later stages. If there is spasmodic dyspnoea or asthma, no late meal should be permitted. Cod-liver oil or halibut liver oil in the winter seems to help some patients. Clothing should be warm, but the excess of under-garments, often worn in fear of chill, is harmful.

In other respects treatment is largely symptomatic. In acute bronchitic attacks the measures to be adopted are in no way different from those in bronchitis uncomplicated by emphysema. In the more chronic bronchitis so commonly present in the winter, iodides with alkalis and balsamic expectorants seem beneficial. Terebene (min. 10) in emulsion or in capsule has been recommended. Counter-irritation to the chest by liniments, such as the lin. terebinthinæ aceticum, is often comforting to the patient, when cough is troublesome. When asthma or paroxysmal dyspnoea occurs, antispasmodic drugs and measures similar to those used in spasmodic asthma may be employed. When cardiac failure supervenes, the appropriate treatment must be vigorously applied. If there is marked cyanosis and venous engorgement, oxygen administration, venesection, leeching, purgative and diuretic drugs may be employed, and digitalis and other cardiac tonics administered. American authorities have suggested the use of an abdominal belt to increase the intra-abdominal pressure and raise the diaphragm.

2. SMALL-LUNGED EMPHYSEMA (ATROPHIC OR SENILE EMPHYSEMA)

Ætiology.—This condition occurs in old age and forms part of the general atrophy of the tissues.

Pathology.—The alveolar walls become thinned and disappear, so that adjacent alveoli coalesce. The condition is primarily atrophic, and therefore differs from true emphysema, although the result is to produce a diminished area for aeration. Post mortem the lungs are small and do not bulge or obscure the pericardium. They are often deeply pigmented, and are more spongy than normal, but although bullæ occur they are small. On section the lung tissue is bloodless and friable. The bronchi may be slightly dilated and show catarrhal changes.

Symptoms.—These are slight and are masked by the enfeeblement due to the general atrophy and debility. There is shortness of breath only on exertion, or on exacerbation of the chronic bronchitis which is frequently present. The chest is small, flat and thinly covered, the movements are poor and there is elevation of the chest as a whole, with poor expansion. Kountz and Alexander maintain that there is very little diminution in vital capacity, that the movements of the diaphragm are increased and that the intervertebral discs are abnormal. There is little cyanosis, and no clubbing. The vocal fremitus is unaltered or slightly diminished. The percussion note is hyper-resonant, but there is no encroachment on the cardiac and hepatic areas of dullness. Breath-sounds are weak, and there is but little prolongation of expiration. Rhonchi and râles may be heard, especially if bronchitis is present, or if the heart is failing.

Diagnosis.—The condition is generally so obvious that no difficulty arises.

Treatment.—This is chiefly a matter of careful regimen and diet, with treatment of coexisting bronchitis or cardiac failure.

3. COMPENSATORY EMPHYSEMA (LOCALISED OR SECONDARY EMPHYSEMA)

Ætiology.—Localised emphysema is a sequel to some process inducing collapse, contraction or destruction of areas of lung tissue. It may be lobular in distribution in bronchitis, broncho-pneumonia, tuberculosis and diphtheria. It may affect one or more lobes, or the whole of one lung, especially in cases of fibrosis following tuberculosis, pneumonia, chronic pleural effusion and empyema.

Pathology.—It is generally conceded that the inspiratory theory of Laennec and Gairdner satisfactorily explains the genesis of this condition. When shrinkage of an area of lung occurs, the chest wall may fall in, if there is pleural adhesion, but otherwise inspiration tends to expand the normal parts of the lungs. None the less, it must be admitted that the expiratory strain of cough may assist in its production.

Although it may be compensatory and physiological at its inception, it is doubtful whether a true hypertrophy takes place after adolescence. In any case it soon leads to atrophy of the alveolar walls, as in true emphysema, and thus becomes pathological and harmful. Post mortem the condition may be found in an upper lobe around contracted scarred lung tissue, or in a lower lobe when the upper lobe is contracted or disorganised. In cases where one lung is fibroid and contracted, compensatory emphysema may be found throughout the sound lung. The resulting adaptations caused by enlargement of one part and shrinking of another may produce some striking displacements, the lower lobe extending upwards nearly to the clavicle, or the anterior margin of the sound lung crossing the mid line. The general appearances are closely similar to those of ordinary emphysema, except that bullæ do not occur, at any rate until the process is advanced and definitely pathological.

Symptoms.—This condition does not produce symptoms that can be differentiated from those of the primary disease. When it affects a lobe or the whole of one lung, there is hyper-resonance over the area involved, which often contrasts strikingly with the dullness due to the primary lesion. The hyper-resonance may extend across the sternum and even for an inch or more beyond it. The heart is displaced towards the side where fibrosis is in progress. Vocal fremitus and vocal resonance are little altered, but may be increased at first and subsequently diminished. In the early stages, when there is alveolar dilatation without degenerative mural changes, the breath-sounds are exaggerated, harsh or puerile, but when such processes develop, they become weak and there are indications of dyspnoea and cyanosis on exertion.

Diagnosis.—This is easy, owing to the difference between the diseased and “compensatory” areas, and to the indications of contraction and displacement.

Treatment.—No special treatment apart from that of the primary condition is required.

4. ACUTE VESICULAR EMPHYSEMA

Although custom has included this condition with emphysema, it is in reality only a temporary acute distension of the alveoli resulting from any condition causing widespread obstruction of the smaller bronchi. It is sometimes observed after death in cases of acute bronchitis, whooping-cough or asphyxia and in anaphylactic shock, and its existence may be inferred in severe asthma. Post mortem the lungs are bulky and the alveoli distended.

The symptoms are dependent upon the primary condition, although dyspnoea is invariably present. The chest is found to be fully expanded, the vocal fremitus is diminished, the percussion note is hyper-resonant, and the breath-sounds vary with the condition inducing it.

5. ACUTE INTERSTITIAL EMPHYSEMA

In acute interstitial emphysema air is present in the stroma of the lungs, and in the subpleural connective tissues. It may follow external trauma, such as fractured ribs, or wounds penetrating the lungs. The alveoli may rupture with violent expiratory efforts, as occur in whooping-cough or influenzal broncho-pneumonia. It may occur in diphtheria. The air sometimes tracks along the pulmonary roots to the mediastinum, and appearing in the neck or on the chest-wall gives rise to surgical emphysema.

Post mortem, subpleural bullæ may be seen containing air, and on section of the lung minute air bubbles may be found in the inter-alveolar connective tissue. A diagnosis cannot be made unless the physical signs of surgical emphysema are present. The air is usually completely absorbed, and a perfect recovery takes place. No special treatment is required beyond keeping the patient at rest, and giving sedative drugs to allay cough.

ABSCESS OF THE LUNG

Definition.—Abscess of the lung includes any circumscribed collection of pus formed in the lung tissue, but softened tuberculous areas and bronchiectatic accumulations are usually excluded.

Ætiology.—*Predisposing causes.*—These include any diseases producing general cachexia or malnutrition, notably diabetes and chronic alcoholism, also any conditions leading to diminished resistance locally in the lung, such as injury, disease or exposure.

Exciting causes.—These are pyogenic organisms, which reach the lung by inhalation, by extension from adjacent suppurative processes, or by the blood stream, either directly or in septic emboli. The common organisms found are streptococci, staphylococci, the pneumococcus, Friedländer's pneumo-bacillus, *Bacillus welchii* and the *Bacillus coli*—sometimes acting in conjunction with putrefactive bacteria. Spirochætes, *Bacillus fusiformis*, treponemata and anaerobic organisms are often present, especially after rupture has occurred. Pulmonary abscess may form under the following conditions:

(1) After inhalation of foreign material into a bronchus. This may be a foreign body, or may occur in association with septic conditions in the nose,

nasopharynx and larynx, or during and after operations in these regions. These are referred to as inhalation abscesses, though some post-operative cases are regarded as due to embolism and not to inhalation. (2) As a result of lobar or lobular pneumonia, especially after the deglutition and aspiration varieties of the latter. Such abscesses are sometimes called metapneumonic. (3) Embolic causes—in pyæmia, or following on septic pulmonary emboli due to right-sided septic endocarditis, or derived from distant septic processes, such as otitis media, and infective thrombo-phlebitis. Amœbic abscess occurs occasionally after dysentery, and pulmonary abscess may be found as a rare complication of enteric fever. (4) From infection of the lung tissue due to spread from adjacent disease. This may occur in bronchiectasis, in ulcerating new growths of the lung, bronchi, œsophagus or mediastinal glands, in caries of the vertebræ or ribs, and in suppurating mediastinal glands. Rupture of an empyema, of a subphrenic abscess, of a liver abscess, or of infected hydatid cysts of the lung or liver may also lead to pulmonary suppuration. Ten per cent. of cases of abscess are due to new growths. (5) As a sequel of perforating chest wounds, or of fractured ribs piercing the lung.

Pathology.—Abscess of the lung is generally single and basic when consequent on pneumonia, whereas embolic abscesses are often small and multiple and may be found in any part of the lung. Abscesses due to extension from adjacent disease are generally solitary, and are often large and irregular. The walls of acute abscesses are generally formed of congested and œdematous lung tissue, or of a zone of unresolved pneumonia. Since acute abscesses commonly rupture quickly into a bronchus, a fibrous capsule is unusual, but in chronic abscess there is often considerable fibroid change in the neighbouring lung tissue. The pleura may become involved over superficial abscesses, leading to empyema, or to pyo-pneumothorax if rupture follows.

Symptoms.—Abscess may develop insidiously, with comparatively slight symptoms. More commonly they are an intensification of those due to the primary or antecedent condition. The patient often appears seriously ill, the fever becomes of septic type, remittent or intermittent in character, and of a high range. Rigors and sweating are common. The pulmonary symptoms at first may be only slight cough with scanty muco-purulent expectoration. Dyspnoea may be present and pain of acute character develops if the pleura is involved. Hæmoptysis occurs in 70 per cent. of cases of abscess. A considerable leucocytosis, up to 20,000 or 30,000 may be found, and occasionally the breath may be offensive, even before rupture into a bronchus occurs, followed by the sudden expectoration of a large quantity of pus. The pus is sometimes unpleasant or offensive-smelling, but has not the extreme fetor of gangrene. Microscopical investigation will demonstrate the presence of pulmonary debris, especially elastic tissue, together with pus cells and micro-organisms. After the expectoration of the pus, the temperature usually falls and the general condition of the patient is much improved, though cough and expectoration persist. In chronic cases after rupture the temperature may become irregular and periodic, a few days of normal temperature being followed by a period of fever and later by increased expectoration. The physical signs in a deep-seated or small abscess are often inconspicuous, and comprise slight dullness over a small area, weak breath-sounds and possibly a few râles in the surrounding infiltrated or œdematous lung tissue. With a large or a

superficial abscess, the signs before rupture may be those of consolidated or collapsed lung. After evacuation occurs, the characteristic signs of excavation usually develop at once. In multiple embolic abscesses the signs are usually those of disseminated broncho-pneumonia.

Complications and Sequelæ.—The commonest complication is dry pleurisy. This may progress to empyema, or to pyo-pneumothorax, if rupture into the pleura occurs. In some cases mediastinitis or pericarditis may develop. Gangrene is described, but is a rare sequel. Metastatic abscesses may be produced in other parts of the body, especially in the brain, and meningitis is a rare and serious complication. The most important sequelæ are fibrosis of the lung, with bronchiectasis, pleural adhesion, and rarely indurative mediastinitis.

Diagnosis.—This is difficult before rupture, but abscess may be suspected from the gravity of the symptoms in relation to the history and signs, especially if leucocytosis and fetor of the breath are present. X-ray examination may be helpful, by demonstrating a localised shadow before rupture, and excavation afterwards, and also by establishing the situation of the abscess. A fluid level can often be seen in films taken in the erect position. The sudden expectoration of pus, followed by retrogression of symptoms and signs of excavation is very suggestive of abscess. After rupture has occurred the differential diagnosis has to be considered from:

1. *Interlobar empyema.*—This may be very difficult or even impossible. In this condition the signs are generally most marked in the region of an interlobar septum, there may be some cardiac displacement, and the sputum, though purulent, does not contain elastic tissue.

2. *Bronchiectasis.*—The history, the characteristic cough and sputum, and the variation of the physical signs with the state of the cavity usually suffice to distinguish this condition. An X-ray examination after lipiodol or neo-hydriol will distinguish in doubtful cases, since they do not as a rule enter the abscess cavity and the appearances in bronchiectasis are characteristic.

3. *Gangrene of the lung.*—The distinction is not always easy in acute abscess, since the two processes are closely related. The extreme gravity of the patient's general condition and the horrible fetor of the breath and sputum are the most characteristic features of gangrene.

4. *Tuberculous excavation.*—The history, the distribution of the signs, and the characters of the sputum, including the presence of tubercle bacilli, are the distinguishing indications.

5. *Purulent bronchitis.*—The history, the widespread physical signs, and the absence of elastic tissue from the sputum usually serve to establish the diagnosis, and lipiodol or neo-hydriol investigations may be helpful.

In multiple or pyæmic abscesses, it is often impossible to recognise the condition, though it may be suspected from the severity of the symptoms and signs. In any doubtful case an X-ray examination or tomography should be carried out, if the condition of the patient permits. The possibility of malignant growth as a cause of abscess should be borne in mind and, when necessary, bronchoscopy as well as lipiodol investigation carried out. Exploratory puncture as a means of diagnosis is dangerous and should be avoided.

Prognosis.—The prognosis, though grave in many cases, is better than

might be anticipated. Many of those in which rupture into a bronchus occurs recover. Death is inevitable in the pyæmic cases.

Treatment.—(1) *Medical.*—In acute abscesses medical measures should be employed, since in a considerable proportion of cases, recovery may occur after rupture, especially when the abscess is in the upper lobe. Before rupture the treatment should be similar to that for acute pneumonia. After rupture, evacuation should be promoted by “tipping,” or postural drainage. The Nelson bed is of great value in this connection when the relation of the abscess to the nearest patent bronchus has been determined by X-ray examination. Expectorant drugs and antiseptics, such as creosote, should be employed. Antiseptic inhalations may also be used on a Burney-Yeo mask, as for bronchiectasis, or creosote vapour baths may be given. In like manner intratracheal injections of menthol, guaiacol and olive oil have been employed with benefit. In cases with spirochætes, treponemata and anaerobic organisms in the sputum, intravenous injections of salvarsan or neo-arsphenamine may be administered with benefit.

(2) *Surgical.*—If spontaneous rupture does not occur after the abscess has become localised and encapsulated, operation is indicated in order to prevent the walls becoming thick. Repeated bronchoscopic aspiration is sometimes employed after rupture, and at times gives satisfactory results. If, after rupture, there is not satisfactory progress towards cure within 6 weeks, clinically and radiologically, surgical treatment should be considered. Thoracotomy and open drainage is the operation generally employed. This is now usually carried out in two stages: (a) a preliminary exposure of the pleura by rib resection and packing with gauze, to ensure adhesion of the pleura; (b) some days later the abscess is opened along the course of an exploring needle. Artificial pneumothorax has been recommended, especially in deep or centrally placed abscesses. There is a risk of rupture into the pleura, more particularly when re-expansion of the lung is permitted. For this reason, this form of treatment is rarely advisable. Phrenic evulsion may aid in the evacuation of a chronic abscess, either alone or after thoracotomy. Pneumolysis, lobectomy, and thoracoplasty are also used in the treatment of chronic abscess. Lobectomy seems likely to be the most satisfactory in chronic cases in which it is practicable.

GANGRENE OF THE LUNG

In this condition localised or diffuse areas of lung tissue undergo putrefactive necrosis.

Ætiology.—*Predisposing causes.*—These include old age, over-indulgence in alcohol, general debility, diabetes and insanity. In certain rare cases, especially after broncho-pneumonia complicating measles, gangrene of the lung is met with in children.

Exciting causes and associated conditions.—These are, in the main, identical with those of pulmonary abscess (see p. 1183). In addition, the pressure of aneurysm or of new-growth on branches of the pulmonary artery may lead to gangrene. The causal organisms are also very similar to those found in abscess of the lungs, and include staphylococci, streptococci, sarcinæ, the *Micrococcus tetragenus*, the *Bacillus coli communis*, the *B. pyocyaneus*, the

B. fusiformis with its associated spirochæte, and various anaerobes. In some instances acid-fast bacilli, classed as streptothrices, occur. Some of these organisms yield putrefactive products, with the liberation of phenol, indole and skatole compounds in the lung.

Pathology.—It is not quite clear what are the factors determining whether abscess or gangrene occurs in an infected area of lung. Doubtless the general resistance of the body, the degree of local vascular disturbance, and the virulence of the infecting organisms all play their part. Laennec first described the two varieties of gangrene, the circumscribed and the spreading or diffuse. Around the former there are indications of a line of demarcation, formed by congested lung tissue, which may present the appearance of red hepatisation. The surrounding lung tissue is invariably somewhat œdematous. The gangrenous area is soft and pulpy, and its colour varies from reddish-brown to greenish-black. As the necrosis advances, putrefactive liquefaction occurs, with the formation of a horribly reeking fluid, containing shreds and masses of necrotic lung tissue. When this is discharged, excavation results, and isolated vessels may be seen running across the resulting cavity, the walls of which are rough and covered with fetid pus. The diffuse variety of gangrene is less common; there is no attempt at a zone of demarcation, and the whole of a lobe or of one lung may be affected. In both forms, the overlying pleura is intensely inflamed, and empyema or pyo-pneumothorax may be produced.

Symptoms.—These are similar to those occurring in abscess of the lung, but are more acute. The patient is desperately ill, rigors are more common and sweating is more profuse. The breath has a peculiar fetor, which, on account of the presence of the skatole group of putrefactive products in the gangrenous lung, has an almost faecal odour. The sputum is intensely offensive, and on standing separates into three layers, similar to those of the expectoration in cases of bronchiectasis. Elastic tissue is usually present, but it may undergo rapid disintegration. Hæmoptysis is not infrequent and may prove fatal. In rare cases gangrene is not accompanied by fetid expectoration, especially when developing in the insane, in young children, and in diabetics, or after pulmonary embolism. The physical signs closely resemble those present in cases of pulmonary abscess, and are those of consolidation before liquefaction occurs, and of excavation afterwards. The signs of the antecedent condition such as bronchiectasis, aneurysm, or malignant disease may also be present.

Complications and Sequelæ.—These are similar to those met with in pulmonary abscess, but owing to the rapid course and greater fatality of gangrene, they are not so common. Cerebral abscess may occur.

Course.—The course is usually rapid, unless the diseased area is small and circumscribed. In rare cases of localised gangrene of small extent, resolution and subsequent fibrosis occur.

Diagnosis.—The differential diagnosis is as for pulmonary abscess, the distinguishing features being the extremely critical condition of the patient and the revolting fetor of the breath and expectoration. X-ray examination may give great assistance if the patient's condition permits it to be made.

Prognosis.—This is always extremely grave, though a few cases of localised gangrene recover spontaneously. The prognosis is improved by

early operation in suitable cases. The outlook is said to be worse if the condition is apical, and diffuse gangrene is invariably fatal.

Treatment.—Operation is indicated when the general condition of the patient permits, if the gangrenous area can be localised by physical signs or X-ray examination. Exploratory puncture should not be carried out. The other operative procedures are similar to those for abscess of the lung. Operation is contra-indicated in cases of diffuse gangrene. The medical treatment is in all respects similar to that for pulmonary abscess. Injections of neo-arsphenamine, in doses of 0.3 g., have given good results, especially in cases due to fuso-spirochaetosis.

PULMONARY FIBROSIS

Synonyms.—Fibroid Disease of the Lung; Chronic Interstitial Pneumonia; Cirrhosis of the Lung.

Definition.—Pulmonary fibrosis is a late sequel of many acute and chronic inflammatory or irritative processes affecting the bronchi, lungs and pleurae. It is therefore rather of pathological than of clinical interest, and in no sense constitutes a separate disease, although the end-results are remarkably similar in different forms. It is described here partly in deference to tradition, and partly to point out the methods of diagnosis between the various causes producing such strikingly similar effects.

Ætiology.—(1) The commonest cause is pulmonary tuberculosis, particularly the fibroid and fibro-caseous varieties. (2) The group of pneumoconioses contributes a considerable number of cases, and possibly some varieties of gas poisoning may induce fibroid changes. (3) Bronchopneumonic processes, particularly the forms associated with measles and whooping-cough, may be followed by widespread fibrosis, especially in children. (4) Although fibroid induration is commonly described as a sequel of lobar pneumonia, this disease is one of the rarer causes. (5) Localised fibrosis may occur around any circumscribed pulmonary or bronchial lesion, such as that produced by syphilis, leprosy, glanders or streptotrichosis. Similarly it occurs about infarcts, pulmonary abscesses and parasitic cysts. (6) Chronic venous congestion, if prolonged, leads to fibroid change, which is referred to as "brown induration." This is usually of moderate degree and does not affect the clinical manifestations. (7) Chronic pleural affections, particularly those leading to adhesions or causing pulmonary collapse, may induce fibroid changes within the lung, and these forms are described as "pleurogenous cirrhosis." (8) Any condition causing obstruction of a bronchus and leading either to collapse or to bronchiectasis may, if long continued, cause fibrosis of the corresponding lung area. Among such may be mentioned inhaled foreign body, new-growth, cicatricial contraction and thoracic aneurysm.

Pathology.—The fibroid overgrowth may be: (1) Massive or lobar; (2) localised or insular; (3) peribronchial; and (4) reticular.

Any part of the connective tissue framework of the lungs and bronchi may contribute to the fibrosis. In the massive form, which generally affects the whole or the major part of a lobe or even of one lung, the appearances in cases due to tuberculosis differ from those due to other causes. In the

tuberculous variety the primary distribution is usually apical, and evidence of other tuberculous processes may be apparent in the form of large or small dried-up cavities, inspissated caseous material or calcareous masses enclosed in fibrous strands. In non-tuberculous processes, the early localisation is commonly basic, and although the primary cause may be obvious in the form of bronchial obstruction or some pleural condition, this is not always the case. On the other hand, non-tuberculous processes may involve the upper lobe primarily and fibroid tuberculosis may fall with special stress upon the lower lobe. In both forms of fibrosis, bronchiectasis may result, although this is more common in the non-tuberculous cases. Apart from the special tuberculous lesions, the end results are very similar in both forms. The affected area of the lung is shrunken and often devoid of air except for that in the bronchi and in the cavities. It is dark in colour, very firm and hard. On section it presents a mottled appearance owing to the strands of blue-grey fibrous tissue traversing it, contrasting with the pigmented, condensed, airless lung tissue. The fibroid area may be honeycombed by cavities or may present one large excavation, due either to tuberculous cavitation or to bronchiectatic dilatation. There is nearly always thickening and adhesion of the pleura. The contraction of the abnormal fibrous tissue leads to marked displacement of the heart and mediastinum.

The localised form is commonly due to healed tuberculous processes at an apex. There may be simple puckering with or without pleural thickening and adhesion, or a dense mass enclosing dried-up caseous matter or calcareous spicules. In bronchitic or broncho-pneumonic processes a patchy fibrosis may occur, described as insular fibrosis by Fowler.

Reticular fibrosis is a rare condition in which the fibrous tissue in the interlobular septa seems to become increased as well as that around the bronchi. It is at present only of pathological interest.

Symptoms.—The symptoms of pulmonary fibrosis are, in the main, expectoration and dyspnoea together with those of the primary affection. In the non-tuberculous cases, bronchiectasis is so frequently associated that the symptoms and signs found are practically those of this condition. Even in tuberculous cases, some degree of bronchial dilatation is the rule, although the sputum is rarely offensive. The cough is generally periodic and associated with change of posture. The expectoration is abundant, and if bronchiectasis is present, it has the usual characteristic features. The dyspnoea is proportional to the extent of lung involved. It may be extreme in the later stages, when the heart becomes embarrassed and begins to fail. Fever is usually absent, except when complications occur.

The patients are generally spare, although nutrition may sometimes be well maintained until late. They may show signs of deficient aeration in duskiness, cyanosis and congested cheeks. Polycythæmia of some degree is the rule. Clubbing of the fingers is almost constant. Evidence of contraction is generally forthcoming in the flattening and retraction of the affected side, with the dropped shoulder and compensatory spinal curvature. Movement is greatly restricted, contrasting with the increased expansion of the other side. The cardiac impulse is sometimes much displaced, especially in left-sided cases, when it may be in the left posterior axillary line or even under the angle of the scapula. In right-sided cases, it is drawn

to the right of the sternum, even sometimes under or outside the right nipple. Vocal fremitus is usually diminished and percussion gives dullness of varying degree over the fibroid area, while the unaffected parts may be hyper-resonant from "compensatory" emphysema. The diaphragm may be drawn up, and the liver or stomach correspondingly displaced. The breath-sounds are often weak or inaudible unless there is bronchiectasis or cavitation, when the characteristic signs of these conditions can be recognised. The vocal resonance is diminished if there is much pleural thickening, increased if cavities are present. Adventitious sounds may be entirely absent, and when present vary from rhonchi and bubbling râles to coarse metallic râles, according to the presence or absence of excavation. X-ray examination gives useful confirmation, showing displacement, excavation and pleural thickening.

Course.—The course is invariably chronic, and may extend to years, even ten or twenty.

Diagnosis.—The diagnosis is usually easy. The evidence of contraction and of mediastinal displacement towards the affected side, especially if signs of cavitation are also present, is highly suggestive. In the absence of the cavitation some difficulty may arise in regard to chronic pleural effusion or empyema. In the earlier stages the contra-lateral displacement of the cardiac impulse should prevent any mistake, but where partial absorption has occurred, this may be very slight or absent. In such cases an exploratory puncture or an X-ray examination may be helpful.

When the diagnosis of pulmonary fibrosis has been made, the differentiation of the cause is an essential to prognosis and treatment. If the condition is apical, there is a presumption in favour of tuberculosis; if basilar, some other cause is more probable. Repeated examinations of the sputum should be made for tubercle bacilli, and if these prove negative, X-ray examination may reveal some cause such as new-growth, aneurysm or even foreign body. In some cases a careful consideration of the history may afford a clue to the diagnosis.

Prognosis and Treatment.—These depend upon the primary condition, but in most cases the latter is mainly symptomatic.

PNEUMOKONIOSIS

Synonyms.—Pneumonokoniosis; Dust Disease of the Lung.

Definition.—Pneumokoniosis comprises all the pathological changes induced in the bronchi, lung and pleuræ by the inhalation of dust particles.

Ætiology.—*Predisposing causes.*—Pneumokoniosis is one of the occupational diseases. It is practically limited to men, and usually develops between the ages of 25 and 40. Defective ventilation, bad hygienic conditions and alcoholism promote its incidence.

Exciting causes.—Various forms of dust, both inorganic and organic, serve to produce pneumokoniosis, and in general the harder and more gritty the particles, the more marked are the changes induced. Organic forms of dust lead especially to bronchitic changes, the inorganic forms to pulmonary fibrosis.

The following varieties are recognised :

1. *Anthracosis* from coal dust (coal-miners' phthisis).
2. *Siderosis* (silico-siderosis), from the inhalation of fine particles in tin, copper, lead and iron miners, and in grinders of steel goods (grinders' rot).
3. *Silicosis* or chalicosis, met with in workers in quartz, gannister and slate quarries, also in potters (quarts-miners' phthisis, and potters' asthma). Gold-miners' phthisis, the most serious form of pneumokoniosis, and especially prevalent in the South African gold mines, is due to the fine dust caused by the rock drills.

4. *Byssinosis*, a rare variety, is met with in cotton workers, felt-hat makers, and the employees in shoddy mills.

5. *Asbestosis*.—A condition found occasionally in those working in the manufacture of asbestos articles. Asbestos is composed of compound silicates of iron and magnesium.

Pathology.—The lungs of persons living under rural conditions are practically free from deposited pigment. A certain amount of carbon is invariably present in the lungs of town-dwellers, giving them a dark-grey mottled appearance, but producing no pulmonary fibrosis. In coal-miners this occurs to such an extent that the lungs are black (anthracosis), although even here little fibrosis occurs, except in miners of hard coal or anthracite. In siderosis and silicosis, fine sharp particles of metallic oxides or silica are deposited in the lung tissue. According to Stewart, siderosis is in effect silico-siderosis, the damaging agent being silica inhaled at the same time. In asbestosis, curious irregular discoid structures of golden yellow colour and containing iron, now called "asbestosis bodies," are found in the lungs and in the sputum. There is also much fibrosis in pneumokoniosis, and tuberculosis is liable to be a later development.

It is generally accepted that these particles are conveyed to the bronchi and alveoli by inhalation. In normal breathing, most of the coarse particles are detained in the nose, and are discharged with the nasal mucus, whereas in mouth-breathers they readily gain access to the trachea and bronchi. Even then, the coarser particles may be discharged, in the expectoration through the agency of the ciliated epithelium, but, owing to the catarrhal processes induced by the irritation of the inhaled dust, this epithelium may be desquamated and the absorption of the particles is promoted. As a further consequence of this initial bronchitis, the finer particles may reach the alveoli, and passing between the epithelial cells, gain access to the tissue spaces, or in some cases they may be taken up into special "dust cells." In silicosis, particles of crystalline silica become deposited in the connective tissue, and chronic peribronchial and perialveolar fibrosis develops. The bronchial glands also become enlarged by the deposition of similar particles conveyed by the lymphatics. Other changes more or less constantly present are emphysema, pleural adhesion and bronchiectasis.

The relationship to tuberculosis has been much debated. It is now established that pneumokoniosis is non-tuberculous in origin, and that it may remain so throughout its course. On the other hand, certain forms undoubtedly favour the development of tuberculosis. In England and Wales coal-miners suffer less from tuberculosis than do all other males. On the other hand, gold-miners are extremely liable to it. It would appear that the determining factor is the presence of particles of silica. Silicates, as

in clay, do not induce tuberculosis. Workers in freestone develop this disease, limestone workers do not. Slate quarries do not acquire tuberculosis very readily, while metalliferous miners working in quartzite very frequently suffer from it. The Miners Phthisis Bureau recognises two types of silicosis. (1) Simple silicosis, the damage found being due to dust alone. It is non-progressive if exposure ceases. (2) Tuberculo-silicosis, in which most of the damage is due to dust, and tuberculosis is secondary. Haldane and Mavrogordato demonstrated that particles of coal are absorbed by the "dust cells" whose movements are thereby stimulated, with the result that they appear in the black spit, which is therefore a healthy sign. The particles of silica are also absorbed by these "dust cells," but no stimulus to their movement is induced and they remain *in situ*. Kettle and Gye have shown that a silica colloid is slowly formed, which leads to breakdown of the tissue defences and thus favours the activity of tubercle bacilli.

Post mortem, the lungs are generally firm and pigmented, the colour varying with the cause, being black in anthracosis, reddish-brown in siderosis, and greyish-black in silicosis. The pleura is generally adherent, especially at the bases. On section the lungs are firm, and often gritty. Small hard nodules may be felt with the finger. Fibroid changes are especially marked in silicosis. The bronchi are inflamed and sometimes dilated. Some degree of emphysema is usually apparent. If tuberculous lesions are also present, these vary from fibroid areas to miliary nodules. Destructive processes resulting in cavitation may also be seen. Microscopically, the alveolar walls are thickened, the connective tissue is everywhere found to be increased, the "dust cells" may be seen in the connective tissue or in the alveoli, and particles of pigment or silica are found widely deposited in the connective tissue cells.

Symptoms.—The onset is insidious, bronchial irritation and cough, especially in the morning, may be the first indications, but increasing shortness of breath and debility are frequently early symptoms. The expectoration, at first scanty and mucoid, becomes more abundant and may present characteristic features as in the "black spit" of anthracosis. Tinging of the sputum and later hæmoptysis occur, but these suggest the possibility of superadded tuberculosis. The patient may appear healthy and be but little troubled except by the shortness of breath, but later emaciation and an appearance of premature old age are not uncommon.

The physical signs are not characteristic; at first they are simply those of persistent bronchitis, then emphysematous changes may become apparent. Later, signs of fibrosis appear, very similar to those described in the preceding section. Even when tuberculosis develops the signs are often but little characteristic, and repeated sputum tests may be necessary to establish the diagnosis. Examination by X-rays may be helpful; at first there is an increase in the reticulation and later nodulation, somewhat like that of miliary tuberculosis. At a still later stage, the nodules become larger, and there is increased fibrosis. The changes characteristic of tuberculosis may be super-added.

Complications and Sequelæ.—The most important complication is tuberculosis, which forms the terminal stage of many cases of silicosis. This may be suspected when fever, night sweats, hæmoptysis or emaciation develop. Bronchiectasis of considerable degree sometimes results as a

consequence of the fibrosis, and leads to the symptoms and signs characteristic of that condition.

Course.—This is progressive, unless the sufferer is removed from the exciting causes. Anthracosis runs a very chronic course, siderosis somewhat less so, while gold miners only live a few years (5 to 6—Oliver) after the onset of the disease.

Diagnosis.—The diagnosis can usually be made from the history of shortness of breath, cough and expectoration, developing in a worker in a dusty occupation. In the early stages, cigarette-smoker's cough and bronchitis may give rise to difficulty. In the later stages, the possibility of a primary fibroid tuberculosis has to be considered.

Prognosis.—This is unfavourable except in anthracosis. Lyle Cummins suggests that the finely divided carbon particles absorb the toxins of the tubercle bacillus. If recognised early, and if the patient is taken from the dusty conditions, recovery may be anticipated. The development of tuberculosis affects the outlook very gravely.

Treatment.—*Prophylactic.*—Every means should be adopted to avoid the dusty conditions leading to the disease. Mines should be well ventilated, and respirators should be worn where practicable in dusty occupations. Factories and workshops should be provided with apparatus to draw away dust. Sprays or jets should be used with drills to moisten the dust produced.

Curative.—Directly the condition is diagnosed, the patient should be advised to change his occupation. Nutrition should be kept at a satisfactory level. Symptoms and associated conditions, such as bronchitis or tuberculosis, should be treated on general principles.

PULMONARY TUBERCULOSIS

Synonyms.—Phthisis ; Consumption ; Decline.

Pulmonary tuberculosis embraces all the abnormal conditions induced by infection of the lungs, pleuræ and bronchial glands with the tubercle bacillus.

Ætiology.—**PREDISPOSING CAUSES.**—**Age.**—The maximum age incidence is between the 15th and 45th years, although the disease may be encountered at any age. Senile tuberculosis is more common than is generally recognised.

Sex.—The disease is more frequent in males, but between the ages of 5 and 15 the female sex shows a preponderance.

Heredity.—Pulmonary tuberculosis certainly occurs with undue frequency in certain families. Since the direct transmission of the tubercle bacillus to the infant is extremely rare, two explanations seem possible—(1) Children born of tuberculous stock may inherit an increased susceptibility or diminished resistance, the tuberculous diathesis ; or (2) they may contract tuberculosis on account of their exposure to massive infection in early life.

Race.—Differences in racial susceptibility probably depend upon the degree of inherited resistance acquired by the race from infection of previous generations. Native races suffer severely when first exposed. In Europe the Irish are particularly susceptible, whereas the Jews are relatively immune.

Climate.—Tuberculosis occurs in all climates. The prevalence of strong

rainy winds and defective subsoil drainage may tend to increase its incidence.

Occupation.—The highest mortality from tuberculosis occurs in England amongst the workers in dusty occupations, thus Cornish tin miners head the list. On the other hand, coal miners are notably free from the disease. Any conditions leading to overwork or to underfeeding increase the liability to tuberculosis.

Environment.—Overcrowding, defective sanitation, dampness, dirt, lack of sunlight and insufficient ventilation are most potent factors in the spread of the disease, causing both lowering of the resistance and increased facilities for direct infection.

Trauma.—Trauma, involving the chest-wall, may be followed by active pulmonary tuberculosis. This is probably because the injury leads to activity of previously arrested disease, rather than to fresh infection at a spot of lowered resistance.

Gassing.—In certain cases the inhalation of poison gases causes rapid activity and spread in latent disease, or it may possibly prepare the ground for reinfection, but it is not a factor of great ætiological importance.

The influence of other diseases and conditions.—The following diseases predispose to the development of pulmonary tuberculosis: measles, especially when complicated by broncho-pneumonia, whooping-cough, influenza, pneumokoniosis, alcoholism, diabetes, syphilis, congenital heart disease and insanity. Tuberculosis may manifest itself for the first time during prolonged lactation or after repeated pregnancies; when previously existent it often remains quiescent during pregnancy, but it may spread rapidly after childbirth. Contrary to the usual belief, pulmonary tuberculosis not infrequently coexists with mitral stenosis. Cases apparently following pneumonia, pleurisy or bronchitis are usually tuberculous from the onset.

EXCITING CAUSES.—The causal organism is the *Bacillus tuberculosis*, discovered by Koch in 1882. It exists in four main forms, human, bovine, avian and reptilian; only the two former usually occur in man, but avian infection has been recorded. The human type is found in over 97 per cent. of pulmonary tuberculous lesions, though a higher proportion of the bovine type has been found in Scotland. In glandular tuberculosis up to the age of 5 years, over 80 per cent. of the bacilli isolated conform to the bovine variety. In tuberculosis of bones and joints up to the same age, 29 per cent. of the cases are of bovine origin.

The bacilli may gain access to the body by inhalation, by alimentary ingestion, through the tonsils, through the skin, or possibly, in rare instances, by hereditary transmission. It is probable that in the majority of cases of pulmonary tuberculosis in adults, the organisms are carried direct to the lungs in the inspired air, and Ghon showed that in children, who had died of tuberculosis of the lungs, a primary focus was present in the lungs in 92.4 per cent. As, however, extensive tuberculous lesions are frequently found in the bronchial glands in cases of pulmonary tuberculosis, it is believed by some that the glands are primarily affected, and that the bacilli pass from them to the lungs, either against the lymphatic flow or in the blood stream. Calmette and others have demonstrated that the bacilli may gain access to the bronchial glands from the alimentary tract through the

thoracic duct, or from the tonsils through the cervical and mediastinal glands. Cases have been recorded in which primary cutaneous infections have been followed later by active pulmonary tuberculosis. Direct transmission of the tubercle bacillus is believed to occur only when the mother is suffering from advanced tuberculosis, and even then is of great rarity.

The incubation period tuberculosis of is uncertain, owing to the difficulty in determining when infection takes place. It is now believed by many authorities that the majority of individuals are originally infected in infancy or early childhood, either from the ingestion of tuberculous milk, or by the inhalation of bacilli from dried sputum. Pulmonary tuberculosis is thus regarded as a late manifestation comparable with the tertiary stage of syphilis. On this hypothesis, active pulmonary disease in adult life may result either from reinfection or from the activation of a dormant lesion in the body. As the organisms found in early life are frequently of the bovine type, whereas in pulmonary tuberculosis they are almost invariably of the human variety, it is probable that reinfection is the more common, since mutation of type has not so far been proved.

Provided that the proper precautions are taken, the risk of infection from adult to adult is not great, and only exists in "open" cases of tuberculosis, *i.e.* in cases with tubercle bacilli in the sputum. The occurrence of conjugal disease, which is less common than might be expected, has been explained by mating of those with hereditary diathesis.

Pathology.—The earliest lesion in the lung is the formation of tubercles, whose structure is described in the general article on tuberculosis. They usually appear first near the apex. This may be due to the relative immobility of this portion of the lung, possibly as the result of calcification of the first costal cartilage (Freund), but in some cases the bacilli may spread from the cervical to the supraclavicular glands and thence to the adjacent lung. In some cases the earliest lesion is found in the subclavicular region well below the apex. It may commence in a subacute manner. In such cases, an area of localised deposit may be seen on radiological examination—known as Assmann's focus (Redecker's "*früh infiltrat*"). The initial deposit is usually in or around the small bronchioles of the third to fifth degree (Hirschfeld's bronchioles). The inflammatory swelling of the bronchioles obstructs their lumen, leading to collapse of the alveoli beyond and the formation of bronchopneumonic areas. At the same time peri-bronchiolar inflammation develops. In children there is, in the majority of cases, a primary lung focus (Ghon's focus), with secondary deposits in the bronchial glands.

SECONDARY CHANGES.—1. *Caseation.*—The tubercle is avascular, and owing to this, and possibly also to the action of tubercle toxins, coagulation necrosis and fatty degeneration frequently ensue. This combined process is known as caseation and results in the formation of a structureless, cheesy mass. Further changes may now occur, either softening, with the development of a "cold abscess" filled with tuberculous "pus," or calcification, with the subsequent formation of gritty masses known as "pneumoliths."

2. *Cavitation.*—Cavities result from the liquefaction of caseous areas, and the expectoration of the resulting debris. They may be no larger than a pea, or may occupy the whole of one or more lobes. A recent cavity has an irregular outline, with rough, shaggy walls and a vascular line of demarcation. It is often traversed by trabeculae, formed by bronchi and vessels

which may be partly or completely obliterated, while sometimes the trabeculae consist of condensed lung tissue which originally separated adjacent cavities. In chronic cases, the cavity is surrounded by fibrosed lung tissue forming a pseudo-capsule, and its interior becomes lined by a thin, smooth, false membrane. Small aneurysms may be found, arising either from vessels running in the walls or in the trabeculae of the cavity, the former being the more common. In some cases, where hæmoptysis has occurred, rupture of such an aneurysm is the cause.

3. *Fibrosis*.—Reactive changes in the lung stroma lead to the formation of fibrous tissue. This may occur early or after caseation has taken place. Such changes are often classified as productive, whereas infiltration and the earlier reactive changes are referred to as exudative.

In the majority of deaths from all causes, old tuberculous lesions are found post mortem near the apex of one lung. These consist of small nodules of arrested disease, with thickening and dimpling of the adjacent pleura.

DISSEMINATION IN THE LUNGS.—The disease may spread from the primary peri-bronchial deposit—(a) By direct infiltration; (b) By the peri-bronchial lymphatics and capillaries, leading to a racemose appearance or to peri-bronchial fibrosis; (c) By the subpleural and interstitial lymphatics, with localised miliary dissemination; (d) By inhalation into a bronchus of tuberculous material, which is then carried to other parts of the same or to the opposite lung—this not infrequently happens after hæmoptysis and in cavitation; (e) By the blood vessels, *e.g.* generalised miliary tuberculosis may result from erosion of a caseous tubercle into a vein.

The pathology of the clinically distinguishable forms of pulmonary tuberculosis will now be described.

1. ACUTE MILIARY TUBERCULOSIS.—A primary caseous focus may be discovered at the apex of one lung, in the bronchial glands, or at some distant spot in the body. Local erosion of a vein may be found, accounting for the dissemination of the disease. The lungs are usually studded with minute grey tubercles, the smaller ones requiring a hand lens for their recognition. In very acute cases death occurs before any secondary broncho-pneumonic changes take place. Miliary tuberculosis may develop as a terminal event in chronic fibro-caseous or fibroid tuberculosis. The tubercles are then found in large numbers around the old foci of disease, but to a less extent in the more remote portions of the lung.

2. CHRONIC MILIARY TUBERCULOSIS.—The lungs are studded uniformly with firm nodules varying in size from one to several millimetres in diameter. They are grey or white, project from the cut surface of the lung, and in some cases are calcified. There may be, in addition, a diffuse fine fibrosis. Miliary tubercles are at times found scantily distributed in the spleen, kidneys and liver. Not infrequently there is evidence of terminal acute miliary tuberculosis involving the brain and meninges.

3. ACUTE CASEOUS TUBERCULOSIS.—Large areas of consolidation form rapidly, which differ histologically from the common chronic tuberculous broncho-pneumonia in that the alveolar exudate is more definitely inflammatory and contains fibrin. In the rare lobar cases, the rapid caseation and the presence of tubercle bacilli show that the caseous pneumonia is a specific process. Firm yellowish patches, which may be confluent, are seen,

usually scattered throughout both lungs. The affected areas are airless and sink in water. Softening is generally present in varying forms up to actual cavity formation, which may be extensive, involving even a whole lobe.

4. **FIBRO-CASEOUS TUBERCULOSIS.**—This is the commonest variety of the disease; the appearances of the lung vary with the relative preponderance of the caseous and fibrotic changes. The early lesions are miliary or bronchopneumonic, but areas of caseation in varying stages, including cavitation, are almost always present. The older lesions show considerable fibrosis, the strands of sclerotic tissue being pigmented and glistening. The earliest lesion is usually near the apex of the upper lobe at the back, more rarely a little lower and towards the front. The apex of the lower lobe is next affected, and the disease then spreads in the direction of the interlobar septum; the apex of the upper lobe of the opposite lung is next involved (Fowler's law of spread). Pleural adhesions are usually present over the oldest lesions, and in the interlobar fissures. An open cavity, from which infected sputum is discharged, is a danger, being a frequent cause of spread.

5. **FIBROID TUBERCULOSIS.**—Fibrosis may be localised around a small arrested lesion, or may spread throughout a lung in which caseation or excavation has occurred. One lobe or the whole lung is then contracted and firm. In the interstices of the fibrous tissue, which is usually pigmented, inspissated caseous material, calcareous patches, or cavities are seen. The shrinkage may lead to bronchiectasis, especially in the lower lobes. The overlying pleura is much thickened and adherent, and the mediastinum is drawn over towards the affected side. The opposite lung, or the sound portions of the fibrosed one, may show compensatory emphysema.

The bronchial glands.—The tracheo-bronchial glands are affected in all forms of pulmonary tuberculosis. They are enlarged, sometimes pigmented, and may present miliary, caseous, calcareous or fibroid changes, in some cases primary, in others secondary to the lesions in the lungs.

The pleura.—This, too, is almost constantly affected. The commonest changes are an early dry pleurisy, and a later thickening with adhesions which may completely unite the visceral with the parietal layers. In acute disease or active spread, the pleura may be studded with miliary tubercles, leading to a large serous effusion.

The post-mortem appearances of the lesions situated in the other organs, found as complications of pulmonary tuberculosis, are described in the respective sections dealing with them, and include tuberculous meningitis, peritonitis, enteritis and genito-urinary tuberculosis. There is usually atrophy of the skeletal muscles, sometimes lardaceous and fatty degeneration of the liver, and hypoplasia with fatty degeneration of the heart.

Symptoms.—The symptoms fall into three groups (Pottenger)—(1) pulmonary, such as catarrh, expectoration, hæmoptysis and pleurisy; (2) reflex, such as pain, cough and laryngeal irritability; (3) toxæmic, including malaise, tachycardia, pyrexia and loss of weight.

ONSET.—The mode of onset is very variable, but certain forms can be recognised.

(a) *Insidious.*—The early symptoms may be malaise, anæmia, amenorrhœa, cardiac irritability, progressive loss of weight, and slight rise of temperature, generally towards evening. Cough and expectoration often appear only when the signs in the chest are quite apparent. When there is intestinal

stasis, the cutaneous pigmentation may suggest the diagnosis of Addison's disease.

(b) *Catarrhal*.—A series of febrile "colds" may usher in the disease, and such a sequence is always suspicious.

(c) *Phthisis ab hæmoptoe*.—Hæmoptysis may first draw attention to the lungs. It may be slight, and is then due to early congestion around the focus of infection. If it is more marked, it indicates breakdown of an old arrested lesion, or may afford dramatic evidence of extensive disease which had not been recognised previously.

(d) *Laryngeal*.—Hoarseness or aphonia may be the first symptom, but laryngeal tuberculosis is usually secondary to pulmonary disease, although the latter may have been unsuspected.

(e) *Gastro-intestinal*.—Anorexia and flatulence often occur early. When they are accompanied by slight loss of weight and pyrexia, the possibility of pulmonary tuberculosis should be suspected.

(f) *Pleural*.—Dry pleurisy is a frequent manifestation of latent pulmonary tuberculosis. When a serous effusion develops, its tuberculous character can be determined by laboratory investigations. Pneumothorax, developing in a previously healthy individual is a rare but often serious clinical mode of onset.

(g) *Pneumonic*.—"Gallopings" consumption often begins with pneumonic manifestations, especially in the young.

(h) *Traumatic*.—Pulmonary tuberculosis may follow injury or "gassing," as described under ætiology.

(i) *Neurasthenic*.—Neurasthenia may occur as a complication of tuberculosis; but in some cases an initial neurasthenia dominates the picture, and the pulmonary lesion is only detected on careful examination.

(j) *Malarial*.—Regular attacks of sweating and fever may occur, especially in those who are or who have been residing in malarial climates, suggesting malaria, but in reality due to tuberculosis.

(k) *Associated with other diseases*.—Tuberculosis may follow immediately on an attack of measles, influenza or whooping-cough, especially if complicated by broncho-pneumonia. In some cases it develops at a later period after the acute disease.

(l) *Senile*.—In old people an insidious onset is common. The disease may be of bronchitic type, and the signs are often masked by emphysema. There may be little or no rise of temperature.

THE CHIEF SYMPTOMS of pulmonary tuberculosis are—

Cough.—This varies considerably in different types of disease. It may be very slight or absent in generalised miliary tuberculosis, or in any form in the insane. It is sometimes dry, persistent and ineffective, especially in miliary extension in the lungs from an old focus of disease, in bronchial gland tuberculosis, or in pleurisy. When there is associated bronchitis or caseation, the cough is usually accompanied by expectoration, which, if very tenacious, may lead to retching or even to vomiting, particularly in the morning. In laryngeal tuberculosis the cough is husky and frequently painful.

Expectoration.—In early disease there is usually no sputum, and in some cases, more especially in the fibroid type, widespread lesions may be present with practically no expectoration. When caseation is in progress, or when

there is secondary infection with bronchitis, the sputum may be abundant and amount to as much as 20 or more ounces in the 24 hours. It may be clear or mucoid, or thick tenacious muco-pus. If mucoid, it often contains small particles, the size of a pin's head or larger, of yellow caseous material. Nummular sputum may be met with in active caseous disease, especially with excavation. This consists of flat rounded masses of muco-pus, with a somewhat distant resemblance to coins. In tuberculosis the sputum is usually inoffensive, but may have the characteristic sickly odour which is also noticed to emanate from the patient himself (odor phthisicus). If bronchiectasis or gangrene occurs as a complication, the expectoration becomes typically malodorous. Pulmonary calculi or pneumoliths, composed chiefly of calcium carbonate or phosphate, are sometimes expectorated. They vary in size from a pin's head to a pea, are irregular in outline and sometimes branched, being derived generally from the walls of a cavity. Although the occurrence of these does not necessarily indicate fresh activity in the lungs, yet such a possibility should always be suspected, and a careful watch maintained on the temperature during the next few days. In some cases larger pneumoliths, as big as a cherry, may be coughed up, and those are frequently derived from calcified tracheo-bronchial glands. They may give rise to alarming symptoms at once, and be the forerunner of fresh activity in the lungs.

Microscopical examination.—The presence of tubercle bacilli in the sputum is the most decisive test of the existence of this disease. The small yellowish caseous particles should be selected from the sputum, and appropriately stained. If no tubercle bacilli are found, samples from the whole sputum of the 24 hours, concentrated by the antiformin method, can be examined. Droplets collected on a laryngeal mirror by cough induced by it may be examined for the presence of tubercle bacilli, especially in children or in patients who habitually swallow sputum.

Sputum culture by the Loewenstein method may be of value when tubercle bacilli are not found in smears. The cells present are usually of the mononuclear type, either mononuclear leucocytes or altered alveolar epithelial cells. The presence of elastic tissue indicates that destructive pulmonary lesions are in progress. Secondary infecting organisms may be demonstrated by cultural methods.

Dyspnœa.—Slight dyspnœa occurring early in the disease may be due to diminished movement of the diaphragm on the affected side. In more advanced cases the degree of dyspnœa is proportional to the amount of lung tissue involved. In addition, cough and pyrexia play a part in its production. Complications such as pleurisy, pleural effusion, pneumothorax and cardiac failure increase the shortness of breath. It is rare to find orthopnœa even in acute and rapidly spreading disease. In arrested cases the dyspnœa is proportional to the extent of fibrosis.

Cyanosis.—This is not an early symptom of tuberculosis. It is dependent upon the amount of lung tissue involved, but is increased by the coexistence of emphysema or cardiac failure. The typical "hectic flush" of tuberculosis is a vasomotor effect caused by toxæmia.

Pain.—Not every sufferer from tuberculosis experiences pain, even in the acute stages of the disease. The commonest cause of pain is dry pleurisy. When the diaphragmatic layer of the pleura is affected, pain may be referred to the epigastrium or to the corresponding shoulder. In chronic fibroid

phthisis there is frequently a dull, aching pain in the chest, which is more noticeable in damp weather. This is caused by the contraction of the condensing fibrous tissue. Cutaneous tenderness of the chest-wall is met with in some cases of advanced disease, and is probably due to a cachectic neuritis. A "cold abscess" forming along one of the ribs or costal cartilages is a rare cause of localised pain in the chest-wall. Cough may be painful, especially when paroxysmal or frequent, the pain being referred to the costal attachments of the diaphragm and upper abdominal muscles. The sudden occurrence of pneumothorax may cause such severe pain as to induce collapse; but when of more gradual onset no severe discomfort may be experienced. Tuberculous laryngitis may be the cause of very great suffering.

Night sweats.—Although not pathognomonic, night sweats occur more frequently in tuberculosis than in other diseases. They are met with in all stages of active lesions, and may be of great severity.

Loss of weight.—This is often an early symptom. It is most marked in acute disease and in the late stages of chronic fibro-caseous tuberculosis.

Fever.—Pyrexia is one of the most important indications of activity at any stage of pulmonary tuberculosis, although it does not follow that the disease is arrested when there is no fever. During treatment the temperature should be recorded at certain definite hours in the day. (a) On waking. The normal mouth temperature at 7 or 8 a.m. is 97° or 98° F. in the mouth, and 97°·2 to 99° F. in the rectum. This temperature should be taken in bed, before eating or drinking. (b) At 1 p.m., after the hour's recumbent rest. (c) At 6 p.m. (d) At 9 p.m., after retiring to bed. The maximum temperature is usually reached between 4 and 6 p.m., but may be delayed to 8 or 9 p.m. In most Continental sanatoria the rectal temperature is taken, and a centigrade thermometer employed. The temperature is dependent upon the extent and the activity of the disease, and upon the amount of exercise taken.

(a) In acute miliary tuberculosis it may be continuous or remittent, and the "typus inversus" is not uncommon, the morning temperature being higher than the evening. This is generally regarded as a sign of grave prognosis.

(b) In acute caseous tuberculosis the high temperature at the onset is continuous, and the record resembles a pneumonic chart. When caseation occurs it becomes hectic or intermittent, with a daily swing of 4° or 5° F. This is probably due to the action of tubercle toxins, and not to the presence of a secondary infection.

(c) In chronic fibro-caseous tuberculosis there is no characteristic temperature record. There may be only a very slight rise occurring at intervals of a few days. On the other hand, the patient may be afebrile while resting, but febrile when ambulant (Stage 2. Inman's classification). Further an afebrile ambulant patient may over-exert himself, and by excessive auto-inoculation develop a sharp rise of temperature which subsides in a few days with rest. The temperature chart is thus a guide to prognosis and to treatment, and if acute miliary tuberculosis or caseation occurs, a typical temperature variation ensues.

(d) In fibroid tuberculosis the temperature is usually normal, unless excessive auto-inoculation results from exercise, or the disease advances. The occurrence of hæmoptysis may have a very definite effect upon the temperature. In some cases it is not followed by pyrexia, but if the inhaled blood leads

to a hæmoptoic bronchitis, there may be a slight degree of fever lasting for a few days. When a definite and persistent pyrexia follows, it usually indicates activity around an old focus of disease, or fresh spread by inhalation of blood containing tubercle bacilli to distant parts of the lung.

A premenstrual rise of temperature may occur; but as it is also met with in healthy women it is not pathognomonic.

Hæmoptysis.—Hæmoptysis occurs at some stage of pulmonary tuberculosis in about 50 per cent. of all cases. With early lesions the sputum is only streaked. This may result from the congestion of tuberculous bronchitis, or from a small area of collapse or broncho-pneumonia. In the pneumonic or broncho-pneumonic forms rusty sputum may be seen. Profuse hæmoptysis generally occurs in chronic disease; but it is occasionally met with in acute caseous forms. Recovery may take place after coughing up 2 or 3 pints, or death may ensue rapidly from suffocation before any considerable quantity of blood has been expectorated. After the cessation of bleeding the sputum may be blood-stained for several days, the colour becoming darker. The source of profuse hæmoptysis is generally an aneurysm of a branch of the pulmonary artery lying in a cavity or in a fibroid lung, although occasionally ulceration without previous aneurysm formation may occur. In the majority of cases hæmoptysis begins while the patient is lying down or resting, so that exercise or work are not frequent exciting causes.

The patient notices a salt taste, feels a warm gush in the mouth, and then expectorates the blood. He is usually greatly alarmed, flushed and sweating, with rapidly beating heart. The blood at first is as a rule bright and frothy but some clots may be present; later it is mixed with muco-purulent expectoration, in the form of clots or streaks.

Circulatory system.—The heart may be small, but the right side often hypertrophies in chronic fibroid cases. Tachycardia may be due to nervousness, but when constant it generally indicates active disease or over-exertion on the part of the patient. The blood pressure is usually low in the stages of activity, and a steady rise during treatment is a favourable sign. If tuberculosis is coexistent with other diseases, such as atheroma, which raise the blood pressure, higher readings are naturally obtained.

The blood.—The red cells are usually normal in number, but there may be a slight anæmia. On the other hand, when there is much cyanosis, or after sanatorium treatment, the red cells may be increased. The colour index is usually low. In the early stages the leucocytes may be slightly increased. A polymorphonuclear leucocytosis occurs in caseation and in early cavity formation, and at times with secondary infection of the lungs. A special differential count of the polymorphonuclear leucocytes themselves may be made by subdividing them into groups, according to the number of their nuclear lobes, as suggested by Arneth. An increase in number of immature cells with only one or two nuclear lobes constitutes a deviation to the left from the normal, and raises the Arneth index. This lævo-deviation is said to indicate toxæmia, and if it is not present, the disease will probably be chronic. The von Bonsdorff modification in which the number of lobes of the nuclei in one hundred polymorphonuclear leucocytes is counted, is sometimes used. The normal figure is 274, a figure less than this suggests activity. The lymphocyte monocyte ratio is said to be of prognostic value, an absolute and relative increase in the monocytes being unfavourable.

Alimentary system.—The tongue is usually clean and the appetite good even in cases with marked fever. When tuberculosis of the larynx is present, there is frequently severe dysphagia. Dyspepsia may be complained of, usually of a nervous type. Anorexia, flatulence, distension with nausea are the commonest symptoms, pain being rarely noticed. There may be marked intolerance of fat in the diet. Atonic dilatation of the stomach may occur in some cases towards the end of the disease. Constipation is common; on the one hand, diarrhoea may occur apart from intestinal ulceration or lardaceous disease.

Nervous system.—The classical “*spes phthisica*” is distinctly rare, but when present is very striking from its contrast with the realities of the disease. It is most commonly seen in acute caseous tuberculosis. More often the patient becomes emotional and self-centred, depression is common and hard to combat, and melancholia with delusions occasionally develops. Neurasthenia is frequent and, as mentioned above, may lead to errors in early diagnosis. Insomnia may be due to cough, pyrexia, night sweats or pain, especially in laryngitis. With marked cachexia, a definite peripheral neuritis may occur.

Genito-urinary system.—In the early stages there is often an increased sexual desire, and this may recur when arrest is taking place. This is probably in part due to the therapeutic régime, the rest, abundant food and lack of interesting occupation reacting upon the nervous system of young adults. In advanced disease, all sexual desire is lost. Menstruation often ceases early, and the patient may seek advice for amenorrhoea. Women remain fertile even in advanced disease. The urine is normal in the early stages, later a febrile albuminuria may occur, or in advanced cases an amyloid nephrosis with generalised oedema may develop.

THE PHYSICAL SIGNS OF EARLY DISEASE.—The general appearance of the patient may be healthy, or may be that of malnutrition with the characteristics of the “*habitus phthisicus*,” the hair being lank and lustreless, the skin white, thin, dry and shiny, and the thorax of the alar or phthinoid type. Certain stigmata are described, which although useful, are not pathognomonic. The eyelashes may be long, dark and curling, the back hairy and the thoracic cutaneous venules dilated. When present around the upper thoracic vertebral spines, they are sometimes known as “the varicose zone of alarm.” Deficient movement may be observed below one clavicle, at the point of one shoulder, or at the lower costal margin. The corresponding shoulder may be slightly drooping, with flattening above or below the clavicle, and slight hollowing of the supra-spinous fossa with wasting of the trapezius muscle may be observed. Pottenger regarded these shoulder signs, when not due to scoliosis or kypho-scoliosis, as reflex, and comparable with the fixation of a tuberculous joint and wasting of its adjacent muscles. In women it may be noticed that the breast on the affected side is smaller and hangs at a lower level.

Palpation confirms the diminished expansion, and reveals a slight increase of vocal fremitus over the affected area of the lung, usually at one apex. The normal increase in fremitus of the right apex over the left must be borne in mind, in order to prevent mistakes.

With light percussion slight dullness and a small increase in the sense of resistance can be detected. This is usually most apparent in the

supra-spinous and upper interscapular regions. The extent of pulmonary resonance above the clavicle, known as "Krönig's isthmus," may be diminished by $\frac{1}{2}$ to 1 inch on the affected side.

Various types of breath-sounds may be heard over the affected portion of lung. They are—(a) weak inspiration, with expiration vesicular or inaudible; (b) cog-wheel inspiration, with expiration vesicular, prolonged or rarely jerky; (c) the "granular" breathing of Grancher, the breath-sounds being somewhat coarse and irregular, suggesting fine or distant râles, although none can be definitely detected; (d) harsh inspiration, with expiration vesicular or prolonged; (e) broncho-vesicular breathing; (f) definite bronchial breathing when early consolidation is in progress.

Often there are no adventitious sounds. Occasionally a few small rhonchi or fine crackling or bubbling râles may be heard with the first few deep breaths, or only with the inspiration immediately following coughing. If râles are constantly heard, it indicates that the lesion is already of some extent. Care must be taken to differentiate them from atelectatic râles, emphysematous râles audible along the sternal margin, and pleural friction or fascial creaks. There is usually a slight increase in the conduction of both the spoken and whispered voice, and the more definitely this extends away from the trachea in front, and from the vertebral spines behind, the more reliable is the sign as an indication of disease.

Mensuration is seldom practised in routine examination, but graphic records of the chest contour, which are of interest in following the progress of a case can be obtained by cyrtometry.

PHYSICAL SIGNS OF ACUTE MILIARY TUBERCULOSIS.—If the condition develops acutely from breaking down of an infected bronchial gland or small lung focus, the physical signs are generally those of an acute generalised broncho-pneumonia, unless there is meningeal involvement as well, in which case the pulmonary symptoms and signs are masked or obscured by those of the cerebral involvement. When miliary tuberculosis occurs as a terminal event in a chronic case, marked dyspnoea, cyanosis and tachycardia are early symptoms. There may be crepitations or fine crepitant râles widely distributed over both lungs, and sometimes areas of tubular breath-sounds especially in the lower lobes. The original signs are often masked or less apparent. This is especially the case if meningeal involvement occurs also.

THE PHYSICAL SIGNS OF CONSOLIDATION.—Limitation of movement and flattening over the affected part of the lung, usually the apex, is now more noticeable.

The diminution of movement is confirmed on palpation, and vocal fremitus is found to be definitely increased.

The pulmonary resonance is diminished to definite dullness and the sense of resistance is correspondingly increased.

The breath-sounds are bronchial, or in acute caseous disease may even approximate to tubular.

Adventitious sounds may be absent, but usually fine or medium crackling râles are heard with inspiration, especially after coughing. When active softening is in progress the râles frequently become coarse and sticky. The voice conduction is much increased, bronchophony and whispering pectoriloquy being audible.

Mensuration may confirm the presence of flattening.

THE PHYSICAL SIGNS OF EXCAVATION.—Flattening of the chest-wall and diminished movement over the cavity are now more marked; if the cavity is apical there is in addition notable dropping of the shoulder, and wasting of the shoulder-girdle muscles.

The diminution of movement is confirmed by palpation. Vocal fremitus is generally increased owing to the surrounding consolidation, but if the cavity is full or there is much pleural thickening, it is diminished.

The percussion note is dull when the cavity is small or filled with secretion. A peculiar boxy or "cracked-pot" note, the "*bruit de pot fêlé*," is obtained over large superficial cavities, especially when communicating with an open bronchus. This is best heard on percussing with the mouth open, and Wintrich showed that the note may be altered in pitch over such cavities when percussing with the mouth open or closed, apart from the actual presence of the cracked-pot sound. Gerhardt's sign (alteration of note with the position of the body) is supposed to indicate a cavity of oval shape. It is rare, and of little value.

The breath-sounds are bronchial, broncho-cavernous, cavernous or amphoric, according to the size of the cavity, and to the amount of its contents. When it is full the breath-sounds may be distant, weak or even absent, and this is especially noticeable in basal bronchiectasis.

With a dry cavity there may be no adventitious sounds. Usually râles are audible; they may be medium or large, and bubbling or crackling in character. Over a large cavity a metallic tinkle and amphoric echo may be heard. With a very large cavity, extending through the whole of one lung, a typical *bruit d'airain* is at times obtainable. Voice conduction is increased, bronchophony and whispering pectoriloquy are present, and in some instances post-tussive suction is heard. Some cavities are only revealed by X-ray examination or by tomography.

Mensuration affords a graphic representation of the flattening of the chest-wall.

THE PHYSICAL SIGNS OF FIBROSIS.—The chest is asymmetrical, the affected side being flattened and moving little, while compensatory scoliosis or kypho-scoliosis is often present. The cardiac impulse is seen to be displaced towards the affected lung and may be higher or lower than normal. It may be drawn over to the right axilla, or on the left side as far back as to the posterior axillary line, or even to the angle of the scapula. The intercostal spaces may be retracted, and dilated venules are sometimes seen over the front of the chest as the result of obstruction, caused by displacement of the mediastinum and traction on the deeper veins.

Diminution of movement is confirmed by palpation, and the cardiac impulse can be more accurately localised. Vocal fremitus may be increased or diminished; the former occurs when the lung is consolidated and the large bronchi patent, the latter when there is much pleural thickening.

The percussion note over fibroid lung is dull and the sense of resistance increased, unless cavities are present. The opposite lung may be hyper-resonant, and its resonance extend across the mid-sternal line. The cardiac dullness is often continuous with that of the fibroid lung, and its area can only be determined by the cardiac pulsation.

The breath-sounds are weak and distant, unless modified by the presence of a cavity.

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Often there are no adventitious sounds, although fine or medium râles of a sticky or metallic nature may be heard. The voice conduction is usually diminished, and there is no pectoriloquy unless excavation has occurred, when bronchophony and pectoriloquy are audible.

It must be borne in mind that in actual disease the lesions are not so clear-cut and well-defined. In a case of some duration different stages of disease can be found in the same individual, thus infiltration, consolidation with softening, excavation and fibrosis may be present in different lobes of the lungs, and thus it may be possible to determine the site of origin and path of spread of the disease.

Certain other signs are occasionally seen in pulmonary tuberculosis.

Myoidema is an undue irritability of the muscles to direct mechanical stimulation, revealing itself by a flickering fibrillary contraction on tapping with the finger, and may occur in tuberculosis at all stages. It is best seen over the pectoralis major on the affected side. It may be present quite early, but is not pathognomonic, as it may occur in any cachectic state.

Clubbing of the fingers is commonly seen in chronic cases, the nails are curved and present a parrot-beak appearance, the thumb, index and middle fingers being most affected. Drum-stick clubbing is only seen in fibroid lesions with bronchiectasis.

CHRONIC MILIARY TUBERCULOSIS.—*Clinical features.*—Chronic miliary tuberculosis affects males and females equally. The age incidence is between 6 and 40 years, but usually the patient is between 11 and 30. The early symptoms include cough, shortness of breath, pain in the chest, expectoration and at times hæmoptysis. Constitutional disturbance is usually slight with low-grade fever. In some cases the spleen is palpable and in others tuberculous lesions are present in glands, bones or joints. Tubercle bacilli may be found in the sputum, gastric contents or pleural fluid. X-ray examination reveals small symmetrical, rather ill-defined opacities throughout the lung-fields. In some cases they may be limited to one portion of the lung. The course of the disease is more prolonged than that of miliary tuberculosis, being never less than three months and usually considerably longer. Death may occur in about six months, or eventually the disease may be arrested.

The differentiation of chronic miliary tuberculosis from the acute variety depends rather upon the clinical than the radiological findings. The fact that the pulmonary lesions are tuberculous is established in many cases by the finding of tubercle bacilli in the sputum or stomach contents. When tubercle bacilli cannot be detected, other conditions giving rise to scattered opacities throughout the lung-fields must be considered. Thus in secondary carcinomatosis, sarcomatosis or chorio-epitheliomatosis, the opacities are usually larger. Multiple opacities are also found in pneumokoniosis, congestive heart failure, sarcoidosis and more rarely with periarteritis nodosa, xanthomatosis, bilharziasis, leukæmia and disseminated broncho-pneumonia.

HILUM TUBERCULOSIS.—The existence of a special type of pulmonary tuberculosis commencing at the root of the lungs and extending thence in a fan-shaped manner along the bronchi has been postulated by some authorities, chiefly on X-ray evidence. While lesions in the middle zone of the lung are sometimes revealed by X-ray examination, it is more than doubtful whether this condition merits recognition as a separate variety of the disease.

EPITUBERCULOSIS.—This term was applied by Eliasberg and Neuland in 1920 to a condition of consolidation in tuberculous infants, often affecting a whole lobe. In spite of definite physical signs and characteristic radiological appearances, there are few symptoms, and recovery is the rule, with fairly rapid clearing of the X-ray shadows, from the periphery inwards. It is probably due to atelectasis of a lobe in whole or part, due to bronchial obstruction from enlarged hilar glands.

HILAR FLARE is the name given by Burton Wood to a similar condition affecting the right middle lobe, or the upper part of either lower lobe in childhood. The X-ray shadow is generally triangular in shape, the base being at the hilum or mediastinum. This, like epituberculosis, is probably due in many cases to localised bronchial obstruction, but in both conditions it is possible that there may be an allergic factor.

It is important to note that in both epituberculosis and hilar flare, the chief evidence is radiological, and that the prognosis is good.

PULMONARY OSTEO-ARTHROPATHY.—In cases with bronchiectasis the joints may be affected, swelling occurring especially in the wrist, ankles and knees, and rarely in the hips and shoulders. A serous effusion into the joints may be present. Pain is usually slight, but there is much deformity and functional impairment. X-ray examination reveals productive periosteal changes, which may also affect the long bones and the spine (see p. 1376).

RADIOGRAPHY OF THE CHEST.—If possible, the chest should be examined in every case with the fluorescent screen, and a photograph taken on a film. Certain important points can only be determined by a screen examination. The chief of these are the respiratory movements of the diaphragm, lighting up of the apex of the lung with inspiration, and the cardiac pulsation. Unilateral restriction of diaphragmatic movement not infrequently occurs in early apical tuberculosis, but as it may be observed under other conditions, notably with pleural adhesions, it is not diagnostic. The film will show the extent of the disease and in some cases it may suggest the existence of activity; thus areas of consolidation, caseation or excavation can be demonstrated, and thickening of the pleura, pleural effusion and pneumothorax give their characteristic appearances. In films taken in the erect position a fluid level is often apparent in cavities. A curious rounded shadow may be apparent in early disease called Assmann's focus, in which rapid changes, such as softening, may occur. The tomograph sometimes shows unsuspected cavities.

The significance of "root shadows" is still debatable. Although the presence of calcareous deposit in the glands at the roots of the lungs is usually obvious, the interpretation of the radiating shadows is a matter still under discussion. They may be due to peribronchial thickening, caused by the formation of fibrous tissue, or may merely represent the shadows cast by the branches of the pulmonary artery.

A film is often of crucial value in determining the presence or absence of early disease, but slight diminution of translucency of one apex may be due to an old arrested lesion, or, on the other hand, there may be definite physical signs of active disease, without abnormalities being found on X-ray examination. The heart shadow is often narrow and vertical in tuberculosis. Displacement of the heart due to pulmonary fibrosis or to affections of the pleura is clearly indicated. A good film may also give valuable information as to the

extent of lung involved and as to the presence of complications, such as effusion, pneumothorax or bronchiectasis. As the X-rays only cast shadows lacking in all pictorial details, tuberculous shadows cannot always be distinguished from those due to other pulmonary lesions. It is thus clear that the X-ray findings should always be interpreted in connection with the history, symptoms and physical signs of the case. The X-rays, although often of great help, do not afford a simple road to diagnosis or supply infallible evidence in determining the nature of an obscure case. On the other hand, they are absolutely essential in the determination of the suitability of a case for artificial pneumothorax or other collapse treatment and in controlling its application.

Complications and Sequelæ.—Compensatory emphysema is common in chronic fibroid disease, but bronchiectasis occurs less frequently. Gangrene of the lung is not often observed. Colds and catarrhal affections of the respiratory passages are frequent in sufferers from tuberculosis, and lobar pneumonia may develop as a complication. Bronchitis often occurs, due either to spread of the tuberculous process or to a secondary infection. In some instances asthma appears for the first time after tuberculosis has become manifest. A tuberculous abscess occasionally forms about a rib or costal cartilage.

Small areas of dry pleurisy are present at some stage in nearly every case; a serous pleural effusion is common, and an empyema may develop as the result of a mixed infection, or from the tubercle bacillus alone. Pneumothorax may occur as an early complication, or late in the disease, generally from rupture of a caseous focus just under the pleura; this frequently progresses to the formation of a pyo-pneumothorax. The implantation of tubercle bacilli from the expired air or sputum may lead to secondary foci in the larynx, trachea and epiglottis, or more rarely in the pharynx, tonsils, base of the tongue or nose. Swallowing of sputum containing tubercle bacilli gives rise to gastro-intestinal complications in many cases. The most common site of tuberculous ulcers, is the terminal portion of the small intestine, but the appendix may be affected, and the connective tissue around the cæcum is sometimes matted and thickened to form a palpable mass (hypertrophic tuberculoma). Tuberculous peritonitis is not common in adults and is usually secondary to intestinal lesions. The stomach is very rarely ulcerated, but an atrophic gastritis may occur in advanced cases. Fistula-in-ano and ischio-rectal abscess are comparatively common complications and tubercle bacilli may be found in the discharges.

Small vegetations may be found post mortem in the heart on the aortic and mitral valves, but these are usually due to some terminal infection. Fatty degeneration of the myocardium occurs as a result of toxæmia, and infection by direct spread along the lymphatics may lead to pericarditis. The peripheral circulation is not infrequently poor, chilblains are common, and cachectic purpura may be seen. Lardaceous degeneration as a consequence of chronic tuberculosis is not so common nowadays as formerly, but when present may affect the liver, spleen, intestines, lymph glands and kidneys.

The genito-urinary complications include lesions in the kidneys, bladder, epididymis and prostate. If the suprarenal body is affected Addison's disease will usually develop. Spinal caries is occasionally observed. A peripheral neuritis may form part of the lesions occurring with marked

cachexia. Generalised dissemination of the tubercle bacilli by the blood stream is followed by tuberculous meningitis.

Course.—The course pursued by pulmonary tuberculosis is variable, depending upon the clinical type of the disease. In acute miliary tuberculosis, death may occur in from 1 to 3 weeks from toxæmia or generalisation of the lesions. In acute caseous tuberculosis, death usually results in from 1 to 6 months. In chronic fibro-caseous tuberculosis, the disease may be completely arrested, or after a temporary arrest may become active at intervals and again become arrested under suitable treatment; in other instances it progresses steadily to a fatal termination. In fibroid tuberculosis the disease may become completely arrested or smoulder quietly for many years.

Apart from the question of the expectation of life, various stages of tuberculosis are described based upon anatomical lesions, toxæmia and functional disablement. The most important of these are as follows:

1. **THE TURBAN-GERHARDT CLASSIFICATION.**—An anatomical classification based upon the extent of lung tissue involved. Three stages are described. *Stage 1.* Early cases in which physical signs, if unilateral, only extend from the apex to the second rib, and, if bilateral, are limited to the supra-clavicular and supra-spinous regions. *Stage 2.* The signs, if unilateral, do not reach lower than the fourth rib, and, if bilateral, are situated above the second ribs. Excavation is not present in this stage. *Stage 3.* This includes more extensive lesions or localised ones in which excavation is present.

2. **SIR ROBERT PHILIP'S CLASSIFICATION.**—Both the extent of lung tissue involved and the degree of toxæmia present are taken into consideration. Twelve stages are described, which are indicated by the following signs:

L_1 , L_1S , L_2S , and L_3S ; L_2 , L_2S , L_3S , and L_3S ; L_3 , L_3S , L_4S , and L_4S . L_1 , L_2 , and L_3 represent lung involvement to the extent of stages 1, 2 and 3 respectively, according to the Turban-Gerhardt scale. *s* applied to these letters indicates that there is only slight systemic disturbance, whereas *S* signifies marked systemic disturbance; and the signs L_1S , L_2S and L_3S show that the systemic disturbance is excessive in relation to the lung involvement.

3. **INMAN'S CLASSIFICATION.**—This is based solely on the temperature in relation to exertion.

Stage 1. The patient is febrile when resting. *Stage 2.* The patient is resting afebrile, but ambulant febrile. *Stage 3.* The patient is ambulant afebrile. *Stage 4.* The patient is working afebrile.

The course taken by tuberculosis of the lung may lead to several terminations. These are: (1) permanent arrest, either by fibrosis prior to caseation, or if the latter has occurred, by calcification and fibrosis; (2) incomplete arrest, as shown by the persistence of tubercle bacilli in the sputum, or by slight degrees of pyrexia on over-exertion; (3) rapid extension, here the disease spreads, and the toxæmia is out of all proportion to the extent of the lesions; (4) death, this may result from the pulmonary lesion or from complications. The former may prove fatal as the result of progressive asthenia or cardiac failure, from asphyxia due to acute miliary tuberculosis or hæmoptysis, or in a small proportion of cases directly from loss of blood in repeated hæmoptysis. The complications that most often prove fatal are meningitis, enteritis, laryngitis leading to dysphagia and starvation, or

pneumothorax. Intercurrent diseases, such as pneumonia, influenza or diabetes, are occasionally the cause of death.

Diagnosis.—This is easy when definite signs are present in the lungs, and when tubercle bacilli are found in the sputum. On the other hand, the diagnosis of early cases may present one of the most difficult problems in clinical medicine. Tuberculosis may be suspected on account of symptoms, although the physical signs are indefinite. The conditions which most frequently lead to doubt are dyspepsia, neurasthenia, debility, visceroptosis and intestinal stasis, oral sepsis, tachycardia associated with early Graves's disease or heart disorders, affections of the nose and throat, and in children enlargement of the bronchial glands or acidosis. The history and symptoms are of great importance in these cases, and a careful examination should be made of each system. A test meal, opaque meal, or blood examination may be required before the correct diagnosis is established.

On the other hand, there may be definite signs of disease in the lungs which have to be differentiated from those produced by other conditions simulating tuberculosis. The cases included in this group embrace the majority of pulmonary lesions, especially chronic bronchitis, fibrosis, bronchiectasis, asthma, emphysema, apical catarrhs and collapse, pleurisy, new-growths and cysts. Diagnosis depends upon the history and course of the disease, together with a careful record of the physical signs in the chest, investigation of the sputum for infecting organisms, X-ray examination and in some cases the determination of the Wassermann reaction.

A condition of special difficulty is that of the variety of sarcoidosis known as Boeck's sarcoid. The lesion is a benign lymphogranuloma or reticulosis. It affects the lymph glands, lungs, bones especially those of the fingers, and the skin (see pp. 1478-1480). The parotid and lacrimal glands are sometimes involved and irido-cyclitis has been recorded in 10 per cent. of the cases. The intestines, spleen and liver may be affected. The chief diagnostic points are the character of the skin lesions and the chronicity and tendency to spontaneous arrest. Tubercle bacilli are not found and the Mantoux reaction is often negative. The radiological appearances in the lungs are those of a diffuse mottling—coarser than that of miliary tuberculosis. The hilar glands are often markedly enlarged.

When the diagnosis still remains doubtful the patient should be placed under observation, and a series of examinations carried out, the object of which is to determine whether or not active tuberculosis is present. The temperature should be observed with the patient in bed, a daily rise to 99° F. or a swing of 1°·5 to 2° below normal being suspicious. The sputum should be examined repeatedly for tubercle bacilli by the ordinary method, and if not found, the antiformin process should be carried out.

Before applying any tuberculin tests the blood may be examined serologically. The use of the Arneth or von Bonsdorff blood count in diagnosis has been referred to on page 1196. The complement-fixation test has proved disappointing, and in the present form does not afford reliable criteria of activity or quiescence.

The sedimentation test of the blood (stability reaction), i.e. the rate of settling of the erythrocytes in blood diluted with sodium citrate solution, is affected in this disease. In active cases the sedimentation rate is increased, but this reaction is not specific. It is also increased in other conditions such

as pregnancy, carcinoma, syphilis, rheumatism and acute infections. The test is therefore of little or no value in diagnosis, but it affords valuable indications of the degree of activity, and may assist in determining the form of treatment.

It has also been used as a guide to prognosis, since it is affirmed that arrest should not be considered as firmly established until the sedimentation rate has returned to normal. This may not occur until some time after the usually accepted clinical symptoms and signs of activity have disappeared.

THE TUBERCULIN TESTS.—1. *Cutaneous (the Pirquet reaction).*—Scarifications are made on the skin of the forearm through a drop of Koch's old tuberculin, human and bovine, and through a drop of saline as a control. A positive reaction is shown by the formation of a slightly raised, reddened papule at the site of the scarification through one or other varieties of tuberculin, whereas the control is not affected. Unfortunately, except in the first two years of life, this affords no indication of active disease, but only reveals the presence of previous infection with resulting tuberculin sensitiveness. A positive reaction is therefore given by the majority of adults.

2. *Mantoux's intradermal test.*—An injection of 0.1 c.c. of a 1 in 10,000 dilution of old tuberculin (0.01 mgrm.) is injected intradermally. If no reaction occurs, the injection is repeated in a week, with 0.1 c.c. of 1 in 1000 dilution (0.1 mgrm.). A positive reaction is shown by a red areola, with some cedema and occasional vesiculation.

A modification of this test consists in the use of Purified Protein Derivative (P.P.D.). This is supplied in tablets of two strengths, which must be dissolved in a buffered solution immediately before use. The advantage of this preparation is its constant potency.

3. *Vollmer's patch test.*—A strip of adhesive plaster is applied over the sternum, previously cleaned with ether. To this strip are attached three small squares of filter paper, the central one is a control of untreated paper, the other two have been saturated with undiluted old tuberculin and allowed to dry. The plaster is removed in 48 hours and 12 or 24 hours later a positive reaction is shown by redness, infiltration and sometimes by papules or vesicles.

4. *The subcutaneous test.*—The patient must be apyrexial, and must be kept in bed— $\frac{1}{10}$ mgrm. of old tuberculin is injected subcutaneously, and its effect determined. The reactions that may develop are—(a) local, an inflammatory swelling at the side of the injection; (b) focal, an increase of the signs observed at the seat of disease in the lungs, such as the temporary appearance of a few râles at one apex. This is the most important; (c) general, as judged by a rise of temperature and sense of malaise. The temperature should be charted 4-hourly after the injection, and a rise to over 99° F. indicates a positive reaction. If no reaction follows this initial dose, larger injections are given at intervals of 2 or 3 days, in this sequence: $\frac{1}{2}$, $\frac{1}{3}$, 1, 5 and even 10 mgrms.

This test has the drawback that it does not indicate activity of disease, and it has the additional disadvantage that it may cause a quiescent pulmonary focus to light up and spread, and so cause irreparable damage.

Finally, the X-rays afford most valuable assistance in the diagnosis of early cases with doubtful signs, and also help in the differential diagnosis of tuberculosis from other lung diseases with well-marked signs.

Prognosis.—A number of factors must be critically considered in the determination of the prognosis in pulmonary tuberculosis.

A marked family incidence generally suggests an unfavourable course, though this rule is not invariable.

Personal history.—Chronic alcoholism is serious, chiefly because the régime of treatment is then peculiarly irksome, while the digestion and powers of resistance are often impaired in alcoholics. Tuberculosis in syphilitics frequently assumes a fibrotic type, and its course may be beneficially influenced by anti-syphilitic treatment. The outlook is grave when tuberculosis is conjoined with diabetes though less so since the use of insulin. Congenital heart disease and pulmonary stenosis are unfavourable factors; but hypertrophy of the heart and mitral stenosis are said to be beneficial.

The prognosis is very grave in infants and young children; but slightly less serious up to the age of 20. Between 20 and 50 age has little influence; but in later years the outlook becomes progressively less favourable.

Apart from the effects of pregnancy and exposure, sex plays no important part.

Freedom from financial embarrassment improves the prognosis, inasmuch as advice can be sought early, and treatment carried through thoroughly.

Marriage often leads to a breakdown in arrested cases especially in women, and induces more rapid spread of active lesions.

Persistence in an unfavourable occupation, or return to it after completion of institutional treatment, affects the prognosis adversely.

Poor chest development and the "habitus phthisicus" are usually bad prognostic signs, although tuberculosis may run a rapid course even in patients with good physique.

Patients with resolute and persistent personality are more likely to persevere with treatment and to recover, than those of weaker moral fibre.

The prognosis is greatly affected by the type of the disease—acute miliary tuberculosis is usually rapidly fatal, whereas in acute caseous tuberculosis, although the prognosis is very grave, recovery may occur. In fibro-caseous tuberculosis the prognosis is most uncertain and difficult to forecast. Every factor must be carefully considered, and the response to treatment noted. The best outlook is in fibroid disease, which often undergoes complete and permanent arrest.

SYMPTOMS IN THEIR RELATION TO PROGNOSIS.—Persistent cough, by exhausting the patient and disturbing sleep, is often unfavourable.

The amount of sputum is usually dependent upon the type of disease and upon the presence of secondary infection, and may therefore be of value in prognosis.

The significance of tubercle bacilli in the sputum.—The figures obtained at the Midhurst Sanatorium, over a period of 8 years, in which the after-history of the patients was traced for the ensuing 6 years, show that the prognosis is best in "closed" cases; but that it is nearly as good in those cases in which the tubercle bacilli disappear from the sputum during the sanatorium treatment. Persistence of bacilli in the sputum is an unfavourable sign. The actual number of bacilli in the sputum and the presence of "beading" have no definite prognostic significance.

Cases commencing with hæmoptysis progress more satisfactorily than

those with other modes of onset, chiefly because they are diagnosed earlier. Hæmoptysis occurring later may exert an unfavourable influence, either indirectly by spreading the disease into previously healthy portions of the lungs, or actually by the loss of blood.

If dyspnoea is not due to attacks of bronchial spasm, it has usually an unfavourable significance.

The temperature affords a clue to the type and activity of the disease, and is thus a valuable aid to prognosis. Profuse and persistent night sweats, or marked anorexia, especially when occurring early in the disease, are grave signs. Tachycardia due to toxæmia, signs of cardiac failure, cedema and albuminuria are of bad omen. The blood pressure is thought by some to be a useful guide, systolic figures below 100 mm. Hg being unfavourable, whereas a rise of pressure may be associated with amelioration of the disease. In fibroid lesions the pressure may be raised throughout.

THE EXTENT OF PHYSICAL SIGNS.—The activity of the disease rather than its extent is the important factor in determining prognosis. The development of compensatory emphysema is of value only as an indication of fibrosis in the tuberculous portion of lung, and therefore of chronicity.

THE INFLUENCE OF COMPLICATIONS ON PROGNOSIS.—Generally speaking, the presence of complications increases the gravity of the disease. Involvement of the larynx is a serious complication, especially when accompanied by dysphagia; but complete recovery may take place if the pulmonary lesion is quiescent. In early cases spontaneous pneumothorax occasionally acts favourably; but when it develops in association with extensive tuberculosis, and especially if it progresses to pyo-pneumothorax, it is almost invariably fatal though, if the disease is unilateral, surgical measures may prove successful.

Pleural effusion often has a beneficial influence by diminishing the movements of a lung in which there is an early tuberculous focus.

Secondary catarrhal affections tend to increase the cough and expectoration, and may lead to further spread of the disease.

Meningitis is almost invariably fatal. Tuberculous peritonitis or enteritis is a very grave complication, but fistula-in-ano often occurs in chronic cases, and exerts no marked deleterious effect. Involvement of the genito-urinary system increases the severity of the disease, especially if the kidneys or bladder are affected. If the epididymis alone is involved the prognosis is not materially affected, as the lesion can be dealt with surgically, although the administration of a general anæsthetic may cause spread of the pulmonary disease. For this reason when operations are urgently needed on these patients, gas and oxygen, basal anæsthetics, local or spinal anæsthesia should be insisted on.

As shown by the figures obtained at the Midhurst Sanatorium, a fairly accurate guide to prognosis is afforded by observing the condition of the patient on admission to the sanatorium, and his response to treatment. Even in the most favourable cases, which are diagnosed in an early stage, and progress satisfactorily under treatment, the mortality rate is six times greater after discharge from the sanatorium than it is for the remainder of the population of England and Wales for the same age periods; whereas in the cases of advanced disease the mortality rate is thirty-eight times greater than for the average population. As the most critical time is during the two

or three years succeeding discharge from the sanatorium, the prognosis is largely affected by the conditions of life during this period.

The rate of sedimentation of the erythrocytes (see p. 1204) has proved to be a valuable aid to prognosis. A persistently rapid rate is unfavourable.

Treatment.—**PROPHYLACTIC.**—The prophylaxis of tuberculosis involves a consideration of public health questions dealing with the purity of the milk supply, the infection of meat, sanitation and housing, the early diagnosis of tuberculosis, the examination of contacts, and the segregation of "open" cases. Inoculation with the B.C.G. vaccine (attenuated living bovine bacilli) has not met with favour in this country. All these questions are considered in the general article on Tuberculosis.

CURATIVE.—This varies with the type and stage of the disease. In all acute or febrile cases treatment should be commenced at home or in a nursing home or hospital, where the patient can be under careful observation in bed. The various forms of treatment which may be considered are—(1) sanatorium treatment; (2) home or institutional treatment; (3) dietetic treatment and personal hygiene; (4) climatic treatment; (5) graduated rest, exercise and labour; (6) medicinal treatment; (7) specific measures; (8) operative treatment; (9) symptomatic treatment.

1. SANATORIUM TREATMENT.—This constitutes the best mode of treatment for early and for certain types of chronic disease; but is totally unsuited for acute febrile or very active cases. The advantages obtained are: (a) the patient learns the most suitable mode of life, and the methods employed to check the spread of the disease; (b) the housing is specially designed and the climatic conditions are good; (c) the dietary is abundant and adapted to the patient's needs; (d) there is constant skilled medical supervision, and the daily routine is adapted to the actual physical condition of the patient.

On arrival a newcomer is kept in bed for a few days in order that his resting temperature may be observed, and the necessary examinations carried out. If there is pyrexia, rest in bed must be enforced until the temperature falls to normal. If the temperature rises above 99° F. when the patient is up, return to bed is usually necessary. The routine of sanatorium treatment varies in different institutions, the most important divergence being whether or not a system of "graduated exercise" is employed. In nearly all an hour's recumbent rest is enforced before lunch and dinner.

After three months' stay it is usually possible to decide whether the patient is responding to treatment, and, if so, it should, if possible, be prolonged for at least another three months, or until the sputum is free from tubercle bacilli.

2. HOME AND INSTITUTIONAL TREATMENT.—Treatment at home, in nursing homes or in special hospitals, is essential in early cases with fever, and in cases in which it is necessary to establish the diagnosis. Home treatment is also usually necessary on return from sanatorium or climatic treatment, if arrest is incomplete. An endeavour should always be made to carry out the principles inculcated at the sanatorium, and the patient should be under regular medical supervision. Advanced cases are best looked after in special institutions.

3. DIETETIC TREATMENT AND PERSONAL HYGIENE.—It is desirable to graduate the diet in each case so that the patient is restored to the previous maximum weight; but, in order to accomplish this, the food should be slowly

increased and all ideas of enforced overfeeding discountenanced. A total calorie value of 3000 to 3500 is usually ample ; but, if the patient is performing heavy work, as much as 4000 may be necessary. Meat, fish, eggs and fats are usually well tolerated. It is not often necessary to give large quantities of milk when the patient is on a full dietary. If extra food is required, the protein may be increased by raw meat sandwiches. Additional carbohydrates with small daily doses of insulin if they are not well tolerated are often helpful.

In all cases in which there is expectoration the patient should be clean-shaven. Great care must be taken in the disposal of sputum to ensure that it does not become dry, and that flies do not have access to it. All patients who are up should carry special sputum flasks, while those who are in bed should have sputum cups suitably covered and containing disinfectant. The sputum should be burnt, or, if this is impossible, it should be emptied into the water-closet after disinfection with carbolic acid or other simple or cheap disinfectant.

Smoking is best avoided in cases of active disease or laryngeal tuberculosis, and in no instance should inhaling be allowed. Woollen under-garments should be worn ; but all excess of clothing is harmful. Sun-bathing and injudicious uncontrolled sun exposure are dangerous and often activate quiescent lesions. Patients should be strongly warned of this danger.

4. CLIMATIC TREATMENT.—This is undoubtedly of value in carefully selected cases. The climatic resorts fall into three groups—mountain, marine and inland.

The mountain resorts.—In Europe the most suitable places are found in Switzerland. Among these are St. Moritz (6090 feet), Arosa (6000 feet), Davos (5150 feet), Montana (5000 feet) and Leysin (4690 feet). In America the most celebrated resorts are in the Rocky Mountains at Colorado Springs (5000 feet) and Denver (5000 feet), or in the Andes or Adirondack Mountains. The advantages of high altitudes consist in the stillness, purity and rarefaction of the air, and the greater diathermancy of the atmosphere to the sun's rays. Metabolism and the general circulation are thereby increased.

High altitudes are suitable for early cases which are afebrile, or for quiescent cases of more advanced type. Contra-indications are recent hæmoptysis, active disease with fever, extensive fibrotic lesions and complications such as emphysema, asthma, cardio-vascular lesions or nephritis.

Marine and coast resorts.—Among the important coast resorts in the British Isles are Hastings, Bournemouth, the Isle of Wight, Torquay, Falmouth, Llandudno, Penmaenmawr, Scarborough, Mundesley and the various seaside towns in Thanet. Farther afield are the French and Italian Riviera, Madeira, the Canary Isles, Morocco, Algiers and Egypt. The climate tends to be warm, moist and equable, and the amount of ozone is probably increased. These resorts are especially suitable for cases of more advanced and active disease, and for those complicated by hæmoptysis, bronchitis, emphysema and laryngitis. Residence by the sea actually at sea-level is undesirable.

Inland resorts.—These are to be found on the English and Scottish moorlands. The climate of California, the South African veldt, and parts of Australia and New Zealand are admirably suited to this disease, especially for arrested or early uncomplicated cases ; but the laws against the admission of tuberculous patients are strictly enforced at all of them.

Sea voyages.—These are contra-indicated for all except completely arrested

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cases owing to the lack of fresh air in cabins, the possibility of sea-sickness, and the difficulty of obtaining suitable treatment if the disease advances.

5. **GRADUATED REST AND EXERCISE.**—Treatment in bed is necessary so long as there is fever, and if the raised temperature is persistent “absolute rest” should be enforced. This consists in keeping the patient recumbent in bed, sufficiently well covered to prevent any muscular contraction from chill, while feeding and washing are attended to by the nurse, and the use of the bed-pan and slipper for evacuations is insisted upon. When the temperature becomes normal the patient is allowed up for varying periods, commencing with 1 hour daily, and increasing slowly to 6 or 8. If still apyrexial, walking exercise of 1 or 2 miles or more daily can be allowed.

The system of “graduated exercise” which Paterson instituted at the Frinley Sanatorium has proved of great value. There are six grades, the first and lightest consisting of walking up a slope carrying a light weight such as a basket of earth, while the sixth and heaviest involves hard manual labour with a pickaxe or shovel for 6 hours daily. This system is based on the principle that muscular exercise leads to the discharge of tubercle toxins from the pulmonary focus, and by liberating these in gradually increasing doses, a condition of active immunity is induced. A careful watch must be kept during this controlled process of auto-inoculation to prevent excessive doses of toxin being discharged, which are early indicated by rise of temperature and of pulse-rate, headache, increased cough and expectoration, lassitude and malaise. If such occur, the patient should be put back to bed for a few days, and when the condition has subsided the graduated exercise may be resumed at the grade which induced the over-inoculation or that immediately below it.

6. **MEDICINAL TREATMENT.**—No specific drug has yet been discovered for the treatment of tuberculosis. Amongst the medicines in most general use are :

(a) *Cod-liver oil.*—This may be administered by the mouth in doses up to 2 ounces daily. The cod-liver oil may be of value either on account of its fat-soluble A and D vitamins content, or, as suggested by Rogers, it may assist by dissolving the capsules of the tubercle bacilli and so facilitating their disintegration. Halibut liver oil is now sometimes used instead.

(b) *Creosote.*—This may be given in doses of min. 2 to 3 three times a day after food, either in combination with cod-liver oil, or in capsules. It should be discontinued if gastric disturbance or hæmoptysis ensue.

(c) *Hypophosphites.*—These are not so generally used as formerly and, beyond their “tonic” effect upon the nervous system, have no demonstrable influence upon the pulmonary lesion.

(d) “*Nascent iodine*.”—With the idea of liberating free iodine in the tissues, potassium iodide grs. 30 is administered after breakfast in half a pint of water, and throughout the day 3 to 5 ounces of chlorine water are consumed with lemonade. This treatment is of value in certain chronic fibroid cases, but it often produces no appreciable results, and may cause dyspepsia.

(e) *Arsenic.*—Liquor arsenicalis min. 2 to 3 by mouth, or sodium cacodylate gr. $\frac{1}{4}$ to $\frac{1}{2}$ subcutaneously, are of value in some cases associated with anæmia. Neoarsphenamine, administered intravenously, is useful in chronic cases complicated by syphilis.

(f) *Inhalations*.—Disinfectant drugs when inhaled often check cough lessen expectoration and improve the general condition of the patient. Lees's inhalation is of value. It consists of creosote, parts 2; acid. carbol., 2; liq. iodi mitis, 1; sp. ætheris, 1; and sp. chlorof., 2. About 6 drops an hour are placed upon a Burney-Yeo mask, which should be worn almost continuously throughout the day. A modification now more frequently employed is: menthol, 4; olei cinnamomi, 3; olei limonis, 4; creosote, 10; olei pini, 10; sp. chlorof., 10.

(g) Calcium is often given by the mouth in the form of colloidal calcium in doses of 60 minims three times a day; or intramuscularly, as calcium gluconate, 5 c.c. of a 10 per cent. solution once or twice a week. Parathyroid extract is sometimes administered at the same time.

(h) There has been a revival of interest in preparations of gold in the treatment of this disease, notably by Møllgaard, who uses sodium aurothio-sulphate, to which the name of sancocrysin has been applied. It is now administered in smaller doses than when it was first introduced. The initial dose is usually 0.01 g. dissolved in sterile saline solution and injected intravenously. The second dose is given 5 days later, and is as a rule 0.025 g. If no reaction occur, the dose is increased to 0.1 g., and usually later to 0.25 g., the intervals being extended to a week. The total amount given in a course is usually 3 g., or sometimes up to 5. The course may have to be interrupted owing to reactions, chiefly fever, albuminuria, stomatitis, diarrhoea, peripheral neuritis and skin manifestations.

7. SPECIFIC MEASURES.—(a) *Active immunisation*.—Tuberculin and tubercle-vaccines.

The tuberculin treatment has not fulfilled the high hopes held out on its introduction by Koch. There are now numerous forms of tuberculin available, indicated by certain letters, and falling into three groups. (1) Those containing the exo-toxins only. These include Koch's old tuberculin T., O.T., and T.O.A., Denys' bouillon filtré (B.F.) and albumose-free tuberculin, T.A.F. (2) Those containing the endo-toxins chiefly, such as Koch's "new" tuberculin, T.R. (3) Mixtures of endo- and exo-toxins, the most important of which are Koch's bacillary emulsion, B.E., the sensitised bacillary emulsion, S.B.E. and Béranek's tuberculin, T.Bk.

The Therapeutic Substances Act, 1925, restricts the term "tuberculin" to the first of these groups, and recommends the name "tubercle vaccine" for any substance obtained directly from the bacterial bodies.

Tuberculins and tubercle-vaccines may be prepared from human or bovine bacilli; if from the latter the letter P. (perlsucht) placed before the letters indicating the variety of tuberculin, signifies its origin, e.g. P.T. In administration, some aim at producing reactions and establishing tuberculin tolerance by giving large doses at fairly frequent intervals; others believe in minute doses at longer periods, the chief object being to avoid the production of any reaction. The actual doses are either measured in milligrammes of dried tubercle bacilli, or in cubic centimetres or cubic millimetres of the fluid tuberculin. The usual method is to measure the doses in cubic centimetres or fractions thereof, and to make the necessary dilutions in a series of bottles. Smaller initial doses should be used with a strong tuberculin such as the B.E. than with the weaker ones such as the T. or T.A.F. Thus, if adopting minute doses, 800.000 mgrm. of B.E. would be a suitable initial dose, then for T.R.

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$\frac{1}{100,000}$ mgrm. would be used and $\frac{1}{1000}$ mgrm. of T., B.F., or T.A.F. As the different tuberculins are supplied by the makers in varying strengths, 0.001 c.mm. of the original fluid is equivalent to the above doses.

Those who believe in minute doses commence with 0.001 c.mm. and work up to 0.1 c.c., whereas others commence with 0.5 to 1 c.mm. and increase to 1 c.c. The tuberculin should be injected subcutaneously, and a careful observation kept for local, focal and general reactions. Tuberculin, if used injudiciously, can be productive of harm. At Midhurst Sanatorium it was tried for some years, and no better ultimate results were obtained than in the "non-tuberculin" years. It seems wise, therefore, to use tuberculin only in very carefully selected cases, thus T.R. does at times appear beneficial in chronic tuberculosis, promoting the formation of fibrous tissue and leading to the disappearance of tubercle bacilli from the sputum.

Various attempts have been made to remove as far as possible the fatty and waxy constituents from the tubercle bacilli before preparing a vaccine from it. Much and his collaborators have employed partial antigens or partigens derived from tubercle bacilli, singly or in varying combinations. Some good results are on record from both of these methods, but on the whole their use has so far been disappointing.

Grasset has recently recorded success with a preparation described as "tubercle endotoxoid," which he claims is freed from the toxic factor in ordinary tuberculins.

(b) *Passive immunisation*.—The various serums such as those prepared by Marmorek and Maragliano have not proved successful, and this applies to the "contra-toxin" of Mehnarto and the "I.K." (immune bodies) of Spengler.

8. OPERATIVE TREATMENT.—(a) *Artificial pneumothorax*.—This mode of treatment is now becoming more generally adopted in selected cases. It is increasingly employed in cases of early disease, where there are indications of incipient softening or of recent cavitation. In early cases without softening it is not as a rule used, unless there are indications of rapid spread or of pleural involvement when it is wise to start before adhesions have formed. In extensive bilateral disease it may be dangerous. If old and dense pleural adhesions are present, it is impracticable. If there is much emphysema or cardiac embarrassment, it involves risk. It is also of value in certain cases of repeated severe hæmoptysis. Tuberculous laryngitis or enteritis are not contra-indications, providing that other conditions are suitable. Sterile air is introduced into the pleural cavity, and the lung allowed to collapse. The method of induction of artificial pneumothorax is as follows: A preliminary subcutaneous injection of $\frac{1}{2}$ grain of papaveretum (omnopon) is given half an hour before the start. The patient lies on the sound side with the head low and supported on a pillow. A second pillow is placed under the chest to expand the intercostal spaces. The skin and the tissues down to the pleura are anaesthetised with 2 per cent. procaine hydrochloride solution after the application of iodine. The site usually chosen is in the sixth intercostal space in the mid-axillary region. A special pneumothorax needle, attached by a rubber tube to the pneumothorax apparatus, which is carefully examined to see that it is in proper working order, is then pushed through the intercostal space until the pleural cavity is reached. The apparatus is then adjusted so that the intrapleural pressure can be observed. No air should be introduced until the manometer shows a normal negative

pressure range with inspiration, of 5 to 10 or more cm. of water. This is the test of entry into the pleural space, and when this is established 200 to 300 c.c. of sterile air may be allowed to enter the pleural cavity. The final pressures are then recorded and the needle is withdrawn. A refill is given next day and another after two more days, the quantities of air introduced being determined by the final pressures, which should be kept slightly negative. Subsequent refills are gradually spaced out to a week, then ten days and later to two, three and four weeks' intervals. The usual custom now is to maintain the collapse for three years or even longer. If the condition of the patient is satisfactory, re-expansion may then be permitted cautiously. It should be remembered that after expansion pleural adhesion almost invariably occurs and the treatment by artificial pneumothorax cannot be repeated. In some cases of bilateral disease, which is active but not very extensive in either lung, a cautious use of bilateral artificial pneumothorax has proved practicable and helpful, but very great care is necessary in adjusting the pressures.

There are certain dangers in the procedure. These are now rare, and they can usually be prevented by careful attention to the technique. Death has occurred from pleural shock when the needle has reached the pleura and before any air has been introduced. Adequate anæsthesia of the pleura is the only known method of eliminating or minimising this risk. If the lung is adherent to the chest-wall, owing to pleural adhesions, or if the needle is pushed in too far, it may be inserted into the lung or into a pulmonary cavity; the manometer will then show a swing above and below the zero line instead of entirely below it. Under these circumstances, no air should be allowed to enter. The needle may be inserted into a blood vessel. In this case the manometer pressure will rise above zero, and blood may appear in the glass section inserted in the rubber tube leading from the manometer to the needle. The needle should be withdrawn immediately, lest air should enter the vessel.

If the pleura is found to be adherent at the site of the first puncture, another attempt may be made elsewhere, *e.g.* just below the inferior angle of the scapula. This spot may be selected for the initial puncture in left-sided cases where there is marked cardiac displacement. In cases in which localised band or cord adhesions prevent adequate collapse, it is often possible to cut them by electrocautery or diathermy through an operating thoracoscope, thus ensuring completely effective collapse. This is called internal pneumolysis.

(b) *Oleothonax*.—Sterilised olive oil or liquid paraffin with a varying percentage of gomenol is sometimes used to maintain the collapse started by artificial pneumothorax. Paraffin is used when the mediastinum is unduly mobile, since it tends to thicken the pleura. The oil or paraffin is introduced by means of a Dieulafoy syringe, and air is withdrawn at the same time by a reversed artificial pneumothorax apparatus. Whichever is used, it is important to test the sensitiveness of the pleura by small injections of 2 to 5 c.c. to start with. In later injections the amount introduced may be gradually increased to 200 c.c. or more. Oleothorax is now for the most part restricted to cases of therapeutic pneumothorax tending to obliterate from spreading adhesion, and those with mobile mediastinum.

(c) *Evulsion or crushing of the phrenic nerve*.—This is now often performed

in order to produce basal collapse, but it helps to produce relaxation at the apex and may aid in the contraction and closure of a cavity. Division alone is not sufficient; it is desirable to remove as long a stretch of the nerve as possible. In recent years temporary paralysis has been induced by crushing the nerve, the effects lasting about 6 months. This is often carried out instead of evulsion, since it does not prejudice further surgical treatment subsequently.

(d) *Thoracoplasty*.—If owing to adhesions it is impossible to collapse the lung temporarily by an artificial pneumothorax, permanent collapse by thoracoplasty may be considered. In the Sauerbruch operation, the posterior 3 or 4 inches of the ribs from the first downwards are removed, even to the tenth or eleventh, the lower level being determined by the extent of the compression of the lung it is desired to produce and therefore by the amount and situation of the diseased area. This operation is now performed in two or three stages, and generally under local anaesthesia. Good lateral collapse is usually obtained by this operation, but antero-posterior compression is less complete. Semb's operation is more satisfactory and is now more frequently employed. The first, second and third ribs are completely removed and the fascial supports of the apex of the lung are divided, which is thus allowed to sink down, producing a concentric collapse. The second and if necessary a third stage are similar to those in the Sauerbruch operation.

(e) *Apicolysis (extrapleural pneumolysis)*.—Successful local collapse can sometimes be attained by introducing some extraneous material like paraffin between the chest wall and the parietal pleura over a local area of disease, or a cavity which is not too near the pleura.

(f) *Extrapleural pneumothorax*.—In some cases where artificial pneumothorax fails owing to extensive apical adhesions, an extrapleural pneumothorax may be induced. A portion of the fourth rib is removed near the spine and the parietal pleura with the adherent lung is stripped away from the chest wall through the endothoracic fascia. The separation is carried down to the hilum on the mediastinal aspect, to the eighth rib posteriorly and to the fourth costal cartilage in front. The wound is then carefully closed and sutured. The extrapleural space thus produced is maintained by repeated refills of air, at first very frequent, subsequently at longer intervals. After about a month high positive pressures up to +18 and +24 must be maintained, otherwise the space obliterates. This procedure, though sometimes successful, seems less generally satisfactory than apical thoracoplasty. On the other hand it involves less shock than the operation of thoracoplasty, and is therefore practicable in some cases where thoracoplasty cannot be considered. The existence of an intrapleural pneumothorax below where it is necessary to keep the lower lobe under control does not contra-indicate it.

9. SYMPTOMATIC TREATMENT.—When cough is ineffective it may be relieved by a sedative lozenge or linctus containing diamorphine or codeine, or by the well-known liquorice lozenge. If there is difficulty in bringing up the sputum, a simple saline mixture is of value, such as sodii bicarb. grs. 10, sodii chlorid. grs. 3, sp. chlorof. min. 10, and aq. anisi ad fl. oz. 1.

Pain in the chest is usually alleviated by local application of pigmentum iodi, liniments, mustard leaves or other counter-irritants.

Night sweats.—The windows should be widely opened at night. A pill containing zinc. oxid. grs. 3 and ext. belladonn. succ. gr. $\frac{1}{4}$ is often of value.

Picrotoxin, agaricin and strychnine have also been used. A rush mattress as used in the tropics has been recommended.

Fever.—Rest in bed up to the extent of “absolute rest” is the best means of lowering the temperature. Antipyretic drugs have no effect upon the course of the disease, but may alleviate malaise. Amongst these may be mentioned aspirin and cryogenin.

Slight hæmoptysis, in which the sputum is only streaked, calls for no special treatment. Moderate hæmoptysis, with expectoration of 3 or 4 ounces of blood, requires more active measures. The patient should be put to bed, a saline aperient administered, and if there is anxiety or alarm a sedative drug should be given, such as heroin or morphine. In profuse or persistent hæmoptysis the patient should be confined strictly to bed, and if it is known from which side the bleeding has occurred, it is best to lie on this side. If the cough is troublesome, or if the patient is alarmed, morphine gr. $\frac{1}{4}$ to $\frac{1}{2}$, or diamorphine, hydrochloride gr. $\frac{1}{2\pi}$ to $\frac{1}{\pi}$ should be injected subcutaneously. The food is best given cold, and may be iced; no alcohol must be taken. A course of calcium lactate grs. 10, t.d.s., may be commenced; but its action is somewhat uncertain. If the bleeding persists, various other remedies should be tried, these include the inhalation of amyl nitrite, or the injection of hæmoplastin, coagulen ciba, horse serum, or emetine hydrochloride subcutaneously. Congo red, given intravenously, has proved of value in some cases. Ergot and adrenaline are both contra-indicated. If the hæmorrhage is still unchecked, or is frequently repeated, the advisability of establishing an artificial pneumothorax must be considered.

Gastro-intestinal symptoms.—Anorexia or dyspepsia can often be relieved by changes in diet, or by the administration of suitable drugs. Alkalis and gentian are especially valuable, and when hypochlorhydria is present, dilute hydrochloric acid (min. 10–30) should be given well diluted after meals. Digestive ferments, such as taka-diastase or papain, may be required at times. All tendency to constipation should be checked by laxatives, and if diarrhœa develops, avoidance of diet leaving bulky or irritating residues should first be tried, before administering drugs containing lead, opium, bismuth or tannic acid.

Insomnia is often a troublesome symptom, and every endeavour should be made to obtain a good night's rest by administration of mild hypnotics, and by relieving distressing cough and pain.

The treatment of the complications of pulmonary tuberculosis is described under their respective headings. The after-care of patients discharged from sanatoria is an important subject, to which considerable attention is being devoted, and involves a consideration of the advisability of establishing training centres or industrial colonies for consumptives. These are proving of very great value.

THE PULMONARY MYCOSES (PNEUMONOMYCOSES)

A number of fungi produce pulmonary lesions. Considerable confusion exists in regard to their nomenclatures, and at the present time it is difficult to give accurate accounts of them. The pulmonary mycoses have one feature in common, in that they produce chronic pulmonary lesions

practically indistinguishable clinically from those of the chronic forms of pulmonary tuberculosis.

Among the varieties of mycotic infection at present separated clinically may be mentioned—(1) Actinomycosis and other streptothrix infections; (2) Aspergillosis; (3) Blastomycosis; (4) Sporotrichosis; (5) Moniliasis; (6) Mucormycosis.

STREPTOTRICHOSIS (ACTINOMYCOSIS)

Ætiology.—The general characters of the streptothrix group of organisms are described in the section on Actinomycosis. It is now recognised that more than one of these may be pathogenic for man, and some authors give separate descriptions of the forms due to the various streptothrix organisms. At the present time there seems little advantage in so doing, since the important point in regard to treatment is to recognise that the morbid process is due to some form of streptothrix infection, the identification of the variety being a pathological refinement. The manner of infection is at present obscure. The organism is now believed to be present not infrequently in the alimentary tract, but the conditions favouring its invasion of the tissues are not known. A large proportion of cases show the first lesions in the head and neck regions, but primary pulmonary cases occur, and are probably more frequent than is generally recognised.

Pathology.—The streptothrix group of organisms produces an inflammatory reaction which leads to the formation of granulomatous tissue. This, like the granuloma of tubercle, is very liable to undergo secondary changes producing small areas of pus formation or leading to fibrosis. Unlike tuberculosis, however, streptotrichosis tends frequently to transgress anatomical limitations and spreads by contiguity. In the primary pulmonary cases the distribution of the lesions is at first very similar to that of tuberculosis, and the disease may extend in an identical manner. In the forms due to spread from other organs such as the liver, the base of the lung may be first involved, while in cases extending down from the neck the path of the infection is apparent.

Owing to the tendency of the lesions to spread by contiguity, subcutaneous abscesses may form and simulate caries of the ribs. Pleural adhesion is the rule, but occasionally empyema results. When a subcutaneous abscess ruptures or is opened, the characteristic "sulphur granules" may be found, although this is not invariable. The skin around the sinuses which result is often puckered in a somewhat characteristic fashion.

Symptoms.—These are in general identical with those of the chronic forms of pulmonary tuberculosis, such as cough, expectoration, which may be offensive, dyspnoea, fever and night sweats. The occurrence of local abscesses under the skin or the presence of the organism elsewhere may give rise to special features; but these are late developments in primary pulmonary cases.

Complications and Sequelæ.—These are usually due to the other localisations of the organism; but, in addition, empyema and bronchiectasis may be mentioned.

Course.—This is progressive, and leads eventually to asthenia, emaciation and death.

Diagnosis.—This can only be established by the discovery and identification of the organism in the sputum and the discharge. The characteristic "sulphur grains" are not invariably present, and may escape notice unless looked for carefully. In any obscure case of pulmonary disease in which tubercle bacilli are not found after repeated search, the possibility of streptotrichosis should be considered, and direct films should be specially examined.

Prognosis.—This is serious, although some cases respond well to treatment.

Treatment.—Large doses of potassium iodide should be administered, commencing with 5 or 10 grains three times a day, and increasing until the dose reaches a drachm or even more thrice daily. In addition, collosol iodine (Crookes) may be given intravenously in doses of 5 c.c. at least once a week. If the organism grows well in culture, a vaccine may be prepared and employed cautiously, especially if the iodides do not act satisfactorily. A stock vaccine may be helpful in other cases. Surgical treatment of local abscesses or of empyema may be required. External application of a radium pack is sometimes useful.

PULMONARY ASPERGILLOSIS

Ætiology.—Infection of the bronchi and lungs sometimes occurs by the *Aspergillus fumigatus*, more rarely by the *A. nidulans*. The disease has been most frequently observed in France. It occurs among pigeon breeders and hair sorters and combers. The former acquire the disease from the process of artificial feeding, from grains in the mouth to the beak of the bird; the latter from the use of rye flour in cleaning the hair. Millers and farm labourers have also been the subjects of the disease.

Pathology.—The fungus induces nodular formations in the lung tissue somewhat resembling aggregated tubercles. Bronchitis, patchy lobular consolidation and fibrosis result. Emphysema, bronchiectasis and cavity formation may follow. A secondary aspergillosis may occur in chronic cases of bronchitis or lung disease, but is of little clinical importance.

Symptoms.—Primary aspergillosis produces symptoms similar to those of bronchitis, broncho-pneumonia or pulmonary tuberculosis, according to the localisation and degree of the lesions. The sputum may be blood-stained, or definite hæmoptysis may occur. There is generally wasting with irregular fever.

Course.—Acute broncho-pneumonic forms may be fatal in a few weeks or months. The chronic lesions may extend to years, and arrest with fibrosis is not uncommon.

Diagnosis.—The condition has to be differentiated from pulmonary tuberculosis, and from other varieties of pneumonomycosis. This depends upon the recognition of the fungus by microscopical and cultural examination of the sputum.

Treatment.—This consists in avoiding further infection, and giving large doses of potassium iodide as in streptotrichosis. Open-air measures and general tonic treatment are also to be recommended.

OTHER MYCOTIC INFECTIONS

Fungi of the genera *Blastomyces* (*Oidium*, *Coccidioides*) and *Sporotrichum* are well known to produce cutaneous affections simulating chronic gummatous or tuberculous lesions. They may also give rise to pulmonary disease producing symptoms like those of tuberculosis.

Castellani has described various broncho-pulmonary conditions due to species of the genus *Monilia*, including the "tea-tasters' cough" and "tea-factory cough." Another fungus, *Mucor mucedo*, has been found in the sputum, and is regarded as pathogenic to man.

All these moulds produce bronchitic symptoms and mild infections, while more severe forms simulate pulmonary tuberculosis. The diagnosis in each case depends upon the recognition of the fungus, and the treatment recommended is large doses of potassium iodide.

SYPHILIS OF THE LUNGS

Ætiology.—Clinically recognisable pulmonary syphilis is a rarity; but syphilitic lesions occur in the lungs in both the congenital and acquired forms of the disease.

Pathology.—Even post mortem it is often difficult to establish the syphilitic nature of the pulmonary lesions found in cases of syphilis, owing to the fact that they tend to the formation of scars presenting no characteristic features.

Congenital syphilis.—The essential changes are—(1) Round-celled infiltration with eventual fibrosis, starting round the bronchi and spreading to the inter-alveolar framework; (2) periarteritis of the smaller arteries; and (3) desquamation and degeneration of the epithelium of the alveoli and bronchi. Gummata may be present, but are rare. Spirochaetes can be demonstrated in the lesions by Levaditi's method. The microscopic appearances comprise the "white pneumonia" of Virchow and an interstitial pneumonia, which is commoner, although both conditions are frequently associated. White pneumonia is found in premature or still-born infants, and in those dying soon after birth. The condition may be widespread or localised. The affected areas are firm, consolidated, smooth and greyish-white in colour. There are no interstitial changes, and the consolidation is due to the filling of the alveoli with desquamated, degenerating epithelial cells.

In the commoner interstitial form the lung is firmer, harder and darker grey in colour, and the connective tissue is mainly involved. To this condition the term "pancreatisation of the lung" has been applied by Rogers.

Acquired syphilis.—Syphilitic lesions of the bronchi have already been described in the section on diseases of the bronchi. Gummata may occur in or around the intra-pulmonary bronchi or in the lung tissue. They may be single or multiple, and vary in size from that of miliary granules to a hen's egg. They are said to be more common in the deeper parts of the lung near the roots and in the lower lobe. They undergo changes similar to those occurring in gummata elsewhere, but tend more to fibrosis and contraction than to softening. Owing to these secondary changes, the following

conditions may result: broncho-pneumonic processes, widespread fibrosis and contraction with pleural adhesion, bronchiectasis and occasionally excavation.

Symptoms.—Small gummata may be latent and give rise to no symptoms or signs. When fibrosis occurs, they are similar to those of pulmonary fibrosis from other causes. It is generally recognised that in rare cases a destructive process occurs, formerly called "syphilitic phthisis," and almost exactly similar in its clinical manifestations to those of caseous or fibro-caseous tuberculosis.

Complications and Sequelæ.—Syphilitic lesions in the larynx, trachea or bronchi may complicate the course. Bronchiectasis has already been mentioned, and tuberculosis may occur as a complication.

Diagnosis.—This is often difficult and sometimes inconclusive. Obscure pulmonary signs in a syphilitic subject should arouse suspicion. The Wassermann reaction should be determined, and other indications of syphilis looked for in all fibrosing and destructive lung conditions when no tubercle bacilli are found in the sputum. The difficulty of diagnosis is increased by the association of syphilis and tuberculosis mentioned above.

Course and Prognosis.—Where the lesions are localised and can be recognised early, the course is favourable if anti-syphilitic treatment is applied. Where fibrotic changes occur, leading to bronchiectasis, the course is less favourable, and in the destructive form it is serious. An inter-current tuberculous infection increases the gravity of pulmonary syphilis.

Treatment.—When a diagnosis of pulmonary syphilis has been established, vigorous anti-syphilitic treatment should be carried out. Its beneficial effect is undoubtedly promoted by open-air treatment. In cases where tuberculosis coexists with syphilis, anti-syphilitic treatment is strongly recommended, especially by French physicians.

NEW-GROWTHS IN THE LUNGS

Both simple and malignant tumours may occur in the lungs, the latter being the more common.

Ætiology.—Malignant tumours occur more frequently in the male sex in the ratio of five to one; carcinoma is rare before the age of 40, but sarcoma may develop in earlier years. Simple tumours may arise at any age, but are found chiefly in adult life. The exciting cause is unknown. In some cases of malignant growth there is a history of thoracic trauma or disease.

Pathology.—Simple tumours found in the lungs usually arise in the bronchial mucous glands or in the bronchi. They include adenoma, fibroma, lipoma and chondroma (see p. 1141).

Malignant tumours may be primary or secondary. The primary growths are carcinoma, sarcoma or endothelioma. Carcinoma arises in the bronchi, usually as a columnar-celled growth. A variety of bronchial new-growth formerly regarded as a lympho-sarcoma is now called an oat-celled tumour. It is probably derived from the basal cells of the bronchial mucous membrane. Squamous-celled growths of the bronchi are rare and are now said to be derived from basal cells. Round-celled and spindle-celled sarcomata growing from the pulmonary connective tissue are met with, while endotheliomata are

usually derived from the endothelium of blood vessels and lymphatics, or from the pleura. A primary carcinoma of the breast, œsophagus or mediastinum may directly invade the lungs. Secondary carcinoma may have its primary focus in the breast, stomach, intestines, liver, pancreas or prostate, whereas a secondary sarcoma most often results from metastasis of a primary bony growth. Chorion-epithelioma and hypernephroma also give rise to secondary deposits in the lungs.

Primary malignant tumours are unilateral; but secondary growths are often multiple and diffuse. Dissemination in the lungs may occur by spread along the bronchi or vessels, and a condition of miliary carcinomatosis is at times produced. The pleura is often affected by direct extension. Infiltration of, or pressure upon, the mediastinal structures frequently occurs.

Symptoms.—Simple tumours except adenomata are pathological curiosities and, as a rule, only produce symptoms when they cause obstruction of a bronchus or press on mediastinal structures (see pp. 1046 and 1280).

The early symptoms of malignant growths are slight, and consist of malaise with, perhaps, cough and expectoration. Later, when the growth becomes more extensive and exerts pressure on, or involves the larger bronchi, mediastinum or pleura, they are more noticeable. Pain, dyspnoea and loss of weight with cachexia usually develop, and the cough and expectoration are more marked. The latter is often of the typical "currant jelly" or "prune juice" appearance due to altered blood. Microscopically, groups of large fatty cells, or irregular epithelial cells may be seen. Malignant cells may be found in 60 per cent. of cases by Dudgeon's wet method. There are usually no definite physical signs until the tumour causes pressure upon the bronchi, mediastinum or deep thoracic veins or nerves. The chest-wall may bulge locally, owing to the presence of a growth near the surface, or it may be retracted if a main bronchus is obstructed. An actual subcutaneous swelling caused by the tumour eroding through the chest-wall may be visible. Enlarged veins often run across the chest, and one or other arm may be swollen or œdematous if there is mediastinal obstruction. Vocal fremitus is often unaffected; but is increased when the growth is near the surface, and diminished if pleural effusion has occurred. The percussion note over a moderate-sized tumour is impaired and may be extremely dull; more often the dullness is due to collapse of the lung. The breath-sounds vary with the size and position of the growth, and with the displacement or pressure effects produced. They may be weak, or loud and stridorous. The stridor is usually unilateral. Adventitious sounds depend upon the presence of complications such as bronchitis. Some degree of fever often occurs. The supra-clavicular and axillary glands are not infrequently enlarged, and evidence of malignant disease may be present in other parts of the body such as the abdomen.

One special variety of apical carcinoma is the superior pulmonary sulcus or Pancoast tumour, which gives rise to a somewhat characteristic or suggestive clinical picture. The chief symptoms are pain in the shoulder, inner side of the arm and forearm together with weakness and wasting of the small muscles of the hand. Paralysis of the cervical sympathetic on the same side develops. There is usually localised dullness at the extreme apex. Radiological investigation reveals a sharply defined apical shadow with destruction

of the posterior part of the first three ribs and sometimes localised vertebral erosion. Pancoast suggests that these tumours may arise from remnants of the fifth branchial cleft.

Complications and Sequelæ.—Bronchitis is nearly always present in some degree. Pulmonary collapse, fibrosis, bronchiectasis, emphysema, gangrene, hæmoptysis, pleural effusion, abscess and empyema are sometimes observed. The effusion is frequently bloodstained. In cases of primary malignant disease of the lungs, secondary deposits may occur in other parts of the body such as glands, brain, suprarenals, heart and bones.

Course.—This is progressive, the patient gradually losing strength and dying from cachexia or some intercurrent affection.

Diagnosis.—This is difficult in early cases, and not easy in some advanced ones. It not infrequently happens that metastases, especially in brain or bone, afford the earliest manifestations to be recognised. Difficulties may arise in connection with pulmonary tuberculosis, fibrosis and gumma of lung, aneurysm, pericardial and pleural effusion and enlargement of the mediastinal glands due to Hodgkin's disease or tuberculosis. The whole body should be searched for evidence of malignant disease elsewhere. The sputum should be examined repeatedly for tubercle bacilli and for cellular elements, and an X-ray examination made of the chest. By the stereoscopic method excellent evidence of pulmonary neoplasms is often obtainable. Lipiodol injection and X-ray examination or tomography may demonstrate the obstruction of a bronchus by the growth which often presents a tapering or "rat-tail" appearance. Bronchoscopy may also serve to establish the diagnosis especially in bronchial carcinoma. Temporary artificial pneumothorax may be helpful in diagnosis, particularly in differentiating simple tumours in the periphery of the lung, growths in the mediastinum and in the chest-wall.

The Pancoast tumour may give rise to special difficulty. It has to be differentiated from syringomyelia, cervical rib, apical pulmonary tuberculosis and secondary sarcoma.

Prognosis.—Apart from those cases in which early recognition may in suitable conditions render lobectomy, with removal of the growth, possible, this is hopeless, death occurring in a few weeks, or being delayed for two or three years.

Treatment.—Simple tumours are often capable of complete removal with gratifying success.

In malignant growths lobectomy or dissection pneumonectomy with complete removal of the growth is only practicable for cases recognised early in which there are no secondary deposits.

Radon seeds are sometimes used; when practicable they are inserted into the growth through a bronchoscope. In other cases they may be introduced directly into the growth by thoracotomy. Treatment by deep X-ray application may be useful by diminishing local pressure and relieving symptoms chiefly in sarcoma or oat-celled carcinoma. Cure by these methods is rare.

In cases unsuitable for lobectomy and radiation treatment this can only be palliative and symptomatic. Useless cough should be checked by sedative lozenges or a linctus. Dyspnoea due to pleural effusion may be relieved by tapping with or without air replacement; but the fluid often reaccumulates

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rapidly. Pain should be relieved by analgesic drugs, and in the later stages those containing opium or its alkaloids may be required.

PARAGONIMIASIS

Synonyms.—Pulmonary Distomatosis; Lung Fluke Disease; Endemic Hæmoptysis; Parasitic Hæmoptysis.

Ætiology.—(See p. 306).

Pathology.—The flukes settle down in the lungs and form burrows. These burrows may coalesce and give rise to cystic swellings, varying from $\frac{1}{2}$ to $1\frac{1}{2}$ inches in diameter. These in turn develop fibrous sheaths and may give rise to abscess formation or pleurisy. The adult fluke is hermaphrodite, and lays numerous eggs which measure $100 \times 70 \mu$. These are coughed up in the sputum, and are easily recognisable owing to their large size. The adult parasites are also occasionally found in the brain, liver, lymph glands and peritoneal cavity.

Symptoms and Complications.—The onset of symptoms, after infection has taken place, is insidious, with cough and expectoration. The latter is very constantly blood-stained, and there may be profuse hæmoptysis. Secondary pleurisy occurs when the cysts reach the surface of the lungs, causing pain. Examination may reveal no abnormal signs, at most there are a few scattered râles, together with signs of dry pleurisy at one point. Later in the disease the characteristic signs of the various complications may appear.

For general, abdominal and cerebral symptoms and complications, see pp. 306, 307.

Course.—This is chronic: the disease often persists for years, without giving rise to any acute disturbance apart from periodic hæmoptysis.

Diagnosis.—Distinction from other forms of hæmoptysis is accomplished by discovering the ova in the sputum. To facilitate the examination a little 0.1 per cent. sulphuric acid should be added to it.

Prognosis.—The immediate prognosis is good, the ultimate unfavourable, as there is considerable difficulty in eliminating the parasites, and permanent damage is wrought in the lungs.

Treatment.—Prophylaxis is important where the disease is endemic. No bathing should be allowed in infected rivers, and all water used for drinking or washing should be boiled or filtered. Crabs should not be eaten. When the disease has developed the patient should move from the infected area. Potassium iodide (grs. 10–20, t.d.s.) is recommended, but other treatment is symptomatic.

CONGENITAL CYSTIC DISEASE OF THE LUNG

Ætiology.—Congenital cysts of the lung may be met with in infants, children or adults. There is no infective or parasitic cause, and as in some instances cysts have been found in the fœtus they are considered to be due to developmental errors.

Pathology.—The following varieties are described: 1. The large balloon

cyst. This may completely compress a lung in an infant or young child. 2. The solitary cyst. This may occupy half the lung-field. 3. Multiple medium-sized cysts, often situated near the root of a lung. 4. Multiple small cysts. These cause a honeycomb appearance of the lung resembling bronchiectasis.

It is probable that in all cases the cysts are of bronchial origin. The lining membrane of the cyst is uniform, the cells having the characters of bronchial epithelium. Microscopically, the irregular distribution of the cartilage, muscle, elastic tissue and mucous glands in the supporting tissues differentiates congenital cysts from bronchiectatic cavities. The cysts may contain air only, or the contents may be watery, mucoid, or purulent if they become infected.

Symptoms.—These vary with the variety of cyst present. The large balloon cyst, met with in infants or young children, may result in severe respiratory and cardiac distress. In such cases there is cyanosis, dyspnoea and displacement of the trachea, mediastinum and heart to the opposite side of the chest. The percussion note over the cyst is hyper-resonant and the breath-sounds are absent. Solitary cysts often give rise to no symptoms and are only discovered on routine X-ray examination. When infected the clinical features may resemble those of lung abscess or bronchiectasis. With multiple medium-sized or small cysts no symptoms usually appear until infection occurs, though hæmoptysis may occur early. When infected, toxæmic symptoms develop, such as loss of weight, irregular fever, cough and expectoration which is sometimes offensive. Clubbing of the fingers may then soon be noted. On examination scattered areas of slight dullness, weak air entry with a few persistent râles may be detected.

Course, Complications and Sequelæ.—The onset of complications usually leads to the development of symptoms which call for investigation. Thus spontaneous pneumothorax may result from rupture of a cyst. In other cases suppuration occurs in the cyst with the formation of lung abscess, bronchiectasis or empyema. Cerebral abscess may be a late sequel.

Diagnosis.—This is suggested by X-ray and by lipiodol or neo-hydriol examinations and possibly tomography. If the space in the cyst is free from fluid the X-ray appearances must be differentiated from those of pneumothorax, an emphysematous bulla, a thin-walled tuberculous cavity or, in some cases, a diaphragmatic hernia. If the cyst contains fluid, further investigations are required to exclude the presence of such conditions as lung abscess, encysted pleural effusion or empyema, hydatid cyst, dermoid cyst or a blood cyst. A definite diagnosis can sometimes only be made after operation by microscopical examination of a portion of the cyst.

Prognosis.—This varies with the type of cyst present, the development of complications, and the treatment adopted. In many cases the prognosis is good, apart from rupture or infection. In the large balloon cyst there is risk of sudden death during an attack of distension.

Treatment.—The large balloon cysts which are causing respiratory and cardiac embarrassment call for immediate treatment by the insertion of a needle. Subsequently the only hope of recovery lies in pneumonectomy.

When the cysts are infected, treatment by postural drainage should first be adopted. Failure usually follows attempts at surgical drainage or collapse operations. If the cysts are unilateral and infected, the only hope of cure lies

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in radical removal of the portion of lung involved, either by lobectomy or by pneumonectomy; and so in bilateral cases it necessarily follows that no radical cure is possible.

HYDATID DISEASE OF THE LUNG

Hydatid cysts may develop in the lung in patients infected by the ova of the *Tænia echinococcus*.

Ætiology.—Man is the intermediate host of this parasite, and becomes infected directly or indirectly from the dog. The modes of infection and the life-history of the parasite are elsewhere considered (p. 314). Males are more often affected, and the condition is commoner among the poor than the well-to-do. It is rarely seen in this country except in patients from abroad, especially from Australia.

Pathology.—Hydatid cysts have been described in the lungs in from 5.6 to 16.8 per cent, of cases of hydatid disease in different parts of the world. The right lung is more often the site of the disease than the left, and the cyst is usually basic, though it may occur in the upper parts of the lung. It is generally supposed that infection of the lung is usually secondary to the liver, the ova reaching the lung through the diaphragm; but the occurrence of primary lung hydatid suggests the possibility of the embryo gaining access to the general blood stream, and thus reaching the lung by the pulmonary artery. There is, as a rule, a single cyst in the lung, but multiple or multilocular cysts are occasionally observed. The cyst may become as large as a cricket ball, but usually ruptures before it reaches this size. It has the same structure as hydatid cysts of other organs, with ectocyst and endocyst. It may develop brood capsules and daughter cysts, but is often sterile in this situation.

The reactive changes in the lungs are at first irritative and congestive, but eventually some fibroid changes occur, producing a more or less definite fibroid capsule around the ectocyst. The overlying pleura may become inflamed, thickened and adherent when the cyst grows near the surface. Rupture may occur into a bronchus, into the pleura, pericardium or peritoneum, or occasionally into the aorta or pulmonary vein. Rarely the contents of a small cyst may become inspissated, thus producing spontaneous cure.

Symptoms.—Until the cyst becomes large enough to cause irritation, there may be no symptoms, but sooner or later cough and expectoration develop. The latter is generally mucoid, and frequently bloodstained. Dyspnoea becomes apparent and pain results if the pleura is involved. The signs may be: diminished vocal fremitus, localised dullness and weak or absent breath-sounds and voice-sounds over a limited area, generally in the lower lobe. A few râles may be audible round the dull area. Occasionally with a large cyst there may be some bulging on the affected side, and "hydatid fremitus" has been described. The heart may be displaced in rare cases. Examination by the X-rays generally shows a suggestive rounded shadow with very little change in the surrounding lung, except in chronic cases where some fibrosis may be observed.

Some degree of eosinophilia is common but not invariable. When rupture into a bronchus occurs, there is usually sudden copious expectoration of watery

fluid containing hooklets. Daughter cysts and parts of the ectocyst may be coughed up and lead to dyspnoea and even suffocation from laryngeal obstruction.

After rupture, spontaneous cure may result if the ectocyst is expectorated. More commonly the cavity becomes infected and the symptoms and signs become those of chronic abscess (see p. 1179). Rupture into the pleural cavity produces great pain, dyspnoea, cyanosis and shock, similar to the condition induced by pneumothorax. Rupture into the pericardium or into a vein is usually quickly fatal. When rupture occurs into a serous cavity, urticaria and severe toxic symptoms sometimes develop.

Course.—This is generally progressive, though occasionally spontaneous cure occurs either before or after rupture. More commonly the cyst causes increasing pressure or irritative symptoms, and eventually rupture or suppuration produces acute manifestations.

Diagnosis.—The clinical features of pulmonary hydatid may be suggestive of pulmonary tuberculosis, pleural effusion or new-growth. Diagnosis may be difficult before rupture occurs; after this the discovery of hydatid hooklets or pieces of cyst-wall may establish the diagnosis. In suspicious cases the X-ray findings may be of great assistance, and confirmatory evidence may be obtained from cytological and serological examination. The former frequently shows eosinophilia, and the latter gives complement deviation when a suitable antigen, such as extract of hydatid cyst-wall, is used. A precipitin reaction may also be obtained with the fluid from another cyst. The Casoni intradermal test with the appropriate antigen has established itself as having special diagnostic value.

Prognosis.—The prognosis is serious owing to the risks of rupture and suppuration. Spontaneous cure is rare, but can occur. After rupture into a bronchus, recovery may ensue, but more commonly abscess formation results. Rupture into a serous cavity is frequently fatal. Early surgical treatment either before or after rupture improves the outlook.

Treatment.—Aspiration of the cyst, either exploratory or therapeutic, is to be avoided. If the cyst can be diagnosed or localised before rupture, the lung should be exposed by thoracotomy, the pleura stitched together and the cyst incised, the endocyst removed, and the cavity drained. Suppuration of a pulmonary hydatid must be treated as a pulmonary abscess.

THE PNEUMONIAS

The term pneumonia has been somewhat loosely applied to any inflammatory condition of the lung producing consolidation. When the consolidation affects large areas of lung uniformly it is described as lobar pneumonia, and when it is patchy or lobular in distribution it is called lobular or broncho-pneumonia.

1. LOBAR PNEUMONIA

Synonyms.—Croupous or Pleuro-Pneumonia.

Definition.—This is an acute infectious disease characterised by an inflammatory lobar consolidation.

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Ætiology.—*Predisposing causes.*—Pneumonia may occur at any age. It is common in children up to the sixth year, the incidence being about equal in the two sexes. It is commonest between the ages of 15 and 40, when there is a preponderance in the male sex of two or three to one. It is also a frequent terminal malady in the aged of both sexes. It may be doubted whether race has much influence, although in America and in the Rand mines the incidence and mortality among the black races are both high. Pneumonia is met with all over the world, but it is more rife in localities with changeable climate and cold winds. Its seasonal incidence is well marked; it is uncommon in the summer and autumn, and is most prevalent from November to March in this country. Although pneumonia is as a rule endemic and sporadic in its incidence, it is generally admitted that localised epidemics occur. Urban conditions, defective sanitation, overcrowding and insufficient ventilation all conduce to the incidence of pneumonia. It is not uncommon to obtain a history of several previous attacks. Although the disease often attacks those in normal robust health, there can be no doubt that debilitating conditions and diseases predispose to it, among them being chronic nephritis, diabetes, over-fatigue, exposure and alcoholic excess.

Exciting causes.—The exciting cause in most cases is the presence of the pneumococcus of Fränkel. It may be the only pathogenic organism found in the lung lesions and in the sputum, but not infrequently others, such as streptococci, staphylococci or Pfeiffer's bacillus are also present. Occasionally these organisms, and others, such as Friedländer's pneumobacillus, the *Bacillus typhosus*, the gonococcus and the *B. pertussis*, cause lobar consolidation; but these conditions should be regarded as varieties of secondary pneumonia, and differentiated from the acute primary condition now under consideration.

The pneumococcus.—The pathogenicity of the pneumococcus has been the subject of an interesting study by Cole, Dochez, Avery and Gillespie and more recently by Georgia Cooper and her co-workers. Originally three types were described; Types I., II., III., which together account for more than 50 per cent. of all cases. The remainder were included in a group referred to as Group IV. This has now been separated into 29 other types, making 32 in all, by means of serological reactions.

The American observers have shown that 40 per cent. of contacts with cases of pneumonia due to types I. and II. may harbour the corresponding organism for an average period of 23 days, and that they may develop pneumonia from it. They have further demonstrated that a convalescent patient may carry pathogenic pneumococci in his mouth for as long as 90 days from the onset of the disease. They have also found pathogenic pneumococci in the dust of rooms in which patients suffering from pneumonia have been nursed. The significance of this work is obvious. It confirms the view that pneumonia is an infectious disease, capable of being spread by carriers, by the convalescent patient, and by the dust of rooms.

Although the pneumococcus is the specific exciting cause, its activities are often determined by some other factor, such as chill, exposure, over-exertion or injury. The presumption is that these conditions lower the general resistance of the individual, and thus impair the defensive mechanisms. Post-operative pneumonia may be a further instance of this, but doubtless some supposed cases are in reality due to lobar collapse.

Pathology.—The pneumococcus is found in the pulmonary lesions and elsewhere when complications occur. In some patients it is found in the blood. These are referred to as bacteriæmic cases and are usually more severe and often associated with complications. Experimental investigations on animals indicate that the avenue of infection is to the lungs by way of the trachea and bronchi, the blood infection being secondary to the pulmonary lesion. Four stages are commonly described in the process by which the lung becomes consolidated and returns to normal, namely, engorgement, red hepatisation, grey hepatisation and resolution.

In the stage of engorgement the affected part of the lung is slightly enlarged, deep red in colour, and heavier than normal, although it still crepitates and floats in water. The pleura over it may be injected and lustreless and may even show early fibrinous exudate. On section, the hyperemia is obvious and there may be some cedema. On squeezing, frothy, bloodstained fluid exudes. Microscopically, the engorgement of the capillaries, the swelling and partial desquamation of the alveolar epithelium are the chief changes to be noted. In the stage of red hepatisation the affected area becomes completely consolidated, the general aspect on section being remotely similar to liver, hence the name hepatisation. The pleura is now notably inflamed and may be obscured by yellow fibrinous exudate. The hepatised area of lung is larger and much heavier than normal and bears the impress of the ribs upon it. On section, it is seen to be red in colour, solid and completely airless. It does not crepitate and it sinks in water. The lung tissue is found to be more friable than normal. On scraping the cut surface, which has a granular appearance, a reddish fluid is collected, containing small fibrinous plugs, which are practically alveolar casts. Microscopically, the alveoli are occupied by a coagulated exudate rich in fibrin and red blood corpuscles, with scanty leucocytes and a few larger cells derived from the alveolar epithelium. In the stage of grey hepatisation the lung tissue, although still solid, airless and non-crepitant, is greyish in colour, softer in consistence and still more friable. The surface of the section is less granular, and on scraping, a pale yellowish, almost puriform fluid is obtained. Microscopically, the blood vessels are found to be relatively empty, the alveoli are now incompletely filled, the fibrin and red corpuscles have largely disappeared, and the alveoli are occupied by leucocytes and desquamated alveolar cells. In the stage of resolution, the exudate becomes more liquid and its cellular constituents undergo fatty degeneration. The liquefied exudate is largely absorbed, although expectoration may possibly assist in its removal. The lung returns to its normal spongy state and the alveolar epithelium is replaced. Some pleural thickening or adhesion may, however, result. In very severe and fatal cases, the stage of resolution may be replaced by one of purulent infiltration, in which the lung becomes paler, softer and in places almost diffuent. The scrapings are practically purulent.

Although these four stages are described, it should be remembered that they are not sharply defined from one another, and that they only represent special appearances in a continuous process. Consequently, although the major part of the affected area of lung may be characteristic of any one of them, all four stages may be recognisable, especially in cases of a spreading type. The base is more often affected than other parts, and the right side more than the left, in the ratio of 3 to 2. The unaffected parts of the lung

may show some catarrhal bronchitis, or some degree of collateral hyperæmia or œdema. Pleurisy is an integral part of the affection, but it may proceed to serous or purulent effusion. Pericarditis and less frequently acute endocarditis may be found in fatal cases. Pneumococcal meningitis, arthritis and otitis are very occasionally observed. The liver and kidneys may show cloudy swelling, and the spleen is often slightly enlarged and soft. Jaundice may be observed, especially in right-sided cases. The right side of the heart may be engorged and dilated.

Symptoms.—The exact incubation period is not yet established, but it is short, being probably from 1 or 2 days up to a week. The onset is sudden and acute, with chill, shivering or rigor in the majority of cases. In children convulsions take the place of rigors. Vomiting at the onset is not infrequent, occurring in about one-third of the cases. Less commonly the onset is insidious, or is preceded by malaise and catarrhal symptoms. The temperature rises with the rigor, and as a rule a short, dry, irritating cough develops quickly, accompanied by a severe cutting pain on the affected side. The pain often becomes intense, and coughing may cause the patient great distress. The cough is frequently restrained as much as possible, and the breathing is rapid and shallow. By the second or third day the pain becomes less and the cough easier and more effective. Sputum, which at first is scanty, extremely viscid, tenacious and difficult to expectorate, now becomes more abundant, although remaining viscid. In typical cases it is characteristically rusty at this stage, containing mucus, altered red blood corpuscles, alveolar epithelium and large numbers of pneumococci. In a few instances a small but definite hæmoptysis occurs. Occasionally the sputum is thinner and of "prune juice" type.

Sleeplessness is often a distressing symptom, especially in the early and late stages. In some cases there are marked cerebral symptoms. Headache at the onset is common. Delirium is frequent, particularly in the asthenic type, in apical cases, and in alcoholics. In the latter it may be violent and is often like delirium tremens. The temperature is usually of high continuous type throughout, reaching 103°, 104° and even 105° F. or more on occasions, especially in the sthenic type. In the asthenic it is often of lower range. Defervescence is by crisis in about 60 per cent. of the cases. Crisis is commoner in sthenic patients in types I., IV. and VII., and occurs more often on the odd than on the even-numbered days of the disease. The most common day for the crisis is the seventh. It is rare before the third or after the ninth day. At the crisis the temperature falls to normal or subnormal in about 12 hours. The patient often sleeps soundly at this time and may sweat profusely; respiration is slower and easier and the pulse-rate falls. On waking, a dramatic change in the condition is usually noticeable. Pain and distress are ameliorated, cough is loose and easy, and the patient feels better, although weak. Looseness of the bowels and free diuresis are not infrequent, constituting the "critical evacuations." The crisis is sometimes preceded by a pseudo-crisis, in which a considerable fall of temperature occurs, with little or no improvement in the general condition. A slight post-critical rise of temperature of 1° or 2° F. is sometimes seen, but as a rule the temperature remains subnormal for a few days and slowly returns to normal. The pulse-rate may be slow for a time. Convalescence is generally rapid, although in cases which have had marked delirium, some mental confusion may be present for a day

or two. Defervescence by lysis is more common in asthenic patients. The temperature remits and may take from 2 to 4 days to reach normal or sub-normal levels.

The physical signs vary with the stage of the disease. At first there is some restlessness, but soon the patient assumes a dorsal decubitus, or lies more on the affected side. The cheeks are flushed, often markedly so on the side of the lesion. The eyes are bright, but the expression is one of pain or anxiety. A crop of herpes on the lips is very common. The tongue is thickly coated and white, becoming dry and cracked in bad cases at a later stage. The skin feels dry and pungently hot. The *alæ nasi* are in action, and in children a puff or grunt accompanies each expiration, while the pause follows inspiration, instead of expiration. The respiration and pulse-rate are increased, the former disproportionately, so that the pulse respiration ratio becomes 3 or even 2 to 1, instead of the normal 4 or 5 to 1.

In the early stage the pulmonary signs are slight. At the most there is lessened movement and diminished vocal fremitus over the affected area, with dubious impairment of note, weak air entry and possibly a few crepitations (indux), or pleural friction sounds, vocal resonance being unaltered. Of these, lessened air entry is probably the most common. Slight hyper-resonance of the opposite lung, with harsh breathing, may lead to error in diagnosis as to the side affected.

The signs of consolidation (hepatisation) are generally apparent on the second or third day, except in cases where the disease starts deeply (central pneumonia). There is definite limitation of movement on the affected side, which is, however, slightly increased in size, as can be demonstrated by mensuration. Vocal fremitus is markedly accentuated over the affected area, except in massive pneumonia, and friction fremitus may be palpable. The note on percussion is dull, but has not the resistant stony character of that over an effusion. The note above or below the consolidated area is sometimes skodaic. The breath-sounds are tubular, and a few crepitations may be heard, but frequently adventitious sounds are absent. In some cases a friction rub is audible. Bronchophony and pectoriloquy are usually very marked over the consolidated area. The breath-sounds in other parts may be vesicular or harsh, and a few rhonchi may be present. The heart is usually in its normal situation, but is sometimes slightly displaced away from the affected side. In later stages the signs of dilatation of the right heart may become apparent.

During resolution, which begins after the crisis or during lysis, the tubular character of the breath-sounds disappears, and they become at first bronchial and later harsh or vesicular. Coarse moist sounds, known as *redux* crepitations, are heard both with inspiration and with expiration. The dullness gradually diminishes, and the voice-sounds return to normal. In basal cases, in which the diaphragmatic pleura is involved early, there may be pain, tenderness and abdominal rigidity simulating peritonitis, perforation or appendicitis. Graham Hodgson has shown by X-ray examination that the diaphragm rises in pneumonia, and in types I. and II. the consolidation begins near the hilum and spreads peripherally. In type III. the appearances are less characteristic, and may begin peripherally and spread centrally. Resolution reverses the order of appearance. It is rare for the spleen to be sufficiently enlarged to be palpable. The blood shows a leucocytosis up to

20,000, occasionally up to 50,000 in young or sthenic patients. Blood culture may yield pneumococci, although this was successful in only 30 per cent. of cases at the Rockefeller Institute. The urine is diminished in quantity, and there is a great reduction in the sodium chloride excretion until the crisis. Albumin and albumose are frequently found in small quantities in the urine during the febrile stage, and a few granular casts may be present. The uric acid excretion is increased to two or three times the normal, commencing the day before the crisis and generally falling to normal during the ensuing week. This is probably due to disintegration of the exudate in the alveoli, and so forms a measure of resolution, although some authorities maintain that it runs parallel with leucocytosis and not with cell destruction. Pneumococci can sometimes be obtained from the urine at the height of the disease. The blood pressure usually falls during the course of pneumonia, and according to G. A. Gibson a sudden rise indicates the imminence of some complication, such as delirium, whereas a sudden fall suggests the onset of cardio-vascular paralysis.

The disease does not always follow the typical clinical course, and certain varieties are described :

Apical pneumonia.—The consolidation may be limited to the apex or upper lobe of one lung. This is more common in children, the aged and alcoholics, and is often associated with marked cerebral symptoms.

Creeping pneumonia (Migratory or wandering pneumonia).—The consolidation spreads irregularly in one or both lungs. Partial resolution occurs, but there is no true crisis, and as successive portions of the lungs become involved the temperature exacerbates, eventually falling by lysis in cases that recover.

Central pneumonia.—The symptoms and appearance of the patient may suggest lobar pneumonia, and yet no abnormal signs can be detected in the lungs. In some of these cases there may be a deep-seated consolidation, which can usually be revealed by X-rays. A typical crisis may occur.

Massive pneumonia.—The bronchi, as well as the alveoli, may be filled with a fibrinous exudate. It is a rare condition and leads to difficulty in diagnosis, as the physical signs resemble those of pleurisy with effusion, vocal fremitus being diminished and breath-sounds weak or absent. The heart, however, is not displaced, or only slightly so.

Post-operative pneumonia.—It is probable that some cases that were formerly described as post-operative pneumonia were in reality instances of massive lobar collapse (see p. 1166). At times a pneumococcal pneumonia follows the administration of a general anæsthetic, but it does not present any peculiar features.

Traumatic pneumonia.—The fact that an injury to the chest may be followed after a short interval by a pneumonic process in the lungs has long been recognised. The condition was called "contusional pneumonia" by Litten in 1881. Külbs showed later that the changes in the lungs in dogs following local trauma were mainly hæmorrhagic, and that the lung opposite to the side injured may be affected by "contre-coup." In the recorded cases of traumatic pneumonia two types can be differentiated—(1) those with hæmorrhagic lesions only, and (2) those showing hæmorrhagic foci with a superimposed bacterial infection. The former recover rapidly, the latter often lead to a fatal issue.

Pneumonia in children.—This often presents certain characteristic features. There is rarely any sputum, the expectoration being swallowed. Convulsions at the onset are common. The lesion is often at the apex of the lung. Cerebral symptoms are frequent, and empyema or otitis media often occurs as a complication.

Pneumonia in the aged.—This occurs frequently as a terminal infection, often leading to a rapid and comparatively painless death. The onset may be insidious and the physical signs slight.

In *pneumonia in the insane*, lobar consolidation is often observed, without marked constitutional disturbance other than fever.

Secondary pneumonia.—Lobar pneumonia may develop during the course of certain acute specific fevers, notably enteric, typhus and plague. It is doubtful whether a true lobar pneumonia occurs in influenza, the condition to which the name *influenzal pneumonia* is applied being due to coalescing lobular pneumonia with hæmorrhagic extravasations.

Complications.—Delayed resolution not infrequently occurs, the signs of consolidation persisting for weeks instead of days. Frequent careful examinations should be made and possible errors in diagnosis considered, such as the presence of tuberculosis or empyema. Gangrene and abscess are rare but recognised complications.

Dry pleurisy is an invariable accompaniment when the consolidation reaches the surface, and in a considerable proportion of cases slight serous effusion occurs. This occasionally becomes frankly purulent and an empyema results. Bronchitis is common and may be due to a complicating secondary infection. Cardiac failure is a grave occurrence and can be recognised by increasing cyanosis, lividity and dyspnoea, with signs of enlargement of the right heart and with enfeeblement of the heart-sounds. Pericarditis is not very uncommon and is a serious complication. It may be dry or proceed to serous or purulent effusion. Acute endocarditis, sometimes of infective type, occurs. Abdominal complications are comparatively rare. They include pneumococcal peritonitis, colitis and nephritis. Acute dilatation of the stomach occurs in rare cases, and is usually rapidly fatal. Meteorism is more common and, although serious, is more amenable to treatment. Jaundice, due to catarrh of the bile-ducts, or to hæmolysis, is sometimes present.

Pneumococcal meningitis supervenes in rare cases, and is invariably fatal. Delirium has already been referred to, and is especially serious when occurring in alcoholics. *Peripheral neuritis* has been described, but is very uncommon. Otitis media and arthritis, proceeding sometimes to suppuration, occur as complications, both being commoner in children. A parotitis, sometimes going on to suppuration, is an occasional and serious complication, especially in old people. During convalescence, *thrombosis of the veins of the legs* may occur in rare instances.

Sequelæ of lobar pneumonia are uncommon. Perhaps the most remarkable is the liability to subsequent attacks possibly due to infection by different types of pneumococcus. Some permanent pleural thickening or adhesion may occur, and after an empyema the usual sequelæ may result. *Pulmonary fibrosis* (chronic interstitial pneumonia) is rare, especially in comparison with its frequency after broncho-pneumonia; this may lead to bronchiectasis.

Course.—The course depends on the type and virulence of the infection and on the resistance of the patient. In a typical *sthenic* case, consolidation

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is well established by the second or third day, defervescence by crisis occurs usually on the seventh day, signs of resolution become apparent a day later, and all signs clear up within 14 days of the onset. In asthenic cases the course is less typical and often prolonged to 9 or 10 days, defervescence occurring by lysis. In fatal cases, death commonly occurs between the fourth and tenth days, although severe cases may prove fatal as early as the first or second day. After the tenth day a fatal result is generally due to complications. An abortive course is described, in which typical symptoms occur with slight or indefinite signs, the temperature falling by crisis within 36 hours, followed by rapid recovery. This group includes the "*maladie de Woillez*." It is difficult in many instances to establish the true causation of such cases.

Diagnosis.—When the disease is well established and the history is available, diagnosis is as a rule easy. To prove the pneumococcal origin, sputum examination, lung puncture, or blood or urine culture is necessary. The investigation of serum reactions is necessary to establish the type of pneumococcus concerned if specific treatment is employed.

At the onset, especially before the signs of consolidation develop, difficulties in diagnosis often occur. The initial rigor or convulsion with vomiting may suggest scarlet fever. In children, especially those with early apical pneumonia, headache, vomiting, convulsions, head retraction, squint and even slight Kernig's sign may lead to an erroneous diagnosis of meningitis. Pain in the side and cough, the altered pulse respiration ratio, and the presence even of slight abnormal physical signs in the chest, usually suffice in both instances to suggest the correct explanation.

Occasionally the onset of pneumonia may simulate an acute abdominal condition, such as appendicitis or perforation of a gastric ulcer, owing to referred abdominal pain, sometimes with rigidity. The diagnosis may be very difficult, and laparotomy has not infrequently been carried out in error. The history, the pulse respiration ratio, the absence of tenderness on rectal examination, and the presence of pulmonary signs usually enable a correct decision to be made.

Influenza may start acutely and simulate pneumonia, but the distribution of the signs and the examination of the sputum generally serve to distinguish between them. Typhoid fever less often gives rise to difficulty, but some cases of pneumonia pass quickly into a typhoid state, while some cases of typhoid fever develop consolidation in the first week.

When consolidation is well established, the chief conditions to be differentiated are—(1) Broncho-pneumonia. The slower onset, the more prolonged course, the bilateral patchy physical signs, and the marked predominance of the bronchitic manifestations usually suffice to differentiate this group of conditions. (2) Secondary pneumonias, such as those in plague, typhoid fever, and influenza, can be diagnosed only from the history, the associated symptoms and signs, and from the bacteriological examinations. (3) Friedländer's pneumonia is rare. Its course is short, its prognosis grave, and it can only be recognised by bacteriological investigation. (4) Massive collapse. The diagnosis of this condition and its differentiation from pneumonia are discussed on p. 1167. (5) Acute pneumonic tuberculosis. The onset and early signs may be identical with those of pneumonia. The persistence of the fever, its tendency to become remittent or intermittent, and the

occurrence of night sweats should suggest looking for tubercle bacilli in the sputum. (6) Pleural effusion and empyema. Differentiation is generally easy, except in cases of massive pneumonia. Investigation of the position of the cardiac impulse, and of vocal fremitus and resonance, affords the most valuable aid. Grocco's triangle may also assist. In some cases the diagnosis can only be established by the exploring needle. (7) Infarction of the lung in cardiac disease, causing pain, cough, blood-stained expectoration, and dyspnoea, may simulate pneumonia. The absence of fever, the presence of the cardiac condition and the localised physical signs are generally characteristic. (8) Acute oedema of the lung, especially in mitral stenosis, may suggest pneumonia. Fever is generally absent, the sputum is typical, and the primary cause may be apparent. An attack of paroxysmal tachycardia may give rise to difficulty, when it leads to dullness and crepitations at the bases, but careful examination should establish the very rapid action of the heart and the evidence of venous engorgement in other parts.

Prognosis.—Lobar pneumonia is a serious disease, with a high mortality rate. This is profoundly influenced by age and by recent methods of treatment. It is but little fatal in childhood, except in the first years of life. After the age of 60, the mortality until the new chemo-therapeutic measures were employed was from 60–80 per cent. The New York investigations at the Rockefeller Institute demonstrated the importance of the type of pneumococcus in prognosis; thus, it was found that the mortality of cases with types I. and II. was about 25 to 30 per cent., of those with type III. 50 per cent., and of other types collectively only 12 per cent. With M. and B. 693 (Dagenan, sulphapyridine) the average mortality of all types has been reduced to about 8 per cent.

The previous habits and history of the patient influence prognosis considerably; chronic alcoholism doubles the risk of a fatal issue, and the outlook is grave in patients who are the subjects of diabetes, chronic cardio-vascular disease, nephritis, marked debility or obesity. Unfavourable indications during the course of the disease are profound toxæmia, a pulse-rate persistently 130 or more, a blood pressure in millimetres of mercury lower than the pulse-rate, and a temperature remaining at 105° F. or over for several days. Absence of the usual leucocytosis is generally of sinister import. Dilatation of the right heart, with cyanosis progressing to lividity, is most grave. Modern statistics confirm the traditional view that labial herpes is a favourable prognostic sign.

Of complications, meningitis is invariably fatal, unless it responds to treatment by sulphapyridine while septic endocarditis is extremely grave. Cases with abscess or gangrene, although serious, sometimes recover, especially if operative treatment is practicable. The prognosis of those with pericarditis is serious, but not uniformly unfavourable. Cases with bilateral empyemata show a high mortality. Late delirium is a very serious indication.

Treatment.—**PROPHYLACTIC.**—Prophylactic vaccination has been used with success by Lister in South Africa. He employed a triple vaccine, made from three types, and gave 6000 millions of each. Three injections were made at weekly intervals.

When a case has occurred, all contacts should have a throat examination, and if virulent pneumococci are found a suitable antiseptic mouth wash

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should be used. The room in which the patient has developed the disease should be disinfected afterwards. If possible, no case of pneumonia should be nursed in a general ward of a hospital, and the doctor and nurse in attendance should wear gauze masks. All sputum should be disinfected. The patient should lie in a narrow bed away from a wall to facilitate nursing. The room should be well ventilated, and the temperature maintained at 60° to 65° F. Treatment in the open air is not advisable except in very mild weather. Two important factors are rest and sleep. The patient should, therefore, be disturbed as little as possible by the examination of the physician and by the attentions of the nurse. He should not, however, be allowed to lie flat all the time, to avoid basal congestion. The diet should be restricted to fluids and semi-solids, eggs, milk, meat extracts and the various invalid foods being given up to 2 or 3 pints in the 24 hours. Dextrose, in the proportion of 2 to 4 ounces to the pint of lemonade or orangeade, is useful. Too much milk should be avoided, as it is liable to cause indigestion and flatulence. The irritating cough, which induces such intense pain, should be checked by a sedative linctus, or by lozenges, but it may be necessary to inject $\frac{1}{2}$ th grain diamorphine hydrochloride (heroin), or even $\frac{1}{4}$ th grain morphine to relieve pain and to induce sleep in the early stage. Local applications to the chest help to relieve pain. Hot linseed poultices to the back and side may be employed, but cataplasma kaolini or antiphlogistine applied on lint does not require such frequent changing and disturbs the patient less. Care should be taken to avoid too hot application, which may injure the skin. A pneumonia jacket is preferred by some, by others the ice poultice or ice-bag is found very soothing. A dose of calomel should be given at the onset, and the bowels should be opened daily, either by a laxative or by a small soap enema unless at any stage the patient is profoundly exhausted.

Sulphapyridine (Dagenan, M. & B. 693) has proved of great value in the treatment of lobar pneumonia due to all types of pneumococcus. It is put up in tablets containing 0.5 grm. for adults, and 0.125 grm. for infants and young children. The usual dosage for adults is 4 tablets repeated in 4 hours, followed by 2 tablets every 4 hours for 2½ days. Subsequently 1 tablet is given every 4 hours for 24 hours, then 1 tablet every 8 hours for 36 hours, making a total of 23 grms. in 5 days. In infants 0.125 to 0.25 grm. is given four-hourly. The dose for children is based on the body weight, but they require proportionately up to 50 per cent. bigger doses than adults, e.g. up to the age of 3 years 0.375 grm., and at 5 years 0.5 grm. is given four-hourly, until the temperature falls to normal, when a smaller dose may be given eight-hourly for two further days. Children tolerate the drug well. The tablets are best administered crushed and suspended in water, milk or fruit juices. The patient should not be given sulphur-containing substances such as eggs, onions, Epsom salts or Glauber salts during the course of the treatment. A sufficient quantity of fluid should be taken to result in the passage of at least 50 ounces of urine in the 24 hours. At whatever stage in the illness the treatment is instituted the temperature usually falls to normal by lysis in 24 to 36 hours when the drug is effective, although the actual process of resolution in the lung is not accelerated. In some cases vomiting prevents an adequate concentration of the drug in the blood and the treatment has to be abandoned, but frequently the difficulty can be overcome by varying the method of administration. The most important toxic effects

which may occur include nausea and vomiting probably central in origin, headache, cyanosis due to methæmoglobinæmia, and hæmaturia. The latter may occur if the urine is too concentrated and is thought to be due to the formation of crystals of acetylated sulphapyridine. If the drug is administered for longer than 5 days there is a possibility of further complications ensuing, such as drug fever and granulocytopenia. In the former the temperature may rise to 104° or 105° F. and morbilliform, scarlatiniform or urticarial rashes appear. A preparation of soluble sulphapyridine (Dagenan—sodium) may also be given intramuscularly or intravenously if the patient cannot tolerate the drug by mouth. It is put up in ampoules containing 1 grm. in 3 c.c. The intramuscular injections are given undiluted and are painful. For intravenous injection the contents of the ampoule are diluted to 10 c.c. with normal saline. The usual dose is 1 grm. every 4 hours. In the early stage, a simple saline diaphoretic mixture may give comfort by promoting the action of the skin and by rendering the sputum less viscid. For this purpose liq. ammon. acetat. min. 120, potass. citrat. grs. 20, syrup. aurantii min. 60 and water to the ounce, may be given every 4 to 6 hours. The use of depressant drugs, such as tartar emetic, aconite, or pilocarpine, although formerly recommended, is now generally discarded. Expectorants such as ammonium carbonate or iodide of potassium in doses of 3 to 5 grains are often recommended after the second day, but are of doubtful utility.

Cardiac embarrassment and failure are the conditions requiring the most active treatment in this disease. A careful watch should be kept upon the colour of the patient, the condition of the pulse and the size of the heart. Digitalis, in doses of 5 to 15 minims of the tincture, may be added to the mixture, or given with brandy. Nikethamide (coramine), either by the mouth or hypodermically, is often of value and has largely replaced the use of camphor, though the latter dissolved in sterile oil may be given in 3-grain doses twice daily. If signs of acute heart failure occur, strophanthin gr. $\frac{1}{200}$ may be given intravenously, or digitalin gr. $\frac{1}{100}$ with strychnine sulphate or hydrochloride gr. $\frac{1}{80}$ to $\frac{1}{40}$ hypodermically. The latter may be repeated in from 4 to 6 hours if necessary. Strychnine alone may be given in doses of gr. $\frac{1}{80}$ every 4 hours, and is often very useful. Other circulatory tonics which may be employed hypodermically are pituitary (posterior lobe) extract $\frac{1}{2}$ to 1 c.c., or adrenaline 5 to 10 minims of 1 in 1000 solution. Alcohol is often useful; it should not be given too early in the attack, but where there are indications of incipient cardiac weakness 4 to 6 ounces daily may be given, and this even to alcoholics.

Oxygen inhalations may be helpful in any case where there is distress or cyanosis. It should be warmed, and may be bubbled through alcohol. It may be administered continuously by means of a double nasal catheter with flow-meter and humidifier if available, or the B.L.B. mask by which with various adjustments an alveolar concentration of oxygen of more than 90 per cent. can be obtained. An oxygen tent is now rarely used except for infants and children. These methods may prove of the greatest value where there is marked anoxæmia. Venesection to the extent of 10 or 12 ounces is of some value if there is lividity from right-sided engorgement, especially in sthenic cases. As a rule it is best not to interfere with the temperature by antipyretic drugs and measures unless it remains over 104° F., when sponging, either tepid or cold, should be tried.

Sleeplessness is a frequent and distressing symptom and requires treatment. In the early stages 10 grs. of Dover's powder or an injection of morphine or diamorphine hydrochloride (heroin) are usually effective. In the later stages, morphine should only be given with care, and then in association with atropine gr. $\frac{1}{200}$ to $\frac{1}{100}$ and strychnine gr. $\frac{1}{80}$ to $\frac{1}{40}$. Paraldehyde min. 120 with syrup of orange in 2 ounces of water, is safe and often effective. Chloralamide grs. 20 to 30, with bromides may be tried. In cases with delirium an ice-cap should be applied to the head, and the patient sponged with tepid water. Morphine may be necessary, and in severe cases hyoscyne, gr. $\frac{1}{100}$, may be injected; but the latter is a dangerous drug and the patient's condition should be watched, and strychnine administered if necessary. Tympanites, when present, is distressing and exhausting, and should be treated by passing a rectal tube or by the administration of an enema of asafoetida or a turpentine wash-out. If these measures fail, carbachol (doryl) or acetylcholine may be employed cautiously.

Specific.—Specific antisera are now available for many of the types, and have been used with some success. Horse serums, highly concentrated and refined by Felton's method, are now available for types I., II., IV., V., VII. and VIII. Rabbit antipneumococcus serum is available for type III., and many others. It has the advantage of ease of preparation and of considerable concentration. If it is proposed to employ serum treatment it is necessary first to determine the type of pneumococcus concerned by serological tests. A rapid method involving the testing of the organisms in the fresh sputum against test serums has been introduced by Armstrong and simplified by Neufeld. The specific serum causes swelling of the capsules in the corresponding type. The patient's sensitiveness to the serum must then be determined by an intradermic injection of 0.02 c.c. of diluted serum (diluted $\frac{1}{10}$ with saline), and if he shows a reaction he must be desensitised by small injections to prevent anaphylaxis. The serum is then diluted with an equal volume of warm sterile saline and 10 to 15 c.c. injected intravenously at the rate of 1 c.c. a minute, followed by 90 c.c. more during the next quarter of an hour. This dose is repeated every 8 hours until improvement occurs. Felton's serum (2000 units in 1 c.c.) is administered intravenously undiluted, after being warmed to body temperature and after careful preliminary tests as to sensitiveness. The initial dose is 10,000 units, slowly injected intravenously. Amounts up to 40,000 or 50,000 units are given in the first 24 hours, though as much as 100,000 units may be necessary in severe cases. The earlier it is given, the greater is the likelihood of success. It is as a rule unnecessary in young children and adolescents, and contra-indicated in advanced age and in patients known to be allergic. Rabbit serums are given undiluted intravenously after being warmed to body temperature. At least 5 minutes should be taken to inject each dose. If anaphylactic manifestations occur adrenaline should be given subcutaneously at once.

Vaccine treatment is recommended by some, but the results are generally disappointing during the acute stage. A common method is to give 20 millions of a stock pneumococcus vaccine, and then to use an autogenous one as soon as it can be made. Sensitised and detoxicated vaccines have also been prepared. Vaccines seem to be more valuable in cases of delayed resolution.

Artificial pneumothorax has been suggested as a method of treatment.

There is as yet no convincing evidence of its value, but in cases with severe pain, the introduction of sufficient air to separate the inflamed surfaces of the parietal and visceral pleura is worth considering.

2. BRONCHO-PNEUMONIA

Synonyms.—Lobular Pneumonia; Catarrhal Pneumonia; Capillary Bronchitis.

Pulmonary consolidation of lobular distribution occurs in a variety of conditions which have little else in common. A satisfactory classification is at present difficult. The term capillary bronchitis is misleading and should be regarded as obsolete, since any inflammatory condition affecting the finer bronchi is invariably associated with alveolar changes. For convenience the following varieties of broncho-pneumonia may be described: (1) Primary. (2) Secondary. (3) Aspiration or deglutition. (4) Tuberculous.

a. PRIMARY BRONCHO-PNEUMONIA

Ætiology.—This form almost invariably affects infants under 2 years of age, in whom a lobular pneumonia seems sometimes to occur under conditions which would induce lobar pneumonia in older children or adults. It occurs equally in the two sexes, and is commoner in the winter and the spring. Rickets, malnutrition and debility are predisposing conditions, but it sometimes develops in healthy robust infants after exposure or chill. The pneumococcus is the organism usually found, either alone or in association with others, such as streptococci, staphylococci, the *Micrococcus catarrhalis* or Friedländer's pneumo-bacillus.

Pathology.—Widely scattered patches of consolidation are found in one or both lungs. These may be small and separated by areas of collapse or emphysema. Occasionally they are almost confluent, and at first sight appear like lobar pneumonia, constituting the pseudo-lobar form; but careful observation shows that the distribution is lobular and that zones of incomplete consolidation or of normal lung tissue separate the solid areas. If the process reaches the surface some degree of pleurisy is present, although this is less than in lobar pneumonia.

Microscopically, the appearances approximate to those of the lobar form; the alveoli are found to be filled with exudate, in which leucocytes and desquamated epithelial cells are present, together with some fibrin and red blood corpuscles. Catarrhal changes are also present in the bronchi.

Symptoms.—The onset is acute, with vomiting and chill, or convulsion, as in lobar pneumonia, but may be more gradual. Cough, cyanosis and dyspnoea develop rapidly. There is no expectoration, since infants and young children swallow the sputum. Cerebral symptoms simulating meningitis are common. The temperature rises quickly to 103°, 104° F., or higher, and the range is of the same character as in lobar pneumonia. Deferrescence by lysis is the rule.

The physical signs are variable. In cases with widespread consolidation they are very similar to those of lobar pneumonia, with dullness, tubular breathing, increased voice-sounds and crepitations. In other cases, although

the aspect of the infant appears characteristic of pneumonia, with rapid breathing, cyanosis, reversed rhythm of inspiration and expiration, sucking in of the lower ribs and dilation of the *alæ nasi*, the signs are more scattered. Tubular breathing and increased voice-sounds may only be heard in localised patches, especially in the lower lobes. Crepitations are commonly present, and rhonchi may be audible over both lungs.

Complications and Sequelæ.—These are similar to those of lobar pneumonia.

Course.—This is usually short, the temperature falling in from 3 to 7 days, but it may be more prolonged and be suggestive of tuberculosis, or some other form of secondary bronchitis.

Diagnosis.—Primary broncho-pneumonia has to be distinguished from the lobar form to which ætiologically and pathologically it is so closely related. The acute onset without previous respiratory symptoms will suggest its primary character, while the patchy distribution of the signs generally suffices to establish its lobular distribution. In pseudo-lobar forms, this differentiation may be almost impossible during life. The cerebral symptoms at the onset, and the early absence of pulmonary signs may give rise to difficulty, as in the first stage of lobar pneumonia.

Prognosis.—The prognosis of primary broncho-pneumonia is generally unfavourable, especially in very young or debilitated infants.

Treatment.—This is practically identical with that of secondary broncho-pneumonia in children.

b. SECONDARY BRONCHO-PNEUMONIA

In this condition there is inflammation of the bronchi, spreading down to and involving the alveoli. It is generally a catarrhal process, but may go on to septic or suppurative manifestations.

Ætiology.—A secondary broncho-pneumonia may occur at any age, but is much more common in early and advanced life. It is equal in its incidence in the two sexes. It frequently occurs as a complication of measles, whooping-cough, psittacosis and influenza, less commonly in cases of diphtheria, scarlet fever, plague and the enteric group. A bronchitis starting in the larger tubes may spread downwards to the alveoli. Broncho-pneumonia may develop during the course of acute gastro-enteritis. A secondary broncho-pneumonia occurs as a terminal infection in many old and debilitated persons and in those with chronic wasting or cachectic diseases, and also in chronic cardio-vascular conditions, chronic renal disease and in many progressive nerve degenerations. Any septic process may produce a metastatic broncho-pneumonia. This occurs in association with otitis media, suppurative processes about the appendix or Fallopian tubes, and cerebral abscess.

Bacteriology.—This is, as might be expected, very varied. Streptococci are frequently present, especially the hæmolytic variety, generally associated with other organisms, such as the pneumococcus, Pfeiffer's *H. influenza*, staphylococci and those found in catarrhal conditions of the upper air-passages. The *B. pertussis* may be found in cases associated with whooping-cough, the *B. pestis* in plague, and occasionally the *B. diphtheriæ* in diphtheritic broncho-pneumonia. The importance of Friedländer's *B. pneumonia* was formerly overestimated in this connection.

Pathology.—When, from any of the above-mentioned causes, an inflammatory process reaches the finer bronchi, the alveoli become affected in three different ways. Owing to the blocking of the bronchi by secretion or exudate, small areas of collapse of lobular distribution are produced. The inflammatory process extends into some or all of these, and areas of lobular consolidation result. Not infrequently the adjacent groups of alveoli become distended and are thus in a condition of acute emphysema. The lungs are normal in size or slightly enlarged. The surface presents a somewhat uneven, mottled appearance. There are small projecting patches of firmer consistence and reddish-grey colour, due to the consolidated lobules. Adjacent areas may be depressed and slaty blue, from lobular collapse, while the intervening lung tissue is normal or pinkish and emphysematous. There may be dimness or slight roughening of the pleura where the consolidated areas reach the surface, but serous or purulent effusion is uncommon. On section, the lung is found to be congested and sometimes œdematous, especially at the bases, while the bronchi exude pus or muco-pus from their cut ends. The reddish-grey areas of consolidation are found to vary in size from a pin's head to a hazel nut. They are generally more abundant in the lower lobes, especially posteriorly. The consolidated and collapsed areas both sink in water, and do not crepitate. There is often some peribronchitis, and the bronchial glands are usually enlarged. Microscopically, the finer bronchi and the consolidated alveoli are found to be filled with an exudate containing large numbers of leucocytes and desquamated, proliferating epithelial cells, but in which few red blood corpuscles and little or no fibrin are found.

In the very acute condition to which the name capillary bronchitis was formerly applied, consolidation may not be apparent, but microscopical examination invariably demonstrates the involvement of the alveoli. In influenzal broncho-pneumonia the pathological changes probably commence as an exudative bronchiolitis, associated with capillary hæmorrhages. Secondary infections are probably responsible for the consecutive broncho-pneumonic process, which results in flooding of the alveoli with an exudate containing red cells, but little or no fibrin.

Symptoms.—In the cases ensuing on bronchitis in infants or old people (formerly called capillary bronchitis), initial symptoms may be slight, and simply those of ordinary bronchitis, namely, malaise, slight fever and cough, with or without expectoration. The implication of the finer tubes and alveoli is usually marked by a rapid rise of temperature, great prostration, quick breathing and an irritating, persistent and often ineffective cough. In children, the *alæ nasi* work, the lower ribs are sucked in, and the pneumonic type of breathing develops. The patient becomes cyanosed, the pulse is rapid, 120 or more, and the respirations 50 or 60 per minute. The physical signs are in general indistinguishable from those of primary broncho-pneumonia, but breath sounds are often harsh and puerile, while tubular breathing is not heard, or only in very localised areas. In old people, cyanosis, restlessness and delirium may occur, and later the cough become less frequent, the patient being drowsy and tending to sink down in the bed, whereas previously there was orthopnoea. These symptoms are ominous and indicate failure of the respiratory centre.

The physical signs are often those of bronchitis, harsh or weak inspira-

tion and prolonged expiration, sibilant and sonorous rhonchi and crepitations or crepitant râles especially at the bases. Patches of tubular breathing with increased voice sounds may develop but are not always present.

In other forms of secondary broncho-pneumonia similar symptoms and signs develop more insidiously in the course of the primary disease. Broncho-pneumonia should be suspected when cough, expectoration and dyspnoea, together with a remittent type of temperature, develop in the course of an acute specific fever or other severe illness. In all forms, anorexia is common, the mouth and tongue become dry, and thirst is complained of. The urine presents the usual high-coloured, concentrated character of febrile conditions. It is often diminished in quantity, may contain a small quantity of albumin, and not infrequently deposits urates.

Complications and Sequelæ.—These are relatively infrequent. Pleurisy may proceed to effusion, and when this occurs it is often purulent. Abscess and gangrene are rare, but develop rather more frequently than after lobar pneumonia. Other complications, such as pericarditis, endocarditis, meningitis and nephritis, are probably due to blood-borne metastasis.

The most important sequel is pulmonary fibrosis, which is often the origin of bronchiectasis later in life. Pulmonary tuberculosis is frequently described as a sequel, especially after measles, and may be due to inflammatory changes in the bronchial glands activating a quiescent tuberculous deposit there. In many cases of tuberculosis described as following on broncho-pneumonia, it is more probable that the original lung affection was tuberculous.

Course.—Secondary broncho-pneumonia generally has a longer course than either the primary form or the lobar variety of pneumonia. The fever often persists in remittent type for two or three weeks, and sometimes even for two or three months, although in this case tuberculosis should be suspected. The decline is almost always by lysis. Convalescence is often slow, the patient being left thin, weak, anæmic and debilitated.

Diagnosis.—The development of pulmonary symptoms, and of more or less characteristic physical signs in the course of measles, whooping-cough or one of the other diseases mentioned above, usually renders the diagnosis easy. Difficulty may arise in regard to tuberculosis, which in one form produces lobular pneumonic lesions with symptoms and signs indistinguishable from other varieties of secondary broncho-pneumonia. In any case where the fever lasts more than three weeks, or where the signs show no tendency to resolve or are chiefly apical, tuberculosis should be suspected. Unfortunately in children sputum is rarely available. An attempt is sometimes made to obtain it on gauze held in forceps, after exciting cough by touching the fauces. The mucus in the fauces may also be examined for tubercle bacilli. The diagnosis may, however, remain doubtful, until signs of softening become established.

Bronchitis rarely gives rise to difficulty. The fever is usually less high, and of shorter duration, while the physical signs are different, signs of consolidation being entirely absent. Hypostatic pneumonia may have to be considered. There is usually some obvious cause for this, such as cardiac disease and failure, or prolonged confinement to bed. The temperature is generally lower and the distribution is lobar.

Pleural effusion and empyema can generally be differentiated by the alteration of vocal fremitus and the displacement of the cardiac impulse.

In case of difficulty the exploring syringe enables a distinction to be made.

Prognosis.—The prognosis in secondary pneumonia is serious. Many deaths occur from this complication in the acute specific fevers, particularly with measles and influenza. Even the form following on severe bronchitis is frequently fatal, especially in old people and in wrongly fed or debilitated infants. The development of delirium, of a pulse-rate over 150, of marked cyanosis and dyspnoea is unfavourable. In old people, drowsiness, sinking down in the bed, and cessation of cough are very grave indications.

Treatment.—The treatment is very similar to that of lobar pneumonia, except that stimulant and expectorant drugs may be necessary from the first. At the present time there is practically no difference in the methods of treatment applicable to the primary and secondary forms. In cases due to pneumococcal infection sulphapyridine should be employed (see p. 1234). If streptococci are established as the infecting agent, one of the sulphanilimide preparations should be given at once.

The patient must be in bed, and the position should often be changed so as to prevent hypostatic congestion. The room should be well ventilated, but without draughts, and the temperature kept at 65° F. both night and day. In the early stages the air may be moistened by a steam kettle, but the use of a tent is generally to be avoided. Poultices are now less generally employed than formerly, especially for children, and a light pneumonia jacket of Gamgee tissue is usually preferred. The diet should be restricted to fluids and semi-solids, as in pneumonia. Stimulants may be given early if the pulse becomes weak, in doses of 10 drops of brandy every 2 hours to infants, and quantities up to 4 or 6 ounces in the 24 hours to old people. The dry, distressing cough at the onset may be loosened by giving a simple alkaline febrifuge mixture, such as liq. ammon. acetat. min. 120, pot. citrat. grs. 10, sod. bicarb. grs. 10, with flavouring agents, such as syrup of tolu and chloroform water. Later, ammon. carb. and tinct. ipecac. may be given, but large doses of expectorants are to be avoided because of their irritant effect on the stomach. Opiates should not be administered except as tinct. opii camphorata or possibly Dover's powder in the early stages. In infants they should not be given at all.

When in infants or children, the bronchi are becoming blocked by the secretion within them, as evidenced by increasing dyspnoea, an emetic should be given. For this purpose tinct. ipecac. or ammon. carb. in emetic doses is the most effective. In old people, ammon. carb. may be given in milk in doses of grs. 10 two or three times a day, and energetic counter-irritation applied to the bases by means of turpentine stupes, dry cupping or strong liniments.

Strychnine either by the mouth or hypodermically is strongly recommended in cases in which the respiratory centre shows signs of failure. It may be pushed, if necessary, to the point of producing slight muscular twitchings. Nikethamide (coramine) or camphor injections and cardiac tonics may be given under the same conditions as in lobar pneumonia. The administration of warmed oxygen may give relief to dyspnoea and distress.

In cases in which resolution is delayed the question of vaccine therapy may be considered. It seems sometimes to be of distinct value.

c. INHALATION, ASPIRATION AND DEGLUTITION BRONCHO-PNEUMONIA

Acute broncho-pneumonic processes may be caused by the inhalation or aspiration of fluid or solid particles, derived from the upper air-passages or from other parts of the lung. To this form the name of aspiration, or inhalation pneumonia is applied. When from any cause food particles are drawn into the bronchi and broncho-pneumonia results, the condition is referred to as deglutition pneumonia. The resultant processes are similar, and are in effect analogous to those caused by other septic or infected foreign bodies inhaled into the bronchi.

Ætiology.—These conditions may occur at any age, but are more common in adult life. They result from septic processes in the mouth, naso-pharynx, larynx or trachea, and from any morbid state leading to anæsthesia of the pharynx, or to difficulty in deglutition. They occur in association with ulcerating growths of the mouth, tongue, tonsil, pharynx or larynx, and after operations for these conditions or upon the nose and throat, including tracheotomy. Aspiration broncho-pneumonia may also result from vomiting during or after the administration of an anæsthetic. Carcinoma of the œsophagus eroding the trachea may be a cause. Diphtheritic or other forms of paralysis, coma from any cause, especially cerebral vascular lesions and uræmia, may lead to the passage of food particles into the air-passages. Other cerebral lesions, such as abscess or tumour and bulbar paralysis, can also produce the same condition. Infected material may be aspirated from diseased to healthy parts of the lung, as in hæmoptysis, abscess, gangrene and bronchiectasis, or after rupture of an empyema into a bronchus.

Pathology.—Any material reaching the air-passages in this manner is certain to be laden with infective micro-organisms, which may induce bronchitis and broncho-pneumonia. Since pyogenic organisms are often present, suppuration is frequent and single or multiple abscesses result, or even gangrene. If the pleura becomes involved, empyema may develop.

Symptoms.—These are in general similar to those of secondary broncho-pneumonia and are superadded to those of the primary condition. There is generally high temperature, sometimes with rigors, cough and expectoration which is occasionally offensive. It may be mixed with food material and with blood. The physical signs are those of bronchitis and widespread broncho-pneumonia.

Complications and Sequelæ.—These are somewhat similar to those of other inhaled foreign bodies, and comprise abscess, gangrene and empyema.

Course.—The course is generally short, owing to the severity of the process and the gravity of the primary cause. In the comparatively rare cases that recover the course may be severe and protracted.

Prognosis.—From the nature of the primary condition and the intensity of the resulting broncho-pneumonia, this is usually grave.

Treatment.—**PROPHYLACTIC.**—The utmost care should be paid to the toilet of the mouth and pharynx in disease of, or operations upon, these parts. In paralysed or unconscious patients it may be necessary to resort to nasal feeding. In hæmoptysis or bronchiectasis the patient should lie rather on the affected side.

The treatment of the developed condition can be only palliative or

symptomatic in many cases. In most instances the general treatment is similar to that of secondary broncho-pneumonia.

d. TUBERCULOUS BRONCHO-PNEUMONIA

This constitutes one form of pulmonary tuberculosis (see Acute Caseous Tuberculosis, p. 1191).

PNEUMONITIS

Definition.—A localised or disseminated inflammatory process involving the whole texture of the lung and bronchial structures in the areas affected.

Ætiology.—Any severe septic infection of the bronchi or lungs may proceed to pneumonitis. The commonest infecting agents are streptococci, especially the hæmolytic and anærobic varieties. The fusi-spirochætal organisms may also cause it. Pneumococci alone seldom lead to pneumonitis, though they may be found in association with other organisms. Pneumonitis is chiefly met with in later adult life, and more in the male sex. It may be produced by the aspiration of foreign bodies. It sometimes occurs in association with bronchiectasis and may be one of the conditions associated with the febrile attacks occurring in that condition.

Pathology.—The affected area is deeply congested, solid and airless. The bronchi may exude pus. Softening is frequent, leading to the formation of one or more abscesses. Empyema may occur as a sequel or complication.

Symptoms.—The onset is usually acute and the general clinical features are identical with those of severe broncho-pneumonia. There is usually troublesome cough, with more or less copious mucopurulent expectoration. Dyspnoea and cyanosis may be marked. There is marked prostration and the patient is almost always gravely ill. The physical signs are usually those of localised pneumonia or broncho-pneumonia. The blood picture is similar to that of abscess of the lung—a leucocytosis with polymorphonuclear preponderance.

Diagnosis is generally established by X-ray examination, showing diffuse dense areas of consolidation, often progressing to abscess formation. The areas may be multiple and the process may spread widely.

Prognosis.—The prognosis is in the main serious, and depends to some extent upon the cause. Many cases, however, recover under treatment either with or without abscess formation.

Treatment is in the first instance that of broncho-pneumonia. When abscess formation occurs, postural drainage, adapted to the situation of the abscess or abscesses, should at once be adopted.

A *simple pneumonitis* has been described in children by Gill. The symptoms are cough, anorexia and loss of weight. On physical examination one or more areas of impaired percussion note with a few râles are found. Radiological examination reveals opacities in the corresponding situations. These symptoms and signs usually clear up in a few days without treatment.

R. A. YOUNG.

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DISEASES OF THE PLEURA

PLEURISY

Pleurisy or pleuritis is an inflammation of the pleural membrane covering the lung, or of its parietal reflexions.

An ætiological classification, based on the bacteriological findings, would be the most satisfactory one, but is at present impracticable, chiefly owing to the difficulty of establishing the bacteria concerned in many cases. The classification usually adopted depends upon the effects produced. If the process leads only to fibrinous deposit it is described as *dry pleurisy*. If, in addition, much serous fluid is poured out, the condition of *pleurisy with effusion* results, while if pus-formation occurs, the affection is described as *purulent pleurisy* or *empyema*.

It is, however, important to recognise that, although such a classification is convenient from a clinical standpoint, the three conditions are in reality only stages or degrees in the pleural response to irritative or noxious agents. The form occurring in any given case depends upon the nature of the cause, the extent of the infection and the degree of resistance possessed by the individual affected. Further, pleural inflammations may be primary or secondary to local disease or to blood infection, and they may be acute or chronic in course.

A.—ACUTE DRY PLEURISY (ACUTE FIBRINOUS OR PLASTIC PLEURISY)

Ætiology.—This affection may be primary or secondary, the latter being much more common. Even in many cases of so-called idiopathic or primary pleurisy, the condition is in reality secondary to latent or unrecognised disease of the lung or adjacent structures.

Primary dry pleurisy.—Predisposing causes include occupation and climate. Exposure to sudden changes of weather or cold winds, and the necessity of remaining in wet or damp clothing, favour its onset. It is commoner in men, particularly in those of poor physique. It may occur at any age, but is most frequently seen between the ages of 20 and 40 years. Chill seems to be common as a determining cause. It is now certain that the great majority of cases are due to the tubercle bacillus, and that chill or injury is simply concerned in lowering resistance and thus promoting activity of the bacillus. It is possible that some cases may be due to acute rheumatism.

Secondary dry pleurisy.—Dry pleurisy is a frequent complication or concomitant of many diseases of the lungs, notably of pulmonary tuberculosis in any form. It is almost invariably present in lobar pneumonia. It occurs in association with pulmonary collapse, interstitial pulmonary fibrosis, bronchiectasis, abscess, gangrene, infarcts and new growths of the lung. Injuries of the chest-wall, disease of the ribs, chronic nephritis, septicæmia or pyæmia may all be complicated by acute dry pleurisy.

Pathology.—The inflamed area is often localised, but the process may be

widespread or even involve the whole pleural surface. Either the visceral or parietal layer may be first affected, but as a rule both become involved. There is at first hyperæmia with exudation of serum into the subpleural connective tissue. The pleura then appears slightly dull or matt, instead of shiny. Further exudation leads to the deposit of fibrin on the roughened pleural surfaces in the form of a thin false membrane, which often presents a rough or even shaggy appearance. This membrane consists of fibrin entangling leucocytes, a few red blood corpuscles and desquamated epithelial cells.

During the process of resolution, localised adhesions commonly form, but this is not invariable, and a patch of thickening without adhesion may be the ultimate result.

Symptoms.—The onset is usually sudden with acute pain in the side, often described by the patient as a "stitch." Occasionally a sense of malaise may precede the development of the pain by a few hours or even days, but this is not the rule. The pain is aggravated by deep inspiration, by coughing or even by movement. Cough is generally an early symptom, and it is characteristically short, dry, ineffective and distressing. The temperature is usually raised, but, as a rule, only to 100° or 101° F., and some cases are practically apyrexial. In secondary pleurisy these symptoms are added to those of the primary condition.

The decubitus is variable. The patient may lie on the affected side, but in some cases this aggravates the pain, and it is more comfortable to lie on the back or slightly turned towards the sound side. There is diminished movement on the affected side, and breathing may be rapid, although not dyspnoëic. On palpation, vocal fremitus is unaffected, but local tenderness is sometimes elicited, and occasionally a friction fremitus may be felt. The breath-sounds are generally unaltered, but they may be short or jerky in the neighbourhood of the lesion. The characteristic sign of dry pleurisy is the friction rub. This is typically a creaking, rubbing or leathery sound heard towards the end of inspiration and sometimes at the beginning of expiration. In the early stages there may be fine crepitant friction sounds only at the end of inspiration. These are very similar in character to intrapulmonary crepitations and can only be distinguished by their association with local pain, and by being unaltered by cough. Pleural friction sounds may be localised to a small area, and may not be present with every respiration. They may sometimes be brought out again after disappearance by moving the arm, or by taking a deep breath. The voice-sounds are not altered.

Complications and Sequelæ.—Dry pleurisy may proceed to effusion, or may lead to pleural adhesion, and this in turn may result in interstitial pulmonary fibrosis. The most common sequel is pulmonary tuberculosis, sometimes after an interval of years, the explanation being that the original pleurisy is frequently tuberculous. Aching pain in the side with some dyspnoëa may be a temporary sequel of dry pleurisy.

Course.—The temperature usually subsides in 2 or 3 days, the pain in the side and cough disappear, and convalescence is rapid, unless effusion occurs.

Diagnosis.—The differentiation of dry pleurisy from the other causes of pain produced in, or referred to, the chest-wall is not always easy and

requires careful observation of the case. The distinction is important, since an erroneous diagnosis of pleurisy may arouse a suspicion of tuberculosis in subsequent febrile diseases. In the conditions comprised in the term pleurodynia, which include fibrositis of the intercostal muscles and membranes, the pain is increased by deep inspiration, by other muscular movements, and by local pressure, but there is no rise of temperature and pleural friction is not present. In intercostal neuralgia, the pain follows the course of the nerve and is often periodic in character. It may be influenced by movement, but is less affected by respiration than that of pleurisy. There may be tenderness and hyperalgesia over the points of exit of the posterior primary, lateral or anterior cutaneous branches of the nerve affected. Similar manifestations may occur at the onset of acute posterior ganglionitis or herpes zoster. Other conditions inducing pain referred to the chest-wall are tumours or aneurysm pressing on the intercostal nerves, malignant disease of the spinal cord or of its membranes, and caries of the vertebræ. Where the pain lasts more than a few days, and no friction is heard, these conditions should be borne in mind.

Occasionally adventitious sounds of extra-pleural origin may give rise to some difficulty. Contraction of the muscles of the chest may cause a muscular "susurrus"; grating sounds may be produced in the shoulder-joint or in the fascial planes of the back muscles. The origin of these sounds can usually be determined by causing the patient to cease breathing while carrying out movements of the shoulder or back muscles. Occasionally true friction sounds may have a cardiac rhythm as well as a respiratory one, when the area of pleura involved is near the pericardium. It is then referred to as pleuro-pericardial friction.

Having established the evidence of dry pleurisy, a careful search should be made for some primary condition before regarding the case as one of simple primary dry pleurisy. Pulmonary tuberculosis, pneumonia, bronchiectasis and the other causes mentioned above should be considered and excluded.

Prognosis.—The immediate prognosis is good, but as has been mentioned already, the condition may be of tuberculous origin, and eventually be followed by active disease of the lung.

Treatment.—The patient should be kept in bed, no matter how mild the attack. The diet should be fluid or semi-fluid, especially if more than a moderate degree of fever occurs. The pain can often be relieved by strapping the affected side. Strips of adhesive plaster are applied from the sternum to the vertebræ, beginning from below and working upwards. Occasionally this fails to afford relief and may even induce dyspnoea. As alternatives, a local application of tincture or ointment of iodine, a mustard leaf, capsicum ointment or small flying blisters may be employed. Leeches may also give relief in severe cases. Sometimes the pain is so intense that a small injection of heroin or morphine is necessary. A small artificial pneumothorax has been suggested as a means of separating the inflamed surfaces and giving relief to the pain in severe cases. A dose of Dover's powder is useful in the early stage to ensure a night's rest. The irritative cough is often relieved by strapping, and a sedative linctus or lozenge may be a comfort to the patient. An aperient is usually advisable. As a rule no other drugs are necessary, but in cases suspected to be due to rheumatism, salicylates and alkalis should

be administered. Convalescence is usually rapid, but the patient should not be allowed to resume work until fully restored to health, and if a tuberculous origin is suspected prolonged treatment on sanatorium lines should be advised.

Certain localisations of dry pleurisy require separate notice. These are the diaphragmatic and interlobar forms.

DIAPHRAGMATIC ACUTE DRY PLEURISY

Ætiology.—This affection may occur primarily under conditions similar to those causing dry pleurisy in other parts; not infrequently it is secondary to pathological changes in the abdomen. Thus hepatic cirrhosis, perihepatitis, perisplenitis, perinephric suppuration or peritonitis may lead to a spread of infection through the diaphragm to the adjacent pleura. It may also occur as a localised variety of secondary dry pleurisy, when the primary lesion is situated near the base of the lungs.

Symptoms.—Pain is usually very severe and may be referred to the shoulder or to the abdomen. The former is caused by nociceptive impulses ascending the phrenic nerve to its origin in the third to the fifth cervical segments of the spinal cord, leading to pain and hyperæsthesia referred to the cutaneous area of distribution of the fourth cervical root, at the summit of the shoulder. The abdominal pain is in the epigastric and hypochondriac regions, and in addition there is a localised tender spot, known as the “bouton diaphragmatique” of Guéneau de Mussy. This is situated in the subcostal plane, about 2 inches from the mid-line. The diaphragm is nearly motionless on the affected side, and there is often some rigidity of the corresponding upper abdominal muscles. Hiccough may be a noticeable and troublesome symptom. The diaphragm, being nearly fixed in the inspiratory position, may cause a slight downward displacement of the liver if the pleurisy is on the right side. A pleural friction rub is rarely heard, the only abnormal signs commonly present being diminution of air entry, and possibly slight dullness over the corresponding lower lobe of the lung.

Diagnosis.—This is often difficult, owing to the fact that the severity of the symptoms and their localisation frequently suggest the occurrence of some acute abdominal catastrophe such as perforation of a hollow viscus. The abdomen should be most carefully examined in every case. The history, the collapsed state of the patient and the evidence of free gas in the peritoneal cavity in perforation may assist in distinguishing between these conditions.

Treatment.—This is similar to that of simple dry pleurisy elsewhere, save that morphine should be withheld until the diagnosis is conclusively established.

INTERLOBAR DRY PLEURISY

Just as inflammation may be limited to the diaphragmatic portion of the pleura, so the membrane in the cleft between two lobes of the lung may be alone affected. This does not give rise to definite symptoms and signs by which it can be diagnosed during life, though its effects are not infrequently seen in X-ray films. It is frequently discovered on autopsy, but is generally secondary to pulmonary tuberculosis or pneumonia, and there is usually

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evidence of pleurisy elsewhere. It only assumes clinical importance when followed by effusion, and this condition is considered later.

B.—CHRONIC DRY PLEURISY

Under this heading a variety of conditions are included. Strictly it should be restricted to those rare cases, probably usually tuberculous in origin, in which the signs of dry pleurisy persist for long periods, or recur at frequent intervals. In such cases coarse dry friction may be heard over large areas of one lung, often with little or no accompanying pain.

Pleural adhesion and thickening are usually included in the group of chronic dry pleurisy. There may be no symptoms, or at most slight dyspnoea on exertion, with aching or pain on straining, or on lifting weights. Signs suggesting adhesion are local flattening and limitation of movement of the chest-wall. Litten's sign is also absent or diminished when the adhesion is basic, that is, the shadow cast by the movement of the diaphragm, best seen in the region of the seventh and eighth ribs in the anterior and mid-axillary lines, is not present or is much restricted. The vocal fremitus may be diminished and the percussion note impaired. The breath-sounds are often slightly weaker, and the voice-sounds may be diminished over the area where the thickening or adhesion exists.

Chronic diaphragmatic pleurisy or adhesion may give rise to a group of symptoms simulating chronic gastric ulcer. There is pain in the hypochondrium extending through to the back and aggravated by food. Radiographic examination may be of value in demonstrating limitation of movement of one cupola of the diaphragm, together with an angularity due to alteration of its normal contour. Investigation of the gastric functions may also prove of value in diagnosis.

The treatment of chronic dry pleurisy is mainly symptomatic.

C.—PLEURISY WITH EFFUSION

Many cases of pleurisy, possibly the majority, proceed to effusion. The effusion is usually serous in character, but may be hæmorrhagic. Inflammatory effusions must be distinguished from passive transudates, which will be considered separately under the heading of hydrothorax.

SERO-FIBRINOUS PLEURISY

Ætiology.—This is in the main identical with that of dry pleurisy, of which it is, in effect, a later stage. It has now been established that the majority of cases of sero-fibrinous pleurisy are due to the tubercle bacillus. The evidence on which this conclusion has been arrived at is—(1) the subsequent history of the cases shows that a considerable proportion develop active lung signs within 5 years; (2) the cytological and bacteriological examination of the exudate; (3) post-mortem examination of fatal cases; (4) the results of tuberculin reactions.

Other conditions which may give rise to serous effusions are lobar and lobular pneumonia, pulmonary infarcts and new growth. It may also occur

in the course of generalised infections such as the enteric group, acute rheumatism, and septicæmia due to streptococci or staphylococci. In most of these conditions the exudate often becomes purulent. Inflammatory serous effusion may also occur as a complication of severe anæmias, leukæmia, chronic nephritis, injury to the chest-wall and inflammatory conditions below the diaphragm or in the pericardium. It is also a common feature of polyorrhœnitis.

Pathology.—The affection commences with dry pleurisy, spreading over the visceral and parietal pleura, the fibrinous exudate soon forming a thick rough layer on the surface. Further exudation of fluid occurs and accumulates in the pleural cavity, the lung collapsing *pari passu* to accommodate it. Owing to the hilar attachment of the lung, it retracts upwards and inwards, allowing the fluid to accumulate at the bases and in the axillary region, where it reaches its highest level, unless previously existing adhesions prevent it. The lung retracts in this way owing to its elasticity, until the pleural negative pressure is completely abolished. In like manner the mediastinal contents, including the heart, are displaced away from the affected side. If fluid continues to be effused after the lung has retracted to the full extent, and after the negative pressure has become abolished, a positive pressure is produced. The lung is now compressed, and the diaphragm with the liver and spleen are pushed down, while the mediastinal structures are now displaced further towards the sound side. In long-standing cases, the lung may undergo the change known as carnification, as the result of the compression apneumatoxis. The lung appears dark red or slaty grey in colour, is firm, airless and heavier than water. If old adhesions are present, the effused fluid may be loculated and the collapse of the lung may be only partial.

If there is much positive pressure collateral hyperæmia of the sound lung may result and progress to œdema. The fluid in the pleural cavity is pale and clear; it often coagulates after withdrawal. Its characters are further described on page 1252. The quantity may amount to as much as 5 or 6 pints.

Symptoms.—The onset is usually similar to that of dry pleurisy, but the constitutional symptoms are often more marked. There may be an initial rigor, but as a rule pain and dry cough are the earliest symptoms. The fever is of moderate degree, although it may reach 103° F. or more. When effusion develops the pain is often relieved owing to the separation of the inflamed pleural surfaces. If a large quantity of fluid is poured out rapidly, distress of another kind becomes apparent, namely dyspnoea caused by the mechanical effects of the fluid, collapsing the lung and dislocating the mediastinum. In more slowly developing effusions there may be little or no dyspnoea, except on exertion. Expectoration is not common, unless there is co-existent pulmonary disease, or unless œdema of the sound lung develops.

The patient often lies on the affected side or may be propped up in bed. Cyanosis is not a marked feature even in large effusions, unless there is collateral hyperæmia of the sound side. There is generally some prominence on the side of the effusion, but the intercostal spaces are rarely bulged. Movement is restricted or absent in the lower part of the chest on the affected side, although with a moderate effusion the apical region may still expand. The cardiac pulsations may be seen in an abnormal position, the impulse being displaced away from the side of the fluid. In left-sided effusions, the

pulsation may be most marked in the fourth space on the right side as far out as the nipple line. On palpation, the position of the impulse should be verified, and then the amount of chest movement and the character of the vocal fremitus determined. The latter is diminished or completely absent over an effusion of moderate or large size, although it may be obtained over the area where the collapsed or relaxed lung is in contact with the chest-wall. The percussion note over the fluid is one of stony dullness, and the sense of resistance is greatly increased. The exact limits of this area of dullness should be determined with the patient sitting up and recumbent. With moderate effusions the upper level is usually found to assume a curved line, with the summit in the mid-axilla; this is known as the S-shaped curve of Ellis or Damoiseau's line. In the recumbent position, a change in the level of this line may be observed, particularly in the front of the chest and in the axilla. This shifting dullness forms one of the pathognomonic signs of fluid, but it is not always easy to demonstrate. It is much more apparent in cases of pyo-pneumothorax. In large effusions, the dullness may extend up to the level of the clavicle and reach across the mid-line of the sternum; moreover, in left-sided effusions it blends with the cardiac dullness, and the area of gastric resonance, known as Traube's space, may be encroached on or obliterated. The relaxed lung above the effusion in front often yields a skodaic note, which becomes dull if the quantity of fluid increases. At the back there is a triangular area of relative or moderate dullness above the stony dull area of fluid. This is known as Garland's dull triangle. It also corresponds with the relaxed or collapsed lung. At the extreme base on the contralateral side there is often a small area of dullness known as Grocco's triangle. The apex is usually at the vertebral column, about the upper level of the effusion, the base extends outwards at the lower margin of the lung for 1 or 2 inches. This paravertebral dull area is believed to be due to mediastinal displacement by the effusion. Elsewhere over the sound lung the note may be slightly hyper-resonant. The area of deep cardiac dullness should be carefully marked out. In left-sided effusions it is displaced to the right and extends beyond the sternum in the third and fourth spaces, even to the nipple line or beyond it. In right-sided effusions, the displacement may be very obvious, the left margin of the dullness extending as far out as the left mid-axillary line. The auscultatory signs are very variable, and much less characteristic than those obtained by palpation and percussion. In some cases, the breath-sounds over the dull area are distant and weak or even absent, in others they are loud and bronchial or tubular. This inconstancy probably depends upon the extent of pulmonary collapse and the degree of patency of the bronchi. With marked collapse and patent bronchi, bronchial breathing is heard; with partial collapse and obstructed bronchi, the breath-sounds are almost or quite abolished. As a rule, no adventitious sounds are heard, but râles may be audible in the lung above the effusion. Conduction of spoken voice is diminished or abolished, but towards the upper part of the effusion and just above it, the sound produced is heard distantly and with a peculiar nasal or bleating twang, a condition known as ægophony. Baccelli stated that the whispered voice is conducted through a serous but not through a purulent effusion, and called this sign "pectoriloque aphonique," but no reliance can be placed upon this as a diagnostic sign. The breath-sounds heard under the clavicle over the relaxed

lung above the effusion are frequently harsh or puerile. In the contralateral lung the breath-sounds may be vesicular or exaggerated, and in cases of large effusions, where there is marked circulatory obstruction, there are frequently signs of congestion or oedema at the base. Similarly pressure on the descending thoracic aorta may cause lowering of the blood-pressure in the leg as compared with that in the arm (O. K. Williamson). There may be a systolic murmur over the cardiac region (displacement murmur). The abdomen should be examined to determine any downward displacement of the liver or spleen. The blood count in sero-fibrinous pleurisy rarely shows any leucocytosis, apart from complications.

Complications and Sequelæ.—Acute oedema with albuminous ex-
pectoration is rare, but is a dangerous condition unless treatment is prompt. Permanent collapse and carnification of the lung may remain after absorption in prolonged cases, and may progress to diffuse interstitial fibrosis. More commonly some degree of pleural thickening and adhesion persists, and expansion of the lower lobe may never be completely restored. Sero-fibrinous effusion due to tuberculosis rarely becomes purulent, but this sequence is common in other forms. Tracking of the fluid externally through the chest-wall and rupture through the lung occur but rarely. An infrequent complication is hemiplegia, probably due to an embolus derived from a thrombus originating in a pulmonary vein. Miliary tuberculosis occasionally follows rapidly on an effusion; more commonly active tuberculosis of the lungs occurs after a lapse of some years.

Course.—In effusions of moderate size the temperature usually subsides in from 7 to 10 days, and spontaneous absorption is complete in 2, 3 or 4 weeks. In large effusions reaching up to the second rib or higher, the course may be less favourable. The fever may persist even for weeks, and absorption of the fluid may be slow or wanting entirely. Aspiration may accelerate the resolution, and usually only one tapping is necessary, the fluid left behind being absorbed rapidly. In rare cases fluid reaccumulates quickly after repeated tapplings, and a so-called inexhaustible effusion occurs. In some such patients fluid may remain in the pleura for the rest of life.

Diagnosis.—The recognition of the presence of fluid in the pleural cavity is generally easy, but with small or localised effusions it may be difficult. The most valuable signs are the displacement of the heart, the absence of vocal fremitus, and the stony resistant dullness. The auscultatory signs are of less value, and may even be misleading. The chief conditions which may simulate effusion are fibroid lung with thickened pleura and bronchiectasis, pneumonia, particularly the massive form, malignant disease of the lung, pleura or mediastinum, massive collapse, a large pericardial effusion, and an aneurysm pressing on one or other main bronchus. Subphrenic abscess may also give rise to difficulty (see Empyema). Fibroid disease can usually be recognised, since there is generally flattening and sinking-in of the affected side instead of bulging. The heart, if displaced, is drawn towards instead of away from the affected side, vocal fremitus is present although possibly diminished, and the dullness is rarely of the stony character obtained over fluid. The breath-sounds are generally weak, and if bronchiectasis is also present, the characteristically variable signs of that condition should be helpful in diagnosis. In massive pneumonia the differentiation may be difficult, since breath-sounds and voice-sounds are sometimes completely

absent, but the position of the cardiac impulse is generally of decisive importance. In malignant disease and aneurysm, careful observation should afford diagnostic indications, such as glandular enlargement or abnormal pulsation, and in both instances the X-rays may establish the diagnosis. Malignant disease of the pleura may first show itself as a pleural effusion; the tendency to recur after tapping, the presence of blood in the effusion, and the onset of emaciation may help to suggest the cause. In massive collapse there is, as a rule, but little difficulty, owing to the displacement of the cardiac impulse to the affected side. In pericardial effusion the shape of the cardiac dullness may be suggestive, and the dislocation of the impulse may indicate the real condition; moreover, the dullness over the lung behind is rarely of extreme degree unless pleural effusion co-exists. In any doubtful case, examination by the X-rays is desirable, since it may give valuable aid in diagnosis. The shadow of serous fluid is generally dense, but does not obscure the rib shadows completely. The upper level is curved and shifts to some extent with the position of the patient. It merges into the shadow of the collapsed lung above. The diaphragm is immobile on the affected side. A further aid to diagnosis consists in exploratory puncture, which has the advantage of establishing the nature of the fluid as well as its presence. The technique of puncture is similar to that of paracentesis described on pages 1253, 1254, save that a 5 or 10 c.c. syringe with a needle long enough to enter the pleura is used instead of an aspirator. The preliminary local anaesthesia by novocain or some similar preparation, with or without adrenaline, should be employed in every case, not only to avoid pain but also to obviate the risk, remote though it be, of pleural shock. Serous pleural fluid of inflammatory origin varies in colour from pale greenish yellow to brown. The specific gravity is usually 1018 or over. Protein is present as serum albumin, serum globulin and fibrinogen, the total quantity being, as a rule, over 4 per cent. The fluid generally clots spontaneously after withdrawal. The cytology of the fluid is varied, showing lymphocytes, polymorphonuclear cells, erythrocytes and altered endothelial cells in varying proportions. A marked preponderance of lymphocytes is very suggestive of a tuberculous origin, while the presence of large numbers of polymorphonuclear cells is usually an indication of some other infection, generally by a pyogenic organism. In rare cases large numbers of eosinophils have been found. The origin of these cases of so-called "eosinophil pleurisy" is at present doubtful. Cultural examination of tuberculous fluid usually proves sterile unless Loewenstein's medium is used, but in fluid from other causes the infecting organism can often be grown. To establish the tuberculous nature of a pleural fluid, inoculation of 15 c.c. of the fluid into a guinea-pig may be tried. Other methods formerly employed were examination of the centrifugate from the fluid, and Jousset's "inoscopy," which consists in examination of the clots derived from the fluid after they have been submitted to artificial gastric digestion. These two methods, however, fail in many cases. The methods of differentiation of an inflammatory exudate from a passive transudate are given on page 1259.

Prognosis.—The immediate prognosis is good, although with large effusions of 4 pints or more, sudden death sometimes occurs from acute oedema of the lungs, cardiac failure or embolism. The ultimate result depends on the cause. In non-tuberculous effusions, recovery may be complete, save for

pleural adhesion, or they may progress to empyema. In tuberculous effusions arrest may remain complete, but, as already stated, a considerable proportion of the cases develop active pulmonary disease in after years.

Treatment.—The patient should be kept in bed in an airy and well-ventilated room until the temperature is normal. Fluid should be restricted, and the diet may be salt-free with advantage. The administration of diuretics, diaphoretics and saline or mercurial aperients may assist in the disappearance of the exudate. The use of iodide of potassium has been recommended, but it is of doubtful value in these cases. The application of counter-irritants to the chest-wall in the form of iodine or of fly blisters is often helpful. A sedative lozenge or linctus may be given for the irritating cough present in the early stage. Exploratory puncture is generally advisable to permit the examination of the fluid. Opinions differ somewhat as to the indications for paracentesis, which, however, is nowadays performed earlier and more frequently than was formerly the case. It is unnecessary in cases in which absorption of the fluid is apparent within 10 days. The following conditions may be considered to suggest its employment: (1) if the effusion is large and causing positive pressure, as shown by dullness up to the clavicle, marked dyspnoea, downward displacement of the liver or spleen, and collateral hyperæmia of the sound lung; (2) if absorption is slow, the fluid remaining at the same level for a fortnight or more; (3) if acute oedema with albuminous expectoration occurs; (4) in cases of bilateral effusion with increasing dyspnoea, the side with the larger effusion may be aspirated. Paracentesis can be performed in various ways. The simplest method is that of siphonage; a long rubber tube filled with sterile saline solution is attached to a trocar and cannula, which are passed into the pleural cavity and the fluid is siphoned into a receptacle at a lower level. This method has the great advantages that the degree of suction employed is under control, and the lung expands gradually as the fluid is withdrawn. It is often difficult to remove a large quantity of fluid by this means, and it fails in loculated effusion. Aspiration is more generally effective, and may be carried out either by Dieulafoy's pump and two-way tap, Martin's syringe, or by Potain's apparatus. With these methods it is impossible to withdraw all the fluid, and removal with air replacement is now often practised with advantage. For this purpose an aspirator and an apparatus like that used in the induction of artificial pneumothorax are required. This method permits of almost complete removal of the fluid, prevents cough and discomfort, lessens the tendency to recurrence of the effusion and promotes expansion of the lung. In performing aspiration the patient should sit up in bed, or lie slightly turned on the unaffected side, and the area for operation should be painted with iodine. The skin and muscles down to the pleura should be anæsthetised with procaine (novocain) or other local anæsthetic, and a small incision made through the skin, though this is not essential in the case of a small instrument. The trocar and cannula are then pushed carefully into the pleural cavity just above a rib to avoid puncturing the intercostal artery. The sites chosen depend on the situation of the fluid, but the most convenient are in the sixth space in the mid-axilla, the seventh space in the posterior axillary line, or the eighth space just below the angle of the scapula. Aspiration should be stopped if cough occurs, if pain is severe, or if albuminous expectoration with signs of oedema supervenes. In rare cases sudden death from pleural

shock has occurred. The risk of this may be obviated by careful local anaesthesia down to the pleural level. Other risks are due to faulty technique, and comprise entrance of air into the pleural cavity from wrong connection of the apparatus or from wounding the lung, and infection of the pleural cavity from failure in the aseptic preparations leading to empyema. Air replacement seems preferable, since it allows of almost complete removal of the fluid.

After absorption or removal of the fluid, re-expansion of the lung may be promoted by the use of Wolff's bottles, or by appropriate breathing exercises. With the former, fluid is forced from one bottle to another by blowing. In the latter, the patient takes deep inspirations while seated in a chair with the sound side partly fixed. In all cases in which a tuberculous origin is proved or suspected, prolonged convalescent treatment on sanatorium principles is advisable.

ANOMALOUS PLEURAL EFFUSIONS

Two unusual forms of pleural effusion require brief mention—they are encysted interlobar and encysted diaphragmatic sero-fibrinous pleurisy. The former of these can only be recognised by X-ray examination followed by exploratory puncture. Encysted diaphragmatic sero-fibrinous pleurisy is rare, but a case has been erroneously recorded as acute serous mediastinitis. This condition is simply one of pleural effusion localised to the space between the mediastinal pleura, the diaphragm and the lung. Both of these conditions, if diagnosed, should be treated on general principles. The effusion may absorb spontaneously, but if not, aspiration may be necessary.

PURULENT PLEURISY (EMPHYEMA)

In this condition the pleural exudate becomes purulent. The fluid may be turbid and the presence of pus be apparent only on microscopical examination, or it may consist of typical pus.

Ætiology.—**PREDISPOSING CAUSES.**—Empyema is common in children under 10 years of age, and the younger the child the greater the probability that any effusion will be purulent. In adults it is commonest between the ages of 20 and 40 years, probably owing to the heavy incidence of pneumonia in this age period. Debility, exposure and alcoholism may promote its occurrence. Purulent pleurisy is but rarely primary, except in the form due to the pneumococcus. It is most commonly due to extension from the lungs, especially from lobar pneumonia and from bronchopneumonia. Other pulmonary causes are tuberculosis, bronchiectasis, abscess, gangrene, new-growth, or septic infarcts in infective endocarditis. It may develop in association with mediastinal lesions, such as suppurating glands, ulcerating carcinoma of the œsophagus, or from suppuration in the neck tracking downwards. Infection of the pleura may occur through the chest-wall as a result of gunshot wounds, stabs, fractured ribs, and faulty technique in aspiration of a serous effusion. The primary source of pleural infection may be in the abdomen, the organisms passing through the diaphragm from a perinephric, subphrenic, or hepatic abscess, or from localised or generalised peritonitis consequent on rupture of a gastric or duodenal ulcer. The involve-

ment of the pleura may take place through the blood in septicæmia, suppurating gunshot wounds, compound fracture of the femur, and in otitis media with lateral sinus thrombosis.

Empyema may develop during the course of many of the acute specific fevers, such as scarlet fever, variola, measles and the enteric group; but since in these conditions it is usually secondary to broncho-pneumonia, it belongs strictly to the pulmonary group.

EXCITING CAUSES.—The organisms most frequently found in purulent effusions are the pneumococcus and the streptococcus, the former accounting for more than half of the cases. Occasionally the pus proves to be sterile on culture; such cases are generally the result of the tubercle bacillus or of a pneumococcus which has died out. Other organisms less commonly found are staphylococci, Pfeiffer's *H. influenzae*, the *B. typhosus*, and Friedländer's pneumo-bacillus. Streptothrix organisms are occasionally found (see Actinomycosis), also various saprophytes and anaerobic organisms, especially in fetid empyema.

Pathology.—The initial stages are similar to those of dry and sero-fibrinous pleurisy, but when the effusion occurs, it proves to be rich in leucocytes undergoing disintegration and to contain the infecting organism. It varies from a slightly turbid semi-translucent fluid to typical thick, opaque, creamy pus. Its colour ranges from pale amber to green or greenish grey. It may be odourless or extremely offensive. In cases secondary to gangrene, it may be thin and horribly fetid, while in pneumococcal cases it may be curdy and of slightly sweetish odour. The pleura is covered with a more or less thick layer of sodden fibrinous exudate. In cases due to the pneumococcus this false membrane may be very thick. Adhesions form quickly, leading to encystment or loculation of the pus. Such adhesions also prevent the lung from expanding after evacuation of the pus, with the result that the lung becomes carnified and interstitial fibrosis results. There is usually some enlargement of the bronchial glands. In long-standing cases there may be lardaceous disease of the liver, spleen, kidneys and intestines.

Symptoms.—Since empyema usually develops in the course of, or as a sequel of, some other disease, its symptoms are often masked by those of the primary disease and may easily be overlooked. In primary cases due to the pneumococcus the onset may be like that of pneumonia; in the more common secondary cases a rise of temperature and increase of signs develop after the crisis. In general it may be stated that the symptoms are similar to those of sero-fibrinous pleurisy, but more severe. There is more malaise, and the patient may appear profoundly ill, with rigors, sweats and dyspnoea. The temperature ranges higher, up to 103° F. or more, and may be of septic type with marked daily remissions, but some cases are almost if not completely apyrexial. The signs are usually exactly similar to those of sero-fibrinous effusion, but in some instances special features may be noticed. In neglected or prolonged cases, wasting, pallor and cachexia become marked. The intercostal spaces may be found to bulge, and œdema of the chest-wall is sometimes apparent. The pus may track through an intercostal space, generally the fifth near the nipple, producing a fluctuating swelling known as a pointing empyema or *empyema necessitatis*. This may infiltrate the skin and simulate a superficial abscess. The swelling so induced may pulsate, especially if it be on the left side—a condition known as pulsating empyema.

Pulsation communicated to the chest-wall may also be observed in some large left-sided purulent effusions without local swelling. The displacement of the liver or spleen may be greater than with serous effusions, probably owing to the higher specific gravity of the pus, which is usually 1030 or more, and to the associated inflammation of the diaphragm. In fetid empyema, the breath and sputum may be offensive, even before rupture into a bronchus occurs. Clubbing of the fingers and toes occurs in empyema of long standing, but may develop in a few weeks. Blood examination reveals a moderate leucocytosis in the majority of cases. Counts of 15,000 leucocytes per cubic millimetre are usual, and in some instances figures up to 100,000 per cubic millimetre are obtained.

Complications and Sequelæ.—In neglected or untreated empyema the pus may track and become discharged in various directions. The commonest is rupture through the visceral pleura into the lung and discharge through a bronchus. This may lead to sudden death from suffocation; on the other hand, in small empyemata spontaneous cure may follow this evacuation of the pus. In other instances pyo-pneumothorax results, and occasionally gangrene of the lung. A second method of discharge is through the chest-wall, as an *empyema necessitatis*. Perforation may occur into the pericardium, or into the œsophagus with the formation of a pleuro-œsophageal fistula. The diaphragm may be perforated with the production of a subphrenic, lumbar or psoas abscess, while in other cases general peritonitis may ensue.

The pericardium or the mediastinum may become infected without perforation; similarly costal periostitis may be induced. After spontaneous or operative evacuation the cavity may fail to close and a chronic empyema or sinus result. This is generally due to the lung being permanently collapsed and adherent, and therefore failing to expand. It subsequently undergoes fibrosis with development of bronchiectasis. Sometimes the failure to close may be due to the nature of the infection, particularly when it is due to tuberculosis or actinomycosis (streptotricosis). In other cases it may be due to a bronchial fistula, or to a foreign body in the pleura. Generalised infection is rare, but cerebral abscess, probably of embolic origin, is not very uncommon, especially in cases due to streptococci. Chronic pulmonary osteo-arthritis is an occasional complication, and lardaceous disease sometimes occurs in cases of long duration. Diphtheritic infection of the wound, with subsequent paralysis, has been recorded after operation, more especially in cases secondary to influenzal broncho-pneumonia.

The sequelæ in untreated cases may be fistulæ, such as pleuro-bronchial, pleuro-œsophageal or external, and various deformities. The sequelæ after operation may be a small amount of pleural thickening, or if operation were delayed, and re-expansion incomplete, there is falling-in of the chest, with flattening, dropping of the shoulder and secondary scoliosis. In other cases, as mentioned above, a chronic sinus may result.

Course.—Apart from spontaneous cure of small empyemata by inspissation of the pus, or discharge through a bronchus or through the chest-wall, death generally occurs in untreated cases within a month or two. As in sero-fibrinous pleurisy, sudden death may occur. Death may occur after operation, from exhaustion or from cerebral abscess.

Diagnosis.—The diagnosis of empyema involves two distinct problems—one, the recognition of the presence of fluid in the pleura, which is considered

under sero-fibrinous pleurisy; the other, the demonstration of its purulent character. In spite of the more severe symptoms, empyema is frequently overlooked even by physicians of experience. This is partly due to the fact that its development may be insidious, with signs increasing but little from day to day, and partly to its secondary character, its onset being obscured by the clinical features of the primary condition. It is wise, therefore, to suspect its existence in any case of obscure lung signs, especially those with dullness, cardiac displacement and fever, consequent on pneumonia of any variety.

There are a few special difficulties as compared with sero-fibrinous effusion which merit separate mention. The first of these is subphrenic abscess. This may lead to immobilisation of the diaphragm on one side, more commonly the right, and cause collapse of the lung and even pleural effusion. The difficulty is the greater when the subphrenic abscess contains gas as well as pus. The history, the absence of displacement of the heart's impulse, and examination by X-rays may all assist, but the differentiation is often extremely difficult.

Empyema necessitatis may simulate a tuberculous or other abscess about a rib, and empyema should always be suspected in any case of local fluctuating swelling about the chest-wall. Pulsating empyema requires to be distinguished from aortic aneurysm; the pulsation is less forcible and little, if at all, expansile in the former. The cardiac displacement, the X-rays and cautious exploratory puncture, enable the nature of the condition to be recognised.

In any case in which empyema is suspected three examinations may be undertaken—a blood count, radiographic methods, and exploratory puncture. A polymorphonuclear leucocytosis of 15,000 per cubic millimetre and over, a dense shadow in the radiogram obscuring the ribs, together with cardiac displacement may be very suggestive, while puncture may prove the presence of pus. Sometimes, however, puncture may fail, although pus is present. This may be due to the pus being too thick to pass through the needle, to loculation of the pus, or to wrong choice of the site for puncture. In this case, if the other signs indicate pus, repeated punctures with a larger needle under anæsthesia are called for, but it is well to be prepared to proceed to operation if pus is found.

Prognosis.—This depends upon the primary cause, the method of treatment adopted, and the duration of the effusion before the operation. The most favourable forms are those due to the pneumococcus, which are recognised and treated at an early stage. In neglected cases, with profound toxæmia, with gangrene of the lung or lardaceous disease, the outlook is extremely grave. Empyemata due to streptococcal infection are serious, unless recognised early; similarly with cases of fetid empyema due to anaerobic infections. Infected hæmothorax consequent on gunshot wounds of the chest is of grave prognosis. The outlook is serious in cases of bilateral empyema, but recovery may follow successive evacuation of the pus on the two sides.

Treatment.—This consists in the evacuation of the pus by operation as soon as the diagnosis is established in pneumococcal cases. In those of streptococcal origin, operation should not be resorted to while the fluid is of thin sero-purulent character, but should be postponed until it is definitely purulent. Premature operation in streptococcal cases has been shown by

the American Empyema Commission to be a very dangerous procedure, since the fluid is not shut off by adhesions and operation may lead to open pneumothorax, with flapping mediastinum. At this stage, the condition is described as pyothorax. A preliminary aspiration is of advantage in large effusions, and may be repeated in streptococcal effusions until they are ready for operation. The operation consists in drainage by removal of a piece of rib subperiosteally and incision of the parietal pleura. For the operation a general anæsthetic may be given, but it is now almost always carried out under local anæsthesia; but if the patient's condition renders this undesirable, an incision under local anæsthesia may be made through an intercostal space and a drainage tube inserted, a piece of rib being removed later under general or local anæsthesia when improvement has occurred. The wound is dressed at least daily and the drainage tube sterilised, every endeavour being made to prevent secondary infections. To this end the pleural cavity may be irrigated daily by the Carrel-Dakin method, or washed out with some antiseptic such as flavine or brilliant green. To avoid pleural shock, free exit for the wash fluids must be ensured. By some authorities pneumococcal empyemata, particularly in young children, are treated by repeated aspirations or by siphon drainage. If the pus is thick and difficult to evacuate, incision of the pleura with immediate suture is performed, any reaccumulation being treated by aspiration with or without oxygen replacement. If, however, toxic symptoms persist, drainage should be effectively established at once.

In cases of chronic empyema, or of sinus failing to close, the question of some plastic operation must be considered. Various forms of operation have been devised, involving removal of portions of many ribs, and the decortication operation of Fowler and Delorme. The general condition of the patient must be carefully considered before these operations are advised. In some cases an autogenous vaccine seems to be of value, if drainage is satisfactory.

SPECIAL VARIETIES OF EMPYEMA.—Certain special localisations of purulent pleurisy require separate consideration, notably apical, interlobar and diaphragmatic empyemata.

Apical empyema.—This condition is usually secondary to apical pneumonia, less commonly to pulmonary tuberculosis. It is one variety of encysted empyema, the pus being shut off from the rest of the pleural cavity by adhesions. The symptoms and signs are not characteristic, but may be suggestive. There is very marked dullness below the clavicle, not transgressing the middle line, with weak or absent breath-sounds, and possibly some indications of mediastinal displacement. Diagnosis can, as a rule, be established only by the X-rays and exploratory puncture, the latter being carried out in the second space near the mid-clavicular line. The treatment consists in drainage by incision as near the lower limit of the effusion as possible.

Interlobar empyema.—Pus collecting between two of the lobes may be difficult to differentiate from pulmonary abscess, gangrene and bronchiectasis. It is often not diagnosed until rupture into a bronchus draws attention to it. The signs are generally most marked in the axilla or near the angle of the scapula. They are often slight until rupture occurs, and even then there may be only a small area of dullness in the line of an interlobar fissure, with distant or weak bronchial breathing and a few râles. The pus expectorated may be fetid, and the patient's breath may be offensive a few days

before rupture occurs. The condition simulates abscess of the lung, and may be almost impossible to differentiate from that affection. Examination by the X-rays gives the greatest help in the diagnosis. Recent observations suggest that interlobar empyema is much less common than abscess. The treatment is identical with that for pulmonary abscess.

Diaphragmatic empyema.—The pus is usually encysted, and may be so deeply situated as to give but few signs. The initial symptoms are generally severe, being those of diaphragmatic pleurisy, but hiccough is often a troublesome feature. When pus forms, there may be marked constitutional symptoms, and obscure signs may develop, such as dullness, at a point just above the base behind, with weak or distant bronchial breathing. With such a history and obscure basic signs, especially when they occur after an attack of pneumonia, the use of the X-rays and of the exploring needle should not be neglected. In cases not recognised and treated, rupture into a bronchus or through the diaphragm may occur. The treatment is similar to that for ordinary empyema.

HYDROTHORAX (DROPSY OR HYDROPS OF THE PLEURA)

Hydrothorax is the name applied to a collection of clear fluid in the pleural cavity, the result of passive transudation from the capillaries.

Ætiology.—The commonest cause of hydrothorax is cardiac failure from chronic valvular disease, or from myocardial weakness or degeneration. It occurs in acute and chronic renal disease, under conditions similar to those leading to dropsy in these affections. It is sometimes found in severe anæmias, especially pernicious anæmia. Obstruction to the azygos veins may lead to transudation into one pleural cavity or into both. This obstruction may be induced by pressure from without by a mediastinal or pulmonary new-growth, or by internal causes such as thrombosis.

Pathology.—The pathology of hydrothorax is that of dropsy elsewhere. It is produced by mechanical or chemical conditions affecting the blood flow through the capillaries, and it must be distinguished carefully from inflammatory effusion. There is a difference in the composition as well as in the origin of the two kinds of pleural fluid. The characters of inflammatory effusions have been described under pleurisy with effusion. The fluid in hydrothorax is pale yellow in colour, and the specific gravity is 1015 to 1010 or less. It is clear and does not clot after removal. There is little protein, often not more than 1 per cent., but transudates due to local obstruction may contain as much as 3 per cent. The cellular elements are scanty, although some endothelial cells may be present, often united together in plaques. The fluid may be definitely bloodstained, when it is described as hæmo-hydrothorax.

Hydrothorax is usually bilateral in cases due to cardiac or renal disease, but in the former there is often more fluid on the right side, or the fluid may be confined to that side. The explanation of this is somewhat obscure. It has been suggested that it is due to pressure or traction on the vena azygos major by the enlarged right heart, but according to Fetterolf and Landis, a more likely explanation is pressure of the distended right auricle upon the pulmonary veins. Fluid may also collect in greater quantity on the side

upon which the patient lies most constantly. In cases with unilateral pleural adhesion, œdema of the lung may occur on that side, while hydrothorax occurs upon the other.

Symptoms.—The symptoms of hydrothorax are generally overshadowed by those of the condition causing it, but the occurrence of dyspnoea and cyanosis in any case of cardiac or renal disease should suggest careful examination of the bases of the lungs. In the absence of inflammatory complications the condition is afebrile. The signs are identical with those of sero-fibrinous pleurisy, save that no friction sounds are audible at any stage. It is, however, more difficult to assess the significance of displacement of the cardiac impulse, owing to the increased size of the heart in the cases of cardiac origin.

Diagnosis.—This depends upon the presence of signs of fluid in the pleura in association with cardiac or renal disease, with absence of fever, and also upon the characters of the fluid withdrawn by puncture or aspiration.

Treatment.—Removal of the fluid may give great relief. It may be necessary to repeat the operation, since the fluid often reaccumulates. The treatment of the primary condition should also be carried out.

HÆMORRHAGIC PLEURAL EFFUSIONS

All fluids poured out into the pleura contain a certain number of red blood corpuscles. It is only when a number sufficient to give a definite red colour are present, that the fluid is regarded as hæmorrhagic.

For convenience of description three forms may be differentiated—

- (1) Hæmorrhagic pleurisy or hæmo-serothorax ; (2) hæmo-hydrothorax ; and (3) hæmothorax.

1. HÆMORRHAGIC PLEURISY.

This is simply a pleurisy with effusion, in which the exudate is blood-stained.

Ætiology.—The usual causes are malignant disease of the lungs, pleura or mediastinum, and rarely tuberculosis of the lung and pleura. Hæmorrhagic pleurisy may occur in association with hepatic cirrhosis, but in this case it is often the result of a terminal tuberculosis. It occurs less frequently in association with blood diseases, such as purpura, and with the malignant or hæmorrhagic varieties of acute infectious fevers such as scarlet fever and small-pox, and occasionally with lobar pneumonia. Sometimes in tapping a sero-fibrinous effusion for the second-time, it is found that the fluid, which was originally clear, is now blood-stained. This is not necessarily an indication of increase in the severity of the process, but may be due to injury of a blood vessel at the first operation.

Symptoms.—The symptoms and signs are identical with those of serous effusion, and the hæmorrhagic character can only be recognised by withdrawal of the fluid. An interesting point is the frequency of excess of eosinophils in these effusions. Diagnosis and treatment are the same as for sero-fibrinous pleurisy.

2. HÆMO-HYDROTHORAX.

This condition has been referred to under hydrothorax. It consists simply in blood-staining of a passive transudate into the pleura.

3. HÆMOTHORAX.

Hæmorrhage into the pleural cavity is the result of injury or disease of the vessels of the lung, mediastinum or chest-wall.

Ætiology.—The chief causes are injury, such as penetrating chest wounds or fracture of the ribs, rupture of an aneurysm, and erosion by new-growth. Experience of the traumatic group has been largely increased during the War of 1914–1918. Hæmothorax was noted in about 70 per cent. of chest wounds.

Pathology.—The effused blood generally comes from the lung vessels, less commonly from the intercostals. It is “whipped” by the movements of the heart and lungs, with the result that fibrin is deposited in layers upon the diaphragmatic pleura, and the parts of the visceral and parietal pleura in contact with the blood. The fluid remaining in the pleura or withdrawn by aspiration is largely defibrinated and therefore does not clot, unless a secondary pleurisy develops.

The lower lobe of the lung on the affected side becomes collapsed and eventually carnified, unless absorption occurs or unless the blood is aspirated. The upper lobe may show some compensatory emphysema, and adhesions may form in the pleura, separating it from the hæmothorax below. When secondary infections of the bronchi or lungs occur, such as bronchitis or broncho-pneumonia, the collapsed lower lobe is not affected.

Symptoms.—The symptoms of hæmorrhage into the pleura from medical causes, such as rupture of an aneurysm or erosion of a large vessel, are collapse and rapid death. When due to disease or injury of an intercostal vessel, they may be insidious and slowly ingravescent until dyspnoea, restlessness and the other indications of internal hæmorrhage develop. When due to injury, similar symptoms occur, but may be masked or overshadowed by the shock, hæmoptysis and cough, induced by the wound of the lung or chest-wall. The signs are those of pleural effusion, but in traumatic cases certain special features may be mentioned. There is a great tendency to retraction of the chest-wall on the affected side, and the cupola of the diaphragm on this side is displaced upwards. This is thought to be due to an active lobar collapse of the lung, the lung contracting, not as the result of the pressure of the effusion, but in consequence of a nervous protective reflex initiated by the trauma. Vocal fremitus is usually diminished or absent. The breath-sounds over the effusion are frequently bronchial, and well-marked bronchophony and pectoriloquy may be present.

Complications and Sequelæ.—The most serious complication is infection of the effusion. This is generally due to organisms introduced at the time of the wound, either by the missile or by portions of the clothing or skin carried in with it. Aerobic organisms, such as a streptococcus, or anaerobic ones, as the *B. aerogenes encapsulatus* or the *B. sporogenes*, may be present. A hæmo-pneumothorax may develop, the gas entering the pleural cavity from the wound in the lung or through the chest-wall. Gas may also be formed by gas-producing infecting organisms in the effusion. Massive collapse may occur in the contralateral lung, or other complications may arise, such as bronchitis, broncho-pneumonia, lobar pneumonia or œdema of the lungs. If the effusion is small and not infected, there are usually no permanent after-effects. In severe cases sequelæ, similar to those of sero-fibrinous pleurisy and empyema, may result.

Course.—This depends upon the cause and size of the hæmothorax, and upon the mode of treatment adopted. It is profoundly and gravely influenced by infection of the effused blood. A small sterile hæmothorax is generally absorbed spontaneously. Medium-sized and large effusions may not disappear unless aspirated. An infected hæmothorax will inevitably prove fatal, if untreated.

Diagnosis.—Hæmothorax should be suspected when basic dullness develops shortly after a gunshot wound of the chest. The mistake that is most likely to be made in such cases is to confuse hæmothorax with lobar pneumonia. The cardiac displacement and the diminution of vocal fremitus over the dull area are the most valuable diagnostic signs. An active lobar collapse is distinguished by the fact that the heart is displaced towards the affected side. The X-rays afford valuable confirmatory evidence in most cases. When air and blood are present, the upper border of the dark area in the radiogram has a sharply defined edge, while the pleural cavity above is very translucent. The use of the exploring syringe generally settles the diagnosis, except in certain cases in which, although a considerable quantity of blood may be present, none is removed by aspiration owing to the needle entering the clot.

Prognosis.—In a sterile hæmothorax due to a chest wound the prognosis is good. If infection occurs, the prognosis depends upon the promptitude with which this condition is recognised and radically treated, although in very acute infections death may occur in 2 or 3 days despite immediate operation.

Treatment.—A small sterile hæmothorax may be left untouched. In medium and large-sized effusions, recovery is accelerated by aspiration. The possible danger of renewal of the hæmorrhage, as the result of lowering the pleural tension by this operation, is very slight, and negligible if it is delayed until 2 or 3 days after the wound. If the temperature in a case of hæmothorax rises suddenly to 102° or 103° F. in the evening, it is criminal to wait until the next morning to see what will happen. A specimen of fluid should be withdrawn and examined microscopically. Direct films may occasionally reveal the presence of organisms, but the important point to determine is the number of polymorphonuclear leucocytes present. When these are numerous, operation should be performed without awaiting the findings of aerobic and anaerobic cultures. A rib should be resected as in empyema, and the blood and clots should be removed from the pleural cavity and drainage established.

CHYLOUS AND OTHER MILKY EFFUSIONS

A milky fluid is occasionally obtained on exploratory puncture or aspiration of a pleural effusion. It is usual to classify such fluids into three groups—(1) Chylothorax; (2) chyloform fluid; (3) pseudo-chyloous fluid.

1. CHYLOTHORAX.

There is an effusion of pure chyle or of serous fluid mixed with chyle.

Ætiology.—Chylothorax is usually the result of injury to, or disease of, the thoracic duct. The traumatic form is, as a rule, secondary to crushing of the chest-wall with fracture of the ribs. In disease, the thoracic duct may be pressed on by a malignant growth or enlarged mediastinal glands,

or the flow may be obstructed by thrombosis of the left subclavian vein. Invasion of the thoracic duct by the *Filaria sanguinis hominis* may also be a cause.

Pathology.—The fluid in true chylothorax is a milky emulsion which remains so on standing, although a cream-like layer may form at the top. With the microscope fat globules can be seen, which stain with the usual fat stains and can be dissolved by ether.

2. CHYLIFORM EFFUSION.

In this condition fat is present, but it is not derived from the thoracic duct.

Ætiology.—Chyliform effusions occur in association with tuberculosis and carcinoma of the pleura or lung.

Pathology.—The fluid is milky and contains fat in emulsion, although in smaller quantities than in true chylothorax. On microscopical examination large fat droplets are seen, and numbers of cells, chiefly leucocytes undergoing fatty degeneration. It is, no doubt, from this process that the fat is derived.

3. PSEUDO-CHYLOUS EFFUSION.

In this condition the milky appearance is not due to fat, but to other particles causing opalescence.

Ætiology.—Pseudo-chylous fluid has been observed in chronic effusions due to heart disease, nephritis, tuberculosis and malignant disease.

Pathology.—The milky appearance is due in some cases to a lecithin globulin complex (Wallis and Schölberg). Other rare causes of milky, opalescent or turbid effusions are the presence of particles of calcium phosphate, cholesterol or filarial embryos. These fluids are distinguished from the above by showing a deposit on standing.

Diagnosis.—This can only be established by microscopical and chemical investigation of the fluid withdrawn.

Prognosis.—The prognosis in most cases of milky effusions is serious, owing to the gravity of the primary condition. Some traumatic cases of true chylothorax recover.

Treatment.—The treatment is for the most part symptomatic and dependent upon the primary condition. In true chylothorax, removal of the fluid is inadvisable, unless it is causing dyspnoea or other symptoms of pressure. The drain of fat caused by it is a serious loss, especially if the tapping has to be repeated frequently. In chyliform effusions there is a marked tendency to recur after removal of the fluid.

PNEUMOTHORAX

In pneumothorax, gas, usually air, collects between the layers of the pleura, which now becomes a real instead of a potential space. When serous fluid is present as well as the gas it is called a hydro-pneumothorax, when pus forms the condition is described as pyo-pneumothorax, and when blood and gas collect the term hæmo-pneumothorax is applied.

Ætiology.—Pneumothorax is more common in men, and the maximum incidence is between the ages of 20 and 40 years, but it may occur at any age. The air may gain access to the pleural cavity in the following ways:

(1) Through the visceral pleura from the air in the lungs and bronchi. This

accounts for 95 per cent. or more of the cases. The commonest cause is rupture of a subpleural tuberculous focus. Rupture of an empyema into the lung is the next most frequent antecedent condition. Other pulmonary causes are gangrene, abscess, septic infarct, bronchiectasis, new growths of lung and pleura, and rupture of an emphysematous bulla or vesicle. Puncture of the lung during paracentesis, or rupture of the pleura over a diseased focus, owing to rapid expansion of the lung during the same operation, may lead to pneumothorax. A broken rib perforating the lung can also induce it. It may occur as a complication of artificial pneumothorax treatment, especially when this is bilateral. (2) Through the chest-wall, as a result of penetrating wounds, although pneumothorax is not a common result. An abscess in the chest-wall opening externally and through the pleura, or a discharging *empyema necessitatis*, may be a cause. (3) Through the mediastinum, by ulceration of an œsophageal growth, or of a diseased bronchial gland, into the pleura, or from accidental perforation of the œsophagus during the passage of an œsophageal bougie or œsophagoscope. (4) Through the diaphragm, from some hollow abdominal viscus, *e.g.* an ulcer of the stomach or duodenum may perforate, leading to the formation of a subphrenic abscess, which in turn may break through the diaphragm into the pleura. (5) Gas may accumulate in the pleura owing to infection of a pleural effusion by gas-producing organisms. This is generally the result of wounds.

Sudden spontaneous pneumothorax in apparently healthy persons occurs more commonly than is generally recognised, and is described as simple or benign pneumothorax. The causation is obscure. Rupture of an emphysematous vesicle, or of a latent or healed tuberculous focus, have both been suggested, though the latter is improbable, since there is usually no pleural reaction and the lung rapidly re-expands. In rare cases, however, the collapse of the lung is long-continued and may even be permanent. The condition is often recurrent, and is exceptionally bilateral. Complete recovery is the rule. Very occasionally spontaneous hæmopneumothorax occurs. The symptoms are usually more severe and a fatal result is not uncommon.

The exciting cause of pneumothorax may be physical strain or violent cough, but many cases occur while the patient is at rest or even during sleep.

Pathology.—The entrance of air between the layers of the pleura disturbs the pressure relations in the thorax in a similar way to the effusion of fluid; but whereas with the latter the process is gradual, in pneumothorax it is rapid, and the pressure within the pleura changes from the normal negative figure to that of the atmosphere, often in a few minutes or less. Mediastinal and cardiac displacements like those in pleural effusion, and due to the unopposed traction of the sound side, are also rapidly produced. The subsequent pressure relations depend upon the source of the air. If the opening is in the chest wall, the intrapleural pressure will remain equal to the atmospheric, until the opening becomes closed. If the opening is in the lung, three varieties occur: (1) the opening may remain patent, when the pressure keeps at atmospheric level; (2) the opening may be valvular, permitting the entry of air into the pleura during inspiration, but preventing its escape during expiration. In this case the pressure in the pleura rises above that of the atmosphere, and

the air within it is at a positive pressure, causing further cardiac and mediastinal dislocation with downward displacement of the diaphragm; (3) the opening becomes sealed, and there is a condition of closed pneumothorax in which the pressure may be equal to, greater or less than, that of the atmosphere.

To demonstrate pneumothorax post mortem, the autopsy may be performed under water, or a flap being made of the skin and muscles at the side of the thorax, this may be filled with water before puncturing the intercostal spaces. A third method is to dissect carefully through an intercostal space down to the pleura, when the lung will be found to be retracted. On opening the thorax the appearances vary. If the air entering the pleura is sterile, no inflammatory reaction occurs, the pleura remains shiny and no fluid is formed, the condition being one of simple pneumothorax. More commonly, bacteria gain access to the pleura with the ingoing air, or subsequently through the opening when this remains patent, with the result that either serous fluid or pus collects. In the former case the condition is described as hydro-pneumothorax, in the latter as pyo-pneumothorax. The appearances of the pleural membrane are similar to those found in sero-fibrinous pleurisy and empyema respectively. The lung is collapsed in every case of pneumothorax, and lies retracted towards the hilum and the spine. In tuberculous disease, a caseous focus or small cavity just under the pleura is the most frequent cause. The perforation may be a large circular rent or a small pin-hole, but multiple apertures may occasionally be present. The opening can usually be found, even if small, by submerging the lung under water while pumping air down the trachea. When extensive adhesions are present, the collapse of the lung is largely prevented and the pneumothorax is only partial. In such cases the perforation is frequently near the adhesions. In cases where fluid is present the diaphragm may be seen to be depressed on the affected side and its curvature lessened or reversed.

Symptoms.—In a considerable proportion of cases the onset is sudden and the condition of the patient becomes alarming at once. On the other hand, pneumothorax may develop insidiously, with surprisingly little pain and dyspnoea, so that its occurrence may be overlooked or only discovered on routine physical and radiographic examination, including a lateral film. This is more likely to be the case when perforation occurs in a lung extensively diseased or when the aperture is small, and the leak of air is slow. In the acute form of onset the patient is seized with severe pain while coughing or engaged in some extra exertion. There is often a feeling of "something having given way," and at once great dyspnoea develops with signs of collapse and severe mental anguish. The patient may appear blue, cold and clammy, breathing is rapid and shallow, the temperature falls to subnormal, the heart beats quickly and the pulse becomes small and weak. The patient is often restless, very alarmed and unable or afraid to speak. Occasionally death occurs in a few minutes. As a rule, the more acute symptoms subside in a few hours, but the temperature rises and the rapid breathing usually persists for some time. On examination the patient will usually be found sitting up, with *alae nasi* working and with rapid shallow breathing. The affected side is almost or entirely immobile and is usually bulged. The displacement of the cardiac impulse towards the unaffected side is generally

obvious, and is almost immediate. Palpation confirms the absence of movement, and vocal fremitus is found to be absent, except where the collapsed lung remains in contact with the chest-wall, over which area it may be increased. The exact position of the cardiac impulse should also be determined: in right-sided cases it will be found in the left axillary region; in left-sided cases it may be under or beyond the right nipple. The liver may be felt much depressed in right-sided cases. The note over a pneumothorax is characteristically tympanitic or drum-like, as a rule, but in cases with positive pressure the tympany may be flat and muffled. The tympanitic area should be carefully mapped out; it may be found to extend across the middle line, or to encroach on or obliterate the liver dullness in right-sided cases. On the other hand, in partial pneumothorax, the area may be small and easily escape recognition. In left-sided cases, the cardiac dullness may be completely wanting on that side, and a dull area found to the right of the sternum. This may give a useful hint as to the diagnosis. On auscultation, the breath-sounds are often absent, but they may be present at the apex, although weak. In other instances distant tubular breathing may be audible from the collapsed lung; in cases with a large patent opening hollow cavernous breathing may be heard. The voice sounds have an amphoric or metallic quality, and an amphoric echo may occur with any sound produced near the pneumothorax. Metallic tinkling is an example of this, being the quality conveyed to râles or other adventitious sounds produced in breathing. The bell sound or *bruit d'airain* is a valuable sign, but is not invariably present. It is elicited by listening to the chest, near where a coin is placed flat on it and tapped with another. A similar sound may be heard with the stethoscope on flicking over a pneumothorax with the thumb and finger. The displacement of the heart can be confirmed by auscultation, and the heart sounds may be found to have a metallic character. When air and fluid are present in the pleura the signs are somewhat modified. There is dullness at the base, which shifts its level with the patient's movements, the upper limit being straight, in contrast with the curved line of ordinary effusions. A marked succussion splash may be heard and felt on shaking the patient, or the patient may demonstrate the sign by a sudden shake or jerk.

Complications and Sequelæ.—Cardiac failure and rapid death occur occasionally. The chief complications are due to the entry of infective organisms into the pleura, leading to pleurisy and the effusion of sero-fibrinous fluid or pus. The sequelæ may be pleural adhesions in cases that recover, especially if effusion occurs. There may be also permanent collapse of the lung in long-standing cases, and in pyo-pneumothorax a fistula, either pleuropulmonary or external, may remain in spite of treatment.

Diagnosis.—The recognition of a large or of a complete pneumothorax is easy as a rule, the signs being characteristic. When a large quantity of fluid is present in an open pneumothorax, the presence of air may not be recognised until after paracentesis or X-ray examination. The latter gives information of the greatest value and sometimes demonstrates the presence of local pneumothorax where it has not been suspected. The air space between the lung and pleura shows most clearly in radiograms, and if fluid is present as well, the dead level of the upper border of the shadow, varying with position, is most characteristic. Diagnosis is more difficult in cases where

pleural adhesions exist, or where the pneumothorax is small and localised, especially if X-ray examination is not available. The following conditions may give rise to difficulty and should be considered in doubtful cases. (1) Total excavation of a lung, or a large pulmonary cavity, in either of which the note may be boxy or even tympanitic, the breath sounds amphoric and the râles metallic or tinkling, while the coin sound may be obtained. These conditions can usually be distinguished by the flattening and retraction of the chest-wall over them, and the absence of cardiac displacement, or if it exists, the traction of the heart towards the affected side by fibrosis. (2) Advanced emphysema, with complete obliteration of the cardiac dullness, may be confused with pneumothorax. Large bilateral bullæ may be mistaken for bilateral pneumothorax. (3) Massive collapse of one lung, with compensatory emphysema of the opposite side, may also be mistaken for it. In both these conditions careful examination will establish the real nature. (4) A subphrenic abscess containing gas (subphrenic pyo-pneumothorax); in this condition the diaphragm may be displaced upwards, and the note over the lower ribs may be markedly tympanitic. These signs are more suggestive when right-sided. Succussion splash and bell sound may be elicited. The heart, if displaced, is pushed upwards. The history of previous abdominal disease may be helpful, and a radiogram may give conclusive evidence of the subphrenic origin of the condition. (5) A hernia of the stomach or bowel through the diaphragm, or eventration of the diaphragm, all rare conditions, may simulate pneumothorax, but in all there is generally abdominal flattening and little if any cardiac displacement. Examination by X-rays after an opaque meal will, as a rule, establish the nature of the condition.

Course and Prognosis.—The course and prognosis of pneumothorax are profoundly influenced by the cause. In cases due to rupture of an emphysematous vesicle, or of a small localised healed tuberculous focus, where the pleura remains sterile and the aperture of entry closes, the air is usually completely absorbed in a few weeks and recovery is often complete. In tuberculous cases with moderate disease, in which the pleura remains sterile, pneumothorax may exert a favourable influence. In tuberculous cases with extensive disease, the pleura becomes infected and death usually results in a few weeks or months, although with judicious treatment life may be prolonged for years in some cases especially where surgical treatment such as thoracoplasty becomes practicable. In pneumothorax secondary to some grave disease, such as carcinoma or gangrene, the course is brief and the prognosis is grave in the extreme. In cases secondary to empyema, surgical treatment may be followed by complete recovery.

Treatment.—The indications in cases of acute onset are to relieve the patient's pain, distress and anxiety, and to lessen the intrapleural pressure, if this is positive. A hypodermic injection of morphine, gr. $\frac{1}{4}$ for an adult, with oxygen inhalations if necessary, may achieve the first of these. If dyspnoea is extreme and the cardiac displacement marked, a trocar or large hypodermic needle should be inserted through an intercostal space to allow air to escape. An initial pneumothorax needle, with a long rubber tube, the end of which is placed under water, is the safest. This simple manœuvre may be the means of saving the patient's life in valvular pneumothorax, as well as of relieving distress. In less urgent cases, the pressure may be taken with

an artificial pneumothorax apparatus, and if the pressure be positive, as much air as is necessary may be removed by its means. In simple cases no other treatment may be required, although the puncture may need to be repeated. In cases of recurrent spontaneous pneumothorax or of persistent valvular pneumothorax, it is sometimes helpful to induce an aseptic pleurisy by means of various injections into the pleural space. Chandler recommends for this purpose a solution of gomenol in olive oil. At first 2 c.c. of a 2 per cent. solution are used, and this is gradually increased if necessary up to 20 c.c. of a 20 per cent. solution. A strong solution of dextrose is sometimes employed for the same purpose. In valvular pneumothorax Chandler has used a self-retaining catheter with a valvular attachment allowing the escape of air. If serous fluid or pus collect in the pleura, they may be withdrawn, preferably by siphonage, and in this case, as also with removal of air, too much should not be withdrawn in the early stages, as a slight positive pressure may assist in closure of the aperture in the lung, whereas a negative pressure may open it, after it has begun to close.

The question of operation in pneumothorax may be difficult to decide. In cases secondary to empyema, resection of a part of a rib and drainage often lead to satisfactory results. In cases of moderately severe or advanced tuberculosis with pyo-pneumothorax, open operation is generally contra-indicated, and if performed is liable to result in a permanently open pneumothorax. It is preferable to remove fluid from time to time by aspiration, with or without air replacement until thoracoplasty can be considered. Pleural wash-outs with mild antiseptics, such as weak methylene blue, flavine, or eusol, are often useful. This method of aspiration sometimes seems to assist the lung to re-expand. Surgical methods sometimes employed are intercostal tube drainage with slight suction, water-sealed drainage, or thoracoplasty in several stages.

HYDATID DISEASE OF THE PLEURA

Hydatid cysts may be primary in the pleura, or may encroach on the pleura, although originating in adjacent structures such as the lung, liver, spleen or mediastinum (parapleural hydatid).

Ætiology and Pathology.—Primary pleural hydatid is rare, but secondary invasion of the pleura is more common. In this situation the cyst may reach a large size, even 5 or 6 inches in diameter, before rupture occurs. As in other situations, a fibrous capsule is developed around the cyst from the irritative changes set up in the adjacent tissue. Contrary to what might be expected, extensive pleurisy is uncommon until rupture or suppuration of the cyst occurs. The pressure of the cyst may lead to collapse of the contiguous areas of lung and to displacement of the heart and mediastinum.

Symptoms.—These may be absent until the cyst is large enough to produce pressure symptoms, such as dyspnoea, pain and cough. There is little or no expectoration, unless rupture into a bronchus occurs, when cyst wall, daughter cysts and hooklets may be found in it. There is no fever until suppuration occurs. The signs are practically identical with those of encysted pleural effusion.

Complications and Sequelæ.—Rupture and suppuration are the two most important complications. Rupture may take place into the lung, into the pleural cavity, rarely through the chest-wall or through the diaphragm. At the time of rupture an urticarial rash may develop. This is probably an anaphylactic phenomenon associated with the liberation of toxin present in the fluid of the cyst.

Course.—The cyst may be latent for some time, but it usually enlarges and produces increasing symptoms, culminating in rupture or suppuration. Very rarely death of the cyst occurs and its contents become inspissated.

Diagnosis.—The symptoms and signs generally suggest either pleural effusion or new growth, and hydatid disease may not be suspected. Obscure basic signs, in patients coming from countries where hydatid disease is common, should suggest special methods of investigation as to the possibility of its presence. Should it be suspected, aspiration is to be deprecated, unless all preparations for immediate operation are complete, if the diagnosis is confirmed. These methods comprise X-ray examination, an eosinophil blood count, the complement-fixation test, the Casoni intra-dermic test and the precipitin reaction (see page 315).

Prognosis.—If untreated until rupture occurs, a fatal result is most probable. If diagnosed and treated before rupture, the prognosis is not unfavourable.

Treatment.—The former practice of aspiration and injection with formalin or iodine, although sometimes successful, is dangerous and should be discarded. Exposure of the cyst by thoracotomy, and its removal entire, should be the treatment if practicable, or if too large, it may be aspirated and then dissected out.

STREPTOTRICHOSIS (ACTINOMYCOSIS) OF THE PLEURA

The general characters of infection by the streptothrix group of organisms, and the special features of the pulmonary localisations have been described. It is possible, although improbable, that the infection may be primarily pleural, more commonly clinical manifestations may point to a predominating involvement of the pleura, although the primary lesions may be in adjacent structures, such as the lungs, mediastinum or liver.

Symptoms.—The symptoms and signs in such cases are those of empyema, but the following points are noteworthy. The empyema is rarely large, and it commonly extends through the chest-wall, producing a local swelling which soon discharges through the skin if untreated, causing a suggestive infiltration and puckering around. Exploratory puncture of a streptothric empyema often fails, since the grumous caseous material it contains may be too thick to pass through the needle.

Diagnosis.—The characteristic "sulphur granules" in the pus may draw attention to the real nature of the condition, but they are not always present. Direct films should always be made from the pus obtained from empyemata. The streptothrix may be found in this way, when culture fails. If the lung is involved as well as the pleura, the organism may be found in the expectora-

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tion, and the nature of the pleural condition may thus be established before operation.

Prognosis.—Some cases respond to treatment, but prognosis is in general unfavourable, death resulting from exhaustion or toxæmia due to dissemination of the disease.

Treatment.—The pultaceous pleural contents should be removed as far as possible by operation, and large doses of potassium iodide given by the mouth, increasing the quantity until 60 to 90 grains, three times a day, are given. An autogenous vaccine may be tried if the organism can be grown. A radium pack has sometimes given successful results.

SIMPLE TUMOURS OF THE PLEURA

These are very rare and are, as a rule, only discovered after death. They are almost invariably of extrapleural origin and their presence in the pleura is due to the direction taken by the growth. Lipoma of the subpleural or of the mediastinal fat may occur as small pedunculated tumours or very rarely as a large mass. They can be differentiated from tumours of the lung by X-ray examination after a diagnostic pneumothorax.

MALIGNANT TUMOURS OF THE PLEURA

Primary malignant disease of the pleura is rare, and may take the form of endothelioma, carcinoma or sarcoma. Secondary carcinoma and sarcoma are more common.

Ætiology.—Primary endothelioma of the pleura is more common in late adult life and in the male sex. Sarcoma is more likely to occur in children and in young adults. Secondary growths may occur at any age, but more commonly in later life.

Pathology.—Endothelioma of the pleura is a growth of obscure origin. It has not been conclusively established that it is derived from the pleural endothelial cells, and by some writers it is classed as a carcinoma. It is at first unilateral, but it involves the affected pleura over a wide area, sometimes universally. The membrane appears to be overlaid with an irregular, rough hard covering, sometimes nodular. In other cases there is more thickening and the condition may be localised. There is nearly always a large amount of blood-stained serous effusion. The condition may spread to the bronchial or supraclavicular glands, the lung, the spine, the diaphragm and the peritoneum.

Primary carcinoma of the pleura has also been described, but is very rare. Primary sarcoma is also extremely uncommon, but the round-celled and spindle-celled varieties may occur, and angio-sarcoma, fibro-sarcoma, myxo-sarcoma and chondro-sarcoma have all been recorded.

Secondary carcinoma and sarcoma of the pleura are relatively common, and may occur from direct *extension* in growths of the lung, bronchi and mediastinum, by *metastases* of growths in almost any distant part, or by *lymphatic permeation* in mammary carcinoma. In the last-named condition

pleural and pulmonary growths are a not infrequent form of recurrence, sometimes occurring months or years after removal of the primary growth.

Symptoms.—These are not characteristic, and increasing dyspnoea due to an accumulation of fluid may be the first indication. More commonly pain and cough, similar to those of pleurisy, may occur acutely or develop more gradually. Although afebrile as a rule, the occurrence of fever does not exclude malignant disease. Cachexia and wasting are often not marked until the condition is advanced. The signs are generally indistinguishable from those of ordinary pleural effusion, unless secondary growths become manifest in the cervical or axillary glands. Sometimes coarse dry friction may be heard, or there may be signs of pleural thickening without fluid. There is often local pain and tenderness over the chest. Exploratory puncture may demonstrate the hæmorrhagic character of the effusion. The specific gravity is generally 1018 or over, and the cytology of the fluid may be suggestive, especially if excess of endothelial cells, often aggregated into plaques, is found.

Complications.—The growth may spread to the lung and cause cough and expectoration, often blood-stained, or it may involve the chest-wall. Metastases sometimes develop along the course of the needle track after aspiration of the fluid. The secondary growths, especially those in the glands, may exert pressure, *e.g.* the axillary glands may cause œdema and swelling of the arm.

Course.—This is almost invariably progressive, the duration being rarely more than 2 years, and occasionally much less.

Diagnosis.—A chronic pleural effusion in a middle-aged man, not associated with fever, and not due to tuberculosis, should arouse suspicion of malignant disease of the lung and pleura. Evidence of fluid in one pleura, at an interval after excision of the breast for malignant disease, is very suggestive of secondary pleural growth. A hæmorrhagic effusion, not due to tuberculosis or renal disease, should also arouse suspicion of malignancy, especially if reaccumulation after tapping is rapid, and if the subsequent tapplings show increasingly hæmorrhagic characters. When aspiration of a considerable quantity of fluid gives little relief to symptoms, or when irregular dull areas remain where resonance might be expected, the probability of growth must be borne in mind. Growth involving the chest-wall, or the presence of cervical or axillary glandular metastases render it certain. Radiological examination after removal of some of the fluid may show characteristic plaques on the pleura.

Prognosis.—Malignant growth of the pleura is invariably fatal unless removal is possible.

Treatment.—From the nature of the condition this can only be palliative. Analgesic drugs may be given freely for the relief of pain, morphine being reserved for the severe forms and later stages, as far as possible. Repeated tapplings may be almost compulsory, if there is much distress from the reaccumulation of the fluid, but it must be remembered that in hæmorrhagic effusions the loss of blood by this means is considerable. Air replacement may sometimes give relief for a longer period than simple aspiration. In rare cases, removal by operation may be practicable if the diagnosis is made early and the growth is localised in an accessible position.

INJURY

Injury to the pleura may occur in fracture of the ribs, the fragments piercing or tearing it. Similarly in penetrating wounds of the chest, the pleura may be extensively lacerated. It may also be torn by direct violence without breaking of the ribs, and in rare cases a hernial protrusion of lung may occur, forming a small swelling in an intercostal space, protruding with inspiration and emptying with expiration.

R. A. YOUNG.

G. E. BEAUMONT.

DISEASES OF THE DIAPHRAGM

SPASM OF THE DIAPHRAGM

Diaphragmatic spasm may be either clonic or tonic, the former being termed *hiccough*.

Clonic spasm.—This may be due to a variety of causes, namely: (a) *Alimentary*: From irritation of the œsophagus or stomach by pungent or irritant substances such as pepper, pickles, or tobacco. It occurs also as a symptom in gastritis, dilatation of the stomach, enteritis, intestinal obstruction, tympanites and peritonitis, and in the late stages of debilitating disease. (b) *Nervous*: as in hysteria, cerebral tumour, meningitis, hydrocephalus, epilepsy and alcoholism. It may also result from peripheral nerve irritation, in such conditions as mediastinal tumour, mediastinitis, enlarged thoracic glands, diaphragmatic pleurisy, or pericardial effusion. *Epidemic hiccough* has been regarded as a form of encephalitis lethargica. There is usually some slight pyrexia, and the condition may persist without intermission for several days. (c) *Renal*: As in chronic nephritis and uræmia.

Tonic spasm.—This may be met with in tetanus, strychnine poisoning, laryngismus stridulus, eclampsia, epilepsy and hydrophobia. If there is associated intercostal or laryngeal spasm, there is grave risk of death from asphyxia.

Treatment.—Simple hiccough may often be relieved by holding the breath, pressure on the chest, or by simple inhalations, such as of ammonia, ether, or spirits of chloroform. Hiccough due to organic disease or to peripheral irritation may only be relieved by removal of the cause. In epidemic hiccough, in obstinate cases of hiccough due to other causes and in the tonic form of spasm, various antispasmodic measures may be tried, such as trinitrin, bromides, or phenobarbitone, by the mouth; adrenaline, or adrenaline and pituitary (posterior lobe) extract, hypodermically; or the inhalation of chloroform.

DIAPHRAGMATIC PLEURISY

This condition is described on page 1247 under the heading of Pleurisy.

PARALYSIS OF THE DIAPHRAGM

Definition.—Paralysis and inactivity of either leaf of the diaphragm, or of both.

Ætiology.—Paralysis of the diaphragm may be caused by disease damaging the centre in the spinal cord, by conditions affecting the phrenic nerve in its course, or by reflex inhibition of the centre. Causes involving the centre include poliomyelitis, hæmorrhage into the spinal cord, and tumours of the spinal cord or its membranes, or of the spine itself. The phrenic nerves may be affected by diphtheritic neuritis. Either or both of the nerves may be compressed by mediastinal tumours, or by inflammatory exudates. They may be severed or injured by wounds in the neck. Evulsion or crushing of the phrenic nerve is now frequently employed therapeutically, in order to promote collapse of the base of one lung and closure of cavities in cases of tuberculosis and in bronchiectasis.

Symptoms.—Diaphragmatic paralysis results in the affected leaf of the diaphragm becoming immobile and remaining at a higher level in the thorax than normal, or showing paradoxical movement, *i.e.* ascending with inspiration. This can easily be seen on X-ray examination. Sometimes this is noted as a reversal of the ordinary abdominal movements during respiration, with the result that there is epigastric recession during inspiration.

Treatment.—This is, in general, that of the condition causing the paralysis.

HERNIA OF THE DIAPHRAGM (see p. 553, 554)

EVENTRATION OF THE DIAPHRAGM (see p. 666)

DISEASES OF THE MEDIASTINUM

The mediastinum is the interpleural space, and occupies the median part of the thorax, from the superior aperture above to the diaphragm below. Strictly speaking, any affection of any of the important structures occupying this space, such as the pericardium, heart, great vessels, air passages and the thymus, might be included under this heading. They are, however, more conveniently grouped under the various systems to which they belong, and diseases of the mediastinum are commonly restricted to conditions arising in, or affecting the connective tissue and glands found in this space.

MEDIASTINITIS

Mediastinitis, or inflammation in the mediastinal connective tissue, may be acute or chronic. In the acute forms there may be an inflammatory serous exudate causing œdema, or the inflammation may progress to abscess formation. The chronic forms are indurative or fibroid in character, although chronic abscess may occur.

ACUTE SIMPLE MEDIASTITIS

Ætiology.—Acute mediastinitis without suppuration may result from injuries to the chest-wall or sternum, and from lacerating wounds of the œsophagus or trachea. It is sometimes secondary to inflammatory processes in the lungs, pleuræ, pericardium or peritoneum, and to periostitis of the sternum or vertebræ. Pneumonia is a not uncommon cause.

Pathology.—There is hyperæmia of the mediastinal connective tissue with inflammatory œdema. Mediastinal serous effusions have been described, but these are, without doubt, encysted pleural effusions encroaching on the mediastinum.

Symptoms.—The clinical manifestations of acute mediastinitis are vague and not characteristic. There is a mild pyrexia, the temperature reaching 99° or 100° F. Pain under the sternum may be complained of, and on auscultation over it a few fine crepitations may be heard on deep breathing, or they may occur synchronously with the heart beats.

Course.—The affection may subside or proceed to abscess formation. It may result in fibroid thickening or adhesions.

Diagnosis.—Mediastinitis is often not recognised or suspected, since it is masked or overshadowed by the clinical manifestations of the primary condition.

Treatment.—No special treatment is required, apart from that appropriate to the condition inducing it.

ACUTE SUPPURATIVE MEDIASTITIS

Ætiology.—Acute suppurative mediastinitis or mediastinal abscess is more common in males, and may occur at any age, although it is more frequently seen in early adult life than at other periods. Some cases are of traumatic origin, and follow perforating wounds or blows on the sternum, not necessarily causing fracture. Perforation or injury of the œsophagus is a comparatively frequent mode of access of pyogenic organisms to the mediastinum. This may occur from ulceration of an œsophageal new-growth, from injury due to a swallowed body such as a tooth-plate, or from the passage of an œsophagoscope or bougie. Perforation of the trachea or main bronchi by an inhaled foreign body is sometimes the cause of mediastinal suppuration. Various pulmonary conditions may lead to pyogenic infection of the mediastinum, such as pulmonary abscess or gangrene, pneumonia and bronchiectasis. Periostitis or osteomyelitis of the sternum, vertebræ or ribs, suppuration in the mediastinal glands, or tracking down of deep cervical abscesses may all lead to mediastinal abscess. Extensions of pyogenic processes from the pericardium, pleura or peritoneum may also be causes. A suppurating hydatid or dermoid cyst may rupture into the mediastinum, and, lastly, the infection is blood-borne in some cases from infective endocarditis, pyæmia, erysipelas or enteric fever. Dieulafoy pointed out that certain cases of empyema, originating near the mediastinum, may, by encroaching on this region, induce predominating mediastinal symptoms, which he described as the "mediastinal syndrome." Such cases, although abscesses in the mediastinum, are not mediastinal abscesses, but are in reality special instances of encysted empyema.

Pathology.—The suppuration may be limited to any part of the anatomical subdivisions of the mediastinum, or may spread from one compartment to another. The pus sometimes tracks in various directions, *e.g.* upwards to the neck, downwards to the abdomen, or it may point in the chest-wall. The abscess may rupture into the œsophagus, trachea, aorta, pleura or pericardium.

Symptoms.—The onset may be insidious or acute. In the latter case it may be ushered in by severe pain under the sternum, radiating to the back and shoulders. The symptoms may be divided into those due to the inflammatory process, and those resulting from the pressure exerted by the collection of pus. The former comprise malaise, fever and sometimes rigors, while blood examination may demonstrate a leucocytosis of 10,000 per c.mm. or over. The pressure symptoms vary according to the amount of pus produced and its situation. They include dyspnœa and paroxysmal or brassy cough, from compression of the vagus nerve or direct pressure on the trachea. There may also be dysphagia from obstruction of the œsophagus, and hoarseness from pressure on the left recurrent laryngeal nerve. Pressure on the spinal nerve roots, intercostal nerves, or brachial plexus may lead to severe neuralgic pains. Partial or complete obstruction of the great veins may be apparent from distension of the superficial thoracic veins or of those in the neck. Œdema of the chest-wall is sometimes seen from this cause, or it may result from the inflammatory process extending to the chest-wall. The signs in severe cases will be those caused by the pressure effects just described. The patient looks ill, distressed, dyspnœic and more or less cyanosed. The respirations may be noisy, as there is sometimes inspiratory dyspnœa with stridor, this being known as the *bruit de cornage*. The dilated veins may be apparent and the direction of the current may help to localise the seat of the obstruction. There is sometimes local redness and œdema from pointing of the abscess, near the sternum, in the neck, or in the interscapular region on either side. Palpation may reveal local tenderness and even fluctuation in any of these areas. There is often dullness over the sternum, sometimes extending to one or other side, or the dullness may be found in the interscapular region. It is said that the dullness may shift with the position of the patient in some cases. Breath sounds are distant, and weak or bronchial over the dull area, except when it is behind the sternum, when they are harsh.

Complications and Sequelæ.—The important complications are those due to rupture of the abscess. If this occurs into the lung or the œsophagus, pus is expectorated, or passes into the stomach. Gangrene of the mediastinum may follow, or death may occur from suffocation or hæmorrhage. Extension of the abscess may lead to purulent pleurisy, pericarditis or peritonitis, or to suppuration in the neck. In cases that recover, chronic mediastinitis with matting together of the mediastinal contents may be a sequel.

Course.—The disease is acute and rapidly progressive, unless relieved by operation or by spontaneous external drainage in a few fortunate cases.

Diagnosis.—The “mediastinal syndrome” of dyspnœa, stridor, paroxysmal cough, hoarseness and dysphagia with signs of pressure on arteries, veins and nerves is common to many conditions causing mediastinal pressure, notably mediastinal new-growth, enlarged mediastinal glands, aneurysm and

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pericardial effusion. The differential diagnosis of these is more fully considered under mediastinal new-growth. The occurrence of fevers and rigors, the presence of a pointing swelling, and the demonstration of a leucocytosis may give strong suggestion as to the inflammatory origin of these symptoms and signs. The X-rays may reveal a localised mediastinal shadow, often non-pulsating, although it must be remembered that in rare cases a mediastinal abscess may pulsate.

Prognosis.—This is very grave, and the majority of cases die unless recognised and treated early. If gangrene develops, a fatal result is inevitable. The outlook is more hopeful when the anterior mediastinum alone is involved.

Treatment.—**PROPHYLACTIC.**—Foreign bodies in the œsophagus and trachea should be removed as soon and as gently as possible. The utmost care should be exercised in the passage of a bougie or the œsophagoscope in cases of œsophageal stricture.

CURATIVE.—As soon as mediastinal suppuration has been diagnosed and localised, surgical measures should be adopted. The mediastinum can be reached by resection of pieces of costal cartilage or by trephining the sternum.

CHRONIC MEDIASTITIS

This also occurs in two forms, chronic indurative mediastinitis and chronic abscess.

CHRONIC INDURATIVE MEDIASTITIS.—This may occur as a sequel of any form of acute mediastinitis. The best known is that associated with chronic adhesive pericarditis, and usually known as chronic indurative mediastino-pericarditis (pp. 961-963). Other forms include the chronic inflammation and thickening which occur around enlarged, sclerotic and pigmented mediastinal glands, and around the same glands when affected by caseous or calcareous tuberculous lesions.

CHRONIC MEDIASTINAL ABSCESS is generally of tuberculous origin, arising from breaking down caseous bronchial or mediastinal glands, or from tuberculous disease of the spine or ribs. A chronic abscess may, however, be caused by a foreign body, such as a bullet.

Symptoms.—Simple indurative mediastinitis may give rise to practically no symptoms or signs. Chronic abscess may cause symptoms of ill-health and of mediastinal pressure, or may only become apparent when it points superficially.

Treatment.—The treatment of chronic mediastinal abscess is practically the same as that for other "cold" abscesses due to tuberculosis, incision and drainage being avoided if possible in favour of aspiration and injection of anti-tuberculous substances. Other cases may require operation.

EMPHYSEMA OF THE MEDIASTINUM

In performing tracheotomy, the pretracheal layer of deep cervical fascia is of necessity incised. If difficulty arises in inserting the tube into the tracheal incision, air may be drawn deep to this fascia by the vigorous attempts at respiration and thus pass into the superior mediastinum, or superficial to

it into the anterior mediastinum. Rupture of the trachea, bronchi or œsophagus, or rupture of air vesicles or pulmonary lesions where the pleura is adherent, may also cause it. In acute interstitial emphysema of the lungs, the escaped air may track along to the root and reach the mediastinum.

Symptoms.—Emphysema of the mediastinum may give rise to very indefinite indications. A few fine crackling sounds may be heard on listening over the sternum, sometimes varying with respiration or with the heart movements. The percussion note over the præcordium may be hyper-resonant, and the heart sounds may be distant and muffled. Small quantities of air escaped into the mediastinum can be rapidly absorbed and may not be of serious import.

Diagnosis.—This is often a matter of speculation, unless the air spreads upwards to the neck and causes superficial surgical emphysema.

Prognosis.—This depends entirely on that of the underlying cause, which is often of serious nature.

Treatment.—No special treatment is required, as a rule, apart from that of the primary condition, except that pain may necessitate the use of analgesic drugs at the onset.

ENLARGED MEDIASTINAL GLANDS

The mediastinal lymphatic glands are arranged in groups. A few small ones are found in the anterior compartment, another group is situated in the posterior mediastinum. The most important of these is the tracheo-bronchial group, situated around the bifurcation of the trachea and extending along the bronchi. It is enlargement of this group that most often gives clinical manifestations.

Ætiology and Pathology.—A simple inflammatory enlargement of these glands may occur in many acute affections of the bronchi and lungs, and in certain acute specific fevers, notably influenza, pertussis and measles. A more chronic enlargement, associated with indurative changes, results from chronic respiratory diseases, such as chronic bronchitis and the pneumoconioses. In the latter case, considerable pigmentary changes may be found, from deposition of the particles derived from the dusty inspired air. In town-dwellers, these glands are often grey or black in colour from deposited carbon. Tuberculosis is the commonest cause of enlargement of the mediastinal glands, particularly of the tracheo-bronchial group, those about the right bronchus being most affected as a rule. This is a frequent early localisation of tuberculous disease in children. The infection spreads from the lungs in the majority of cases (Ghon), but in some instances the path of infection is from the tonsils through the cervical lymphatics and glands, while in others the mode of entrance is from the intestines through the mesenteric glands. The lesions may be miliary tubercles, or small caseous nodules which calcify subsequently, or which may soften and lead to local spread or generalisation. In other cases a fibroid hyperplasia of the glands results.

In syphilis, mediastinal adenitis may occur in the secondary or tertiary stages. In Hodgkin's disease and in lymphatic leukæmia, the mediastinal glands may share in the general adenopathy, and in the former the condition

may be primary in these glands. Enlargement due to malignant disease is of great importance and receives separate consideration.

Symptoms.—These may be slight and escape notice, unless the enlargement is sufficient to produce pressure or irritation. Cough is the commonest symptom; it is usually dry, irritative, noisy and ineffective. It may occur in paroxysms, somewhat suggestive of those of whooping-cough. Dyspnoea and dysphagia occur only when the enlargement is considerable. Vomiting sometimes develops, probably reflexly from vagal stimulation. Pain behind the sternum or in the upper thoracic region posteriorly may be complained of. In children with tuberculous disease in these glands, there is often languor, anorexia, anæmia and wasting, sometimes with slight irregular fever and night sweats. Such symptoms in a child of 5 to 12 years of age are very suggestive. The signs are also variable and frequently inconclusive. In tuberculous cases, the appearance of the child, pale, delicate looking or sallow, with long eyelashes and fine hairy growth over the back, may also be suggestive. In glandular enlargement from any cause, there may be dilated veins over the front or back of the chest, especially in the upper part, and a "hilum dimple" has been described as appearing in the second intercostal space beside the sternum, on holding the breath at the end of inspiration. One pupil may be larger than the other, owing to sympathetic stimulation. Small areas of dullness may be found at the back, near the upper thoracic spines, or in front close to the manubrium. Breath sounds over these areas may be bronchial or harsh. Occasionally the enlarged glands impede the air entry to a lower lobe, generally the right, in which case breath sounds are notably weakened over this area, while the percussion note may be impaired. Normally, whispering pectoriloquy ceases at the seventh cervical spine; with enlarged mediastinal glands it may be heard along the middle line or close beside it, in the upper thoracic region from the first to the fifth thoracic spines. This is known as d'Espine's sign or tracheophony. It is a confirmatory sign, when other indications are present. Eustace Smith's sign is of little value. It consists in a venous hum, audible over the manubrium sterni, when the child's head is thrown back as far as possible. Occasionally pressure on the recurrent laryngeal nerve may lead to an abductor paralysis of one vocal cord. In cases of tuberculosis, syphilis, Hodgkin's disease or leukæmia, enlarged glands may be present in other parts of the body, and may thus assist in diagnosis.

Complications.—A caseous gland may ulcerate into a bronchus or into the trachea, and death has resulted from glottic impaction of a portion of the gland. Ulceration into the œsophagus has been described. Rupture into the mediastinum may lead to mediastinal abscess. Invasion of the pleura, lung or pericardium may occur, or generalisation causing widespread miliary tuberculosis.

Diagnosis.—Whenever the condition of mediastinal glandular enlargement is suspected, an X-ray examination should be made if possible. It may help to distinguish between other conditions causing mediastinal pressure, such as aneurysm, abscess and malignant growth. Unfortunately in regard to tuberculous disease, it shows best the condition of least importance, namely, the old healed calcified glands. "Soft" or "woolly" shadows are regarded as indicative of active disease, but in doubtful cases it is wise to act upon the clinical indications.

Prognosis.—This varies with the cause, being serious in Hodgkin's disease and leukaemia. In tuberculous cases, the prognosis is as a rule good, apart from complications, provided treatment is prompt and adequate.

Treatment.—In tuberculous adenitis, the general condition should be improved by every possible means. The child should be taken from school, rest and exercise are to be carefully graduated, and a liberal diet supplied, with extra milk, cream and butter. In England, the Isle of Thanet seems especially valuable in the climatic treatment of glandular tuberculosis. Cod-liver oil, malt extracts and the syrup of the iodide or phosphate of iron are useful. In afebrile cases, tuberculin cautiously given may be of value in children of 8 years or over, but it is not necessary, as a rule. If given, the initial dose should be small, $\frac{1}{1000}$ mgrm. B.E., and the dosage gradually increased. In glandular enlargements due to syphilis, Hodgkin's disease and leukaemia, the treatment appropriate to these diseases should be employed, and symptoms due to pressure relieved as far as possible.

MEDIASTINAL TUMOURS OR NEW-GROWTHS

The mediastinum may be the seat of either simple or malignant new growths, the latter being much more common.

SIMPLE TUMOURS OF THE MEDIASTINUM.—These, except retrosternal goitre, rarely give rise to symptoms, and the recorded cases have, as a rule, only been discovered in the course of a routine X-ray or post-mortem examination. The chief varieties found are retrosternal goitre and persistent thymus, lipoma, fibroma, chondroma, osteo-chondroma and myoma.

MALIGNANT TUMOURS OF THE MEDIASTINUM.—Although it is certain that some malignant growths arise primarily in the mediastinal tissues, while others invade the mediastinum secondarily by extension or metastasis, it is often impossible, even at autopsy, to determine whether a mediastinal growth originated in the mediastinal tissues or in one of the adjacent organs, particularly the lungs and bronchi. The differentiation between primary and secondary growths is therefore less sharp than in other situations.

SARCOMA OF THE MEDIASTINUM.—Recent research has proved that the majority of primary mediastinal growths are sarcomatous, but these are less common than was formerly supposed. A primary sarcoma may arise in the lymphatic glands, connective tissue, periosteum of the sternum or vertebræ, or in the remains of the thymus gland. The commonest variety is the lympho-sarcoma, but spindle-celled and chondro-sarcomata may occur. Mediastinal sarcoma is commoner in males than females; it may occur in early life, and the majority of cases occur before the age of 40 years. Oat-celled tumours invading the mediastinum and formerly regarded as lympho-sarcomata are now believed to be of bronchial origin and carcinomatous nature.

CARCINOMA OF THE MEDIASTINUM.—This is rare as a primary tumour. It occurs in older people. It may originate from the trachea, bronchi or œsophagus, in the remains of the thymus or in a retrosternal goitre.

SECONDARY MALIGNANT GROWTHS OF THE MEDIASTINUM.—These usually result from direct extension of primary growths of the lung, bronchi, trachea, œsophagus, chest-wall or breast, but true metastases may occur from

mammary growths or from more distant primary tumours. Endothelioma has been described in the mediastinum, but is probably generally secondary to endothelioma of the pleura.

Pathology.—The morbid appearance depends upon the situation of origin, the directions of growth, and the nature of the tumour. Sarcomata are generally soft, pinkish in colour and vascular, while carcinomata are paler and firmer. There may be one large mass weighing several pounds, or there may be multiple growths. When the tumour reaches a considerable size it may infiltrate, surround, compress or displace contiguous structures. This is particularly the case in the lympho-sarcomata. The trachea, œsophagus, and large vessels may be surrounded, the pericardium and heart may be extensively infiltrated, and the nerve trunks may be enclosed and compressed. Secondary deposits are common in other glands, but not infrequently the pigmented bronchial glands may be seen entirely enclosed in growth without being infiltrated.

Symptoms.—The onset is often insidious, and the condition may not be suspected until cachexia and pressure signs develop. Malaise, weakness, shortness of breath, cough and pain are often early symptoms, which become more pronounced as the case progresses. The pressure symptoms and signs constituting the “mediastinal syndrome” comprise—

1. *Pressure on the air passages*, giving rise to dyspnoea, cough and expectoration. The dyspnoea may be inspiratory and associated with stridor, or expiratory and paroxysmal. The cough is harsh and may be “brassy”; it is often associated with mucoid, blood-stained, or even “prune juice” sputum. Bronchiectasis may result in some cases.

2. *Pressure on or infiltration of the lung*, leading to collapse and sometimes breaking down of lung tissue. If the pleura is reached or invaded, pleural effusion, often blood-stained, may result.

3. *Pressure on arteries*.—Compression of branches of the pulmonary artery may lead to local gangrene, or in other cases the growth may ulcerate into a larger vessel and cause fatal hæmorrhage. Pressure on the subclavian artery may cause inequality of the radial pulses, and, according to Ekgren, this may only be present when the patient is lying and not when he is standing.

4. *Pressure on veins*.—Dilated tortuous veins may be seen over the front of the chest and abdomen, or in the neck. The flow of blood in these superficial veins may be reversed in direction, owing to the obstruction of the superior vena cava or its main radicles. The current then runs from above downwards, instead of from below upwards, as normally. There may be œdema of the chest-wall or of the face and neck from the same cause.

5. *Pressure on nerves*.—The vagus may be compressed, causing paroxysmal dyspnoea and cough. Laryngeal paralysis or spasm may result from involvement of the recurrent laryngeal nerve. Dilatation of the pupil, followed later by constriction, drooping of the upper lid and enophthalmos, occurs when the sympathetic is involved. Paralysis of the diaphragm on one side from compression of the phrenic nerve, and pain from involvement of the intercostal nerves, may be present.

6. *Pressure on the œsophagus* may lead to dysphagia of increasing degree.

In addition to the signs afforded by these various conditions, there may be glandular enlargements in the neck, the suprasternal notch, or in the axillæ.

The growth may invade the chest-wall at any spot, and in rare cases it may cause visible or palpable pulsation. The pulmonary physical signs are dyspnoea, sometimes orthopnoea and cyanosis. In some instances the patient prefers to lean forward; this is said to be due to the fact that in this position the antero-posterior diameter of the mediastinum is increased, and the tension caused by the growth is thereby lessened. There may be dullness over the sternum or over the upper thoracic spines, and over any part of the lung invaded or compressed by the growth. The breath sounds heard over the dull area may be harsh, bronchial, tubular, weak or absent. The signs due to any secondary condition, such as bronchitis, bronchiectasis or a pleural effusion may be found in addition.

Complications.—These include the secondary conditions just mentioned. Others are due to ulceration of the growth through the chest-wall, or into the trachea, bronchi, oesophagus or aorta. Pericarditis may occur if the growth invades the pericardium, and hæmopericardium may result from ulceration of a vessel.

Course.—The growth enlarges progressively and the course is often rapid, particularly in lympho-sarcoma. Fulminating cases lasting only a few weeks occur; more commonly the patients live from 6 months to 2 years from the onset, rarely more.

Diagnosis.—When signs of mediastinal pressure become apparent, new-growth should be suspected, in common with aneurysm, mediastinal abscess or cyst, enlarged mediastinal glands and pericardial effusion. The history, the general condition of the patient, the physical signs, blood examination, and the X-rays may all help in distinguishing between these conditions. The evidence afforded by the X-ray may be of the utmost value. The pulsating shadow of an aneurysm, the large area of a pericardial effusion, the indefinite edge of an infiltrating growth extending into the lung, may be shown clearly, but the appearance should always be interpreted in the light of the other clinical features, and a diagnosis should not be made on X-ray findings alone, since a growth may pulsate, or may give rise to an effusion, while a mediastinal abscess or a cyst may give a sharp shadow. An oesophageal new-growth can sometimes be differentiated by the oesophagoscope, but this should only be employed when aneurysm can be excluded. Diagnosis from pulmonary or bronchial new-growths may be almost impossible. Before the onset of pressure symptoms, growth may be suspected from the cough and emaciation, and here again the X-rays may give valuable indications. Chronic tuberculous disease should always be excluded by repeated sputum examinations. The diagnosis of mediastinal growth may sometimes be obscured by some of the complications it induces, notably pleural effusion and bronchiectasis. The rapid onset and progress of these conditions and the bloodstained character of an effusion may all suggest the possibility of a malignant cause. The presence of enlarged glands in the neck or axillæ, or of nodular growth in the chest-wall or episternal notch, may afford almost conclusive evidence of malignancy.

Prognosis.—This is practically hopeless and death occurs from exhaustion, starvation, toxæmia, asphyxia or hæmorrhage.

Treatment.—The treatment of simple tumours is surgical if they are capable of removal. The treatment of malignant tumours is that of inoperable malignant disease elsewhere. X-ray applications, or radium treatment in some

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form may be tried. Otherwise treatment is symptomatic and palliative. Pain may be relieved by aspirin, codeine or morphine. Sleep may be induced, if there is insomnia, by chloral hydrate, papaveretum (alopon, opoidine, omnopon) or other hypnotics. If effusion is causing dyspnoea it may be tapped, but the fluid usually collects again rapidly. Air replacement is sometimes useful.

CYSTS OF THE MEDIASTINUM

SIMPLE CYSTS.—These are usually small and of no clinical importance.

DERMOID CYSTS AND TERATOMATA.—These are rare and become apparent generally in young adult life. They may enlarge, giving rise to symptoms and signs similar to those of a mediastinal tumour, or they may lead to empyema. They usually contain pultaceous material, and sometimes hairs, muscle, cartilage, bone and teeth. Such cases are almost certainly teratomatous in nature and derived from included embryos. This condition may sometimes be diagnosed during life by the expectoration of hair, teeth, bone or cartilage. The prognosis is, as a rule, serious, but some cases recover under appropriate surgical treatment.

HYDATID CYSTS.—A hydatid cyst may be primary in the mediastinum and may give rise to signs of mediastinal pressure, but the condition is extremely rare. Its presence may be shown by X-rays and its nature demonstrated by the blood and skin reactions. Such cysts have been successfully treated surgically.

Other rare mediastinal conditions are hernia of the stomach or colon through the diaphragm into the mediastinum. A retrosternal goitre may also form a mediastinal swelling.

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SECTION XVI

DISEASES OF THE KIDNEYS

THE CHARACTERS OF NORMAL URINE

THE amount of urine normally secreted in 24 hours is 50 ounces or 1500 c.c. The specific gravity of the total should lie between 1015 and 1025, though individual specimens will vary considerably more, according to the amount of fluid imbibed or the quantity excreted by the skin and bowels. The reaction is generally acid, due to the presence of acid sodium phosphate, NaH_2PO_4 . The total acidity is such that about 650 c.c. of decinormal caustic soda will neutralise the daily output. This is equivalent to 82 grains of NaHCO_3 , but it requires a rather larger quantity (120 grains according to Spriggs) of bicarbonate of soda by the mouth to effect this neutralisation. Expressed in terms of H-ion concentration, the pH varies between 4.7 and 10. The total acidity of the urine rises very considerably in acidæmia, and may be more than doubled despite large doses of alkalis. Normally, urine is more acid during fasting than during absorption of food, the acid and alkaline tides being thus produced. The alkaline tide may be partly due to the absorption of organic vegetable salts, as it is more marked in herbivora, but the increased activity of the respiratory centre after getting up in the morning, is chiefly responsible for an increase in the pH of the urine (diminished H-ion concentration), by removal of excess of CO_2 from the body; in fact, it alone may be responsible for a morning alkaline tide. On decomposition, either in the bladder or after excretion, the urine becomes alkaline, from the conversion of urea into ammonium carbonate.

The constituents of urine are partly derived from the food (exogenous) and partly from the katabolism of the tissues (endogenous). We may briefly consider the source and significance of the principal constituents.

Nitrogenous constituents.—The total nitrogen excreted each day on an ordinary mixed diet is about 18 grammes or 270 grains. Of the various nitrogenous constituents urea is by far the most abundant, its output being 33 g., which contains 15.4 g. of nitrogen, or 85 per cent. of the urea total. As so much of the urea comes directly from the food, the amount of urea falls both absolutely and relatively in starvation; the total nitrogen drops to 5 g. or even less, of which urea nitrogen forms about 60 per cent. On a diet rich in carbohydrates and fat, but containing hardly any nitrogen, these figures may fall still lower, as the assimilation of other food-stuffs reduces the waste of tissue nitrogen to a minimum. This is often forgotten, and in nephritis undue importance is attached to a drop in the output of urea, which is simply due to the diet prescribed being poor in nitrogen, whereas the urea excreted depends mainly on the quantity of protein ingested.

Next in importance come the purin bodies. Purins contain the group C_5N_4 , and the best known is tri-oxy-purin or uric acid $C_5N_4H_4O_6$. A small quantity of the less oxidised purins, xanthin and hypoxanthin is also excreted. Exogenous purins come mainly from meat juices, from the nuclei of cellular organs (such as liver and sweetbread), and from tea, coffee or cocoa. Only from one-tenth to one-half of the ingested purins are excreted as such, the remainder being destroyed by the liver, ultimately appearing as urea. The alkaloids of tea, coffee and cocoa give rise chiefly to xanthin and hypoxanthin, the rest to uric acid. On a diet rich in meat the daily output of purins amounts to 0.34 g. of nitrogen, and on a purin-free diet to 0.202 g. This endogenous purin, which forms the larger part, comes mainly from the disintegration of the nuclei of maturing red blood corpuscles, and also the leucocytes and muscles. Anything increasing the leucocytes in the circulation increases the output of endogenous purins, and in leukæmia the excretion of uric acid may rise to 5 g. a day. Unaccustomed exercise diminishes the output of uric acid, while increasing that of the less oxidised xanthin and hypoxanthin, the total purin excretion remaining the same. Uric acid is only excreted as such when the urine is highly acid; normally it appears as acid sodium urate. (See also Urinary Deposits.)

Creatinin is, according to Folin, the most constant of the nitrogenous constituents on a meat-free diet, and serves as a measure of endogenous nitrogenous metabolism. In a healthy young man on a diet consisting entirely of bread, about 0.9 g. is excreted daily, while on a diet containing meat extracts more than 2 g. may be passed. During muscular wasting its output is increased, while in a subject already wasted it is diminished. All this suggests that it is derived from the creatin of muscle—both of the body and of the food. It may be recognised in urine by Weyl's test; with sodium nitroprusside and caustic soda it gives a ruby-red colour, which, unlike that given by acetone, is at once destroyed by glacial acetic acid. Jaffe's test depends upon the deep orange colour, given by even dilute solutions of creatinin on the addition of a saturated solution of picric acid and some 10 per cent. solution of caustic soda; this has been utilised for the colorimetric estimation of creatinin by Folin. Creatinin can be obtained from creatin by dilute boiling mineral acids. Creatin, which is abundantly present in muscles, is not normally present in the urine.

Ammonia is normally excreted to the extent of about 1 g. a day. An increase in this amount is not, as was formerly thought, a sign of incapacity on the part of the liver to form urea, but a sign of acidæmia. The body protects itself against acids in the circulation by forming ammonia from the proteins of the tissues. The increased excretion of ammonia is, therefore, a measure of the degree of acidæmia, and it is estimated by the amount of acid set free from the urine on the addition of formalin, which combines with the ammonia to form hexamine. In the acidæmia of diabetes, the output of ammonia may rise to 4 g. a day, or even more.

Hippuric acid is not an important nitrogenous constituent of urine, but it is of interest as being made by the kidney itself by the combination of benzoic acid with glycoooll. It is, therefore, increased by a diet of green vegetables, and is diminished when there is conspicuous degeneration of the renal tubules.

The pigments of urine are nitrogenous. The principal one, urochrome, to which urine normally owes its colour, though closely related to urobilin, has an independent origin from hæmoglobin. Even when all the bile escapes from the body through a biliary fistula the excretion of urochrome is unaltered. Urobilin, on the other hand, is a reduction product of bile pigment. The reduction is effected by bacterial action in the bowel, whence it is reabsorbed by the blood and excreted by the kidney. Normally it is not excreted as such, but as a colourless chromogen. The appearance of pre-formed urobilin is evidence either of increased hæmolysis or of septic infections of the gall-bladder or bile-ducts, or of increased intestinal putrefaction, or of increased time for reabsorption, as in intestinal obstruction. It can be recognised with the spectroscope by the absorption band it gives in the blue, or by the green fluorescence it shows on the addition of zinc chloride and ammonia. Very little is known of uroerythrin; it is an unstable body and is readily carried down by urates, to which it imparts the characteristic pink colour. A trace of hæmatoporphyrin is also normally present in the urine; but an obvious amount is an abnormality, which will be considered later.

Non-nitrogenous constituents.—These are principally salts. Chlorides are the most abundant, averaging about 10 to 13 g. of sodium chloride a day. Chlorides are retained whenever the body retains excess of fluid. This explains the reduced output of chlorides in such diverse conditions as cedema, serous exudates, pneumonia and acute dilatation of the stomach. Reduced chloride intake or loss through excessive vomiting are other causes. On the other hand the output is much increased in Addison's disease. The phosphates are partly excreted as acid phosphates of sodium and potassium, partly as earthy phosphates of calcium and magnesium. The former are not precipitated on neutralisation, while the latter are. A phosphatic deposit, as stellar crystals of calcium phosphate or tables of magnesium phosphate, is no proof of a real increase in the output of phosphates, but is usually merely an indication of diminished acidity. Ammonio-magnesium phosphate, on the other hand, is evidence of ammoniacal decomposition. It forms a deposit of "coffin-lid" or "knife-rest" crystals. The amount of phosphoric acid excreted daily amounts to about 2.5 to 3.5 g., of which the earthy phosphates form half. Sulphates are present in the urine to the extent of 1.5 to 3 g. of SO_3 a day. Very little sulphate is taken in the food, and most of that which is taken either as food or medicine is excreted by the bowel, so that the urinary sulphates come almost entirely from the oxidation of the sulphur in the protein molecule. About nine-tenths are excreted as sulphates of the alkalis, and the remaining one-tenth as ethereal sulphates, formed by conjugation with putrefactive products from the tyrosin and tryptophan of the protein molecule. Of these, the most striking is indican, or indoxyl-sulphate of potash. It is best detected by adding an equal quantity of strong hydrochloric acid to some urine, then a few drops of hydrogen peroxide, and shaking up the mixture with some chloroform to which it imparts a blue colour. Its presence in excessive amount is some evidence of excessive intestinal putrefaction, especially when due to obstruction of the small intestine. Not so much importance, however, is attached to indicanuria as formerly. All the sulphur in the urine is not excreted as sulphates; some 6 per cent. appears as neutral sulphur, derived from the

sulphocyanide of the saliva, the taurine of the bile salts and substances allied to cystin. The neutral sulphur is diminished in insanity.

Many other substances are normally present in traces in the urine, but except diastase, they are of little clinical importance. Ten to 30 units of diastase are normally present, but less will be found in some forms of impaired renal capacity and a great deal more in most pancreatic diseases. The presence of 50 units suggests a pancreatic lesion, while 100 or more make this certain. In severe pancreatitis 300 to 500 may be found.

THE ESTIMATION OF RENAL FUNCTION

It may be necessary to determine (1) the total renal capacity, or (2) the adequacy of either kidney separately. Generally speaking, the first is more the concern of the physician, and the second that of the surgeon. Estimation of the latter is of vital importance before nephrectomy is considered, lest the remaining kidney should prove inadequate to maintain life. Estimation of the former is an assistance both to diagnosis and prognosis. Some of the tests under the first heading have for their object the determination of the part of the kidney involved. These will be considered first.

A.—ESTIMATION OF CAPACITY OF BOTH KIDNEYS

1. **EXAMINATION OF THE BLOOD.**—The damaged kidney will fail to excrete substances which it should, and examination of the blood may reveal their presence in undue amount. The quantity of urea in the blood throws important light on renal capacity; normally this ranges from 15 to 40 mgm. per cent. in health, but after middle age figures up to 50 mgm. per cent. (urease method) may be within normal limits. The urea content of the blood, as well as that of the cerebro-spinal fluid, is raised in various kidney diseases, and also in alkalosis. A blood urea figure of 200 mgm. per cent. and over is of serious clinical significance. It may rise higher than this, even to 280 mgm. per cent., in acute nephritis, and gradually fall to normal with complete recovery. In chronic nephritis such figures generally indicate a terminal phase of few months' duration, but a patient may live for a year or more with a blood urea of 190 mgm. per cent. The amount of sodium chloride in the blood may be raised from the normal 0.45 to 0.5 g. per cent. to 0.6 or higher. When there is extreme renal failure there may be an increase in the H-ion concentration, the uric acid and the indican of the blood, while the calcium content may fall from the normal 10 mgm. per cent. to 6.

2. **THE UREA CONCENTRATION TEST.**—Although ordinary estimation of the percentage of urea in urine gives no information of value, the response of the kidney to a given dose of urea does. On this MacLean and de Wesselow based their useful urea concentration test. Fifteen grammes of urea dissolved in 100 c.c. of water, and flavoured with a little tincture of orange, are given to a patient just after he has emptied his bladder. The urea in the urine passed one, two and three hours afterwards is estimated by the hypobromite method. If this amounts to 2 per cent. or over in one or more of the three specimens the kidney is efficient according to the test. A concentration of 2.5 per cent. or over is more satisfactory. The volume of urine should not exceed 120 c.c. in the first hour, or 100 c.c. in each of the second and third hours. Excessive

diuresis may be due to release of water previously retained in the tissues, and the test should be repeated. This test is of less value if the patient is taking a low nitrogen diet.

3. **THE BLOOD UREA CLEARANCE TEST.**—This test was introduced by Möller, McIntosh and Van Slyke as a simple and reliable method of estimating the urea-excreting function of the kidneys. In principle it is based on the relation of the blood-urea concentration to the urea excretion in the urine. The result is expressed as cubic centimetres of blood cleared of urea per minute. For details a textbook of clinical pathology should be consulted. It is claimed that this test is more sensitive and will reveal minor defects not revealed by other tests.

4. **VOLHARD'S TESTS AS MODIFIED BY ROSENBERG.**—On the first day the patient, after passing urine, drinks 1500 c.c. of water within half an hour. Urine is passed at half-hourly intervals for the next 4 hours, each specimen being saved separately and tested for volume and specific gravity. Normally the whole 1500 c.c., often more, is excreted within the 4 hours, and the specific gravity falls to 1002, or less. The second day, ordinary meals are given, but the amount of fluid is limited to 500 c.c. for the whole 24 hours, taken in four roughly equal portions. Fruit should not be given, or should be reckoned as fluid. Urine is passed as and when the patient wishes, and each specimen is again collected separately and tested for volume and specific gravity. The total urine for the day should not exceed 750 c.c., and the specific gravity should rise to at least 1027. These are known as the dilution and concentration tests respectively.

In cases of renal insufficiency the volume of urine on the first day is too little, while that on the second day is too much, as excretion tends to continue at the same rate irrespective of variations in the requirements of the body. The limits of variation in specific gravity become more and more narrowed as the disease progresses, until at length a fixed point of about 1009 is reached. Often the dilution test will show a minimum of about 1005, and the concentration test a maximum of about 1015, long before the patient complains of any symptoms or the blood shows any evidence of uræmia.

In our opinion the concentration test is more reliable than the dilution test, but both of them set up too rigid a standard of what constitutes normal function.

5. **FIXATION OF SPECIFIC GRAVITY.**—This is a simpler method. The patient takes no fluid drinks or liquid foods or fruit from after breakfast one day until breakfast-time the next day. The urine passed in the first 12 hours need not be kept, but that secreted in the second 12 hours is collected and pooled. If the renal function is satisfactory the specific gravity of this urine should be at least 1024, and the concentration of urea should be more than 2 per cent.

B.—ESTIMATION OF CAPACITY OF EACH KIDNEY

Catheterisation of each ureter under the direct view of the cystoscope is the only reliable method of obtaining the required information. It is usual to encourage secretion during examination by giving some tea or simple diuretic. Additional information is gained by the intramuscular injection of 15 minims of a 5 per cent. aqueous sterilised solution of methylene-blue. It is first excreted as a colourless chromogen and, later, as methylene-blue itself.

The chromogen turns blue when boiled with acetic acid, and should appear in the urine in from 15 to 20 minutes, after which the excretion of unaltered methylene-blue should begin. It should reach its maximum in from 4 to 5 hours, and should have disappeared in from 40 to 50 hours. Obviously, catheterisation of the ureters cannot be continued all this time, so that observation is directed towards a marked delay in the appearance of blue on one side as compared with the other. Afterwards, hexamine should be given as a precautionary measure, and the patient kept in bed for 36 hours. As in acute or subacute nephritis the rate of methylene-blue excretion is entirely unaffected, the utility of this test is confined to unilateral chronic disease.

Indigo-carmin may be used for a similar purpose; 10 c.c. of a 0.4 per cent. solution is injected intravenously or intramuscularly. The urine should be coloured in about 10 minutes, first appearing green and then blue. Excretion reaches its maximum in about an hour, so that this test has advantages over the methylene-blue method. Delay in the appearance of the dye and a feeble staining of the urine may be taken as evidence of disease.

Pyelography is a valuable means of determining the position of the kidneys and their relation to shadows in or in the neighbourhood of the urinary tract. By this means the position of renal or ureteric calculi may be defined, and such shadows as those caused by calcareous tuberculous glands, gall-stones, and faecal calculi may be recognised as outside the urinary tract. It is an invaluable means of demonstrating the presence of a hydro-nephrosis, especially when small. It will show dilatation or irregularities in the course of the ureter. By the absence of the shadow caused by the dye, a failure in function of one kidney or its absence may be indicated. Renal growths and tumours of the renal pelvis may be diagnosed by abnormalities in the pyelogram, and calculi not evident in a plain radiogram may be shown by this means. The intravenous method is now commonly adopted in the first approach to a urinary case, but instrumental pyelography is often required to confirm the findings obtained.

A drug, opaque to X-rays, which is eliminated by the kidneys, is introduced intravenously, and radiograms are taken at short intervals after its injection. Uroselectan B (a non-toxic iodine-containing substance) is the best preparation for this purpose. For instrumental pyelography a 12 to 20 per cent. solution of sodium iodide or sodium bromide is used. Before iodine is given for the purpose of pyelography, the patient's tolerance of the drug should be tested by giving five or ten grains by mouth, in order to exclude an idiosyncrasy.

ABNORMALITIES OF THE URINARY SECRETION

1.—POLYURIA

Polyuria may be due to—

1. Increase in the quantity of fluid imbibed.
2. Increase in the molecular concentration of the urine as in diabetes mellitus, or after saline diuretics. More water is thereby attracted into the blood stream by osmotic pressure.

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3. Incapacity of the kidney to excrete a concentrated urine, as in chronic interstitial nephritis.

4. Dilatation of the kidney vessels, as produced by stimulating diuretics of the caffeine group. These diuretics have been shown by Curtis, using experimental methods, to act directly on tissue cells, causing the cells to part with their water; hydræmia results, and the excess of water in the blood is immediately excreted by the kidneys.

"Diabetes insipidus" is frequently due to disease of the pituitary gland or of the overlying hypothalamus, or to damage in this neighbourhood by syphilitic meningitis of the base of the brain. It is also probable that hysterical polyuria is due to a temporary inhibition of pituitary secretion through the sympathetic. This appears to affect the renal vessels directly, since pituitrin will check diuresis, even in the denervated kidney.

2.—ANURIA

Suppression, as opposed to retention of urine, may be due to—

1. Acute nephritis with intense congestion and nephrosis, whether the result of an infection or of drugs, such as turpentine, cantharides or carbolic acid.

2. Bilateral obstruction to the ureters.

3. Reflex causes, such as operations on the kidney or trigone of the bladder.

4. Vasomotor conditions, as collapse, shock or irritation of the vasomotor centre. Probably the anuria in diphtheria is due to the last of these (Garratt). In cholera there is not only collapse, but depletion of water by other channels.

5. Hysterical. This condition has been described by Charcot. It is, however, rare and the element of fraud must be eliminated. Thus, in one case, urea was found abundantly present in the contents of the washing-bowl, and this explained how the urine was disposed of.

3.—ALBUMINURIA

Albuminuria should be more correctly termed proteinuria—since blood serum contains two proteins—albumin and euglobulin—and either may appear in the urine, though search is seldom made for the latter. The ordinary tests of heat coagulation, nitric acid or salicyl-sulphonic acid give positive results with either. The presence of euglobulin may be shown by the addition of dilute acetic acid (33 per cent.) to urine in the cold. The acid is added drop by drop, and the precipitation of globulin is shown by an opalescence in the urine to which the acetic acid is added. Mucin is also deposited by the addition of acetic acid, but it is not redissolved by an excess of acid. A more distinctive test is the precipitation of globulin in distilled water. Single drops of urine are dropped into a glass vessel containing distilled water. As the drop of urine falls through the water it assumes a ring form, and the ring has a milky appearance due to precipitated globulin when the latter is present. The globulin can be precipitated for quantitative examination by making the urine alkaline with ammonia, and then half saturating it with ammonium sulphate.

Proteinuria may be classified thus—

I. WITHOUT ORGANIC DISEASES OF THE KIDNEYS, as in—

1. *Functional or orthostatic proteinuria*.—This is common in males between puberty and adolescence; it is much less common in females of the same age. Dukes found it in 16 per cent. of all boys entering Rugby School. Protein appears only in the urine secreted in the upright posture, and is absent from the urine passed on first rising. There is no evidence that the amount of protein in the food influences it, though some constituent of raw eggs may excite a transient albuminuria by a toxic action on the kidneys. Severe physical exercise will excite proteinuria in most healthy young adults. Collier found it present in every one of the Oxford crew of 1906 after rowing a course; to such a condition the term “physiological” proteinuria may fairly be applied. When the protein appears apart from exertion, the subject is often an anæmic weedy youth with a dull heavy aspect and a tendency to fainting. The heart is irritable, and the blood pressure unstable, and fluctuates with change of posture. There may also be a few hyaline casts, and frequently calcium oxalate crystals. In any case of proteinuria in a boy or young man the diagnosis of a kidney lesion should not be made unless casts other than hyaline are discovered, unless the tension of the pulse is definitely and permanently raised, and unless there are signs of cardiac hypertrophy. In the absence of such evidence, the urine passed on first rising should be examined. If this is free from protein, the condition is almost certainly functional. Then 15 grains of calcium lactate should be given three times a day for 3 days, after which the urine should be examined again. If this checks the proteinuria no further anxiety need be felt. Some milder forms of toxæmic kidney may simulate functional proteinuria, so that a search should be made for toxic foci, such as septic tonsils, tuberculous glands, or chronic appendix trouble, in all cases. A holiday is advisable if the patient has been doing hard mental work, as the condition is apt to appear under the strain of competitive examinations. A tepid bath, with cold sponging down the spine, and followed by vigorous towelling, is advantageous, and a general tonic such as strychnine, with iron if there is anæmia, should be prescribed. The condition soon rectifies itself when adolescence is past, and any case of proteinuria in a patient approaching thirty probably does not fall into this category.

2. *Febrile*.—Any acute specific fever may be accompanied by proteinuria due to cloudy swelling of the kidney. It should subside soon after the temperature falls to normal. This type of albuminuria is referred to again under the heading of Toxæmic Kidney, to which it more properly belongs.

3. *Congestive*.—In failing heart there is usually proteinuria from venous congestion of the kidneys. Hyaline casts may also be found. Unlike the urine of nephritis the urine is loaded with urates. After an epileptic fit there is often a transitory proteinuria, probably due to the congested condition of the veins during the fit. For a similar reason protein is apt to be present in the urine of any unconscious person.

4. *Toxic*.—This forms an intermediate group between those with and those without organic disease of the kidney, for if the action of the toxin be prolonged a definite nephritis may be established. Thus the proteinuria of pregnancy is generally regarded as toxic in origin, and may clear up

completely. The proteinuria sometimes seen in jaundice is also toxic in character.

II. WITH ORGANIC DISEASES OF THE KIDNEYS.—

1. *Nephritis, acute and chronic.*

2. *Residual albuminuria.*—This term is applied to cases in which albuminuria persists after complete recovery from an attack of nephritis. Observation of the case over a period of years may be necessary to exclude a low-grade progressive chronic nephritis. If and when residual albuminuria occurs it has the same significance as the scar of a perfectly healed wound in the skin. It would seem that residual albuminuria may persist throughout life unchanged, and there is no reason to think that the persistent passage of albumin of itself damages the kidney.

3. *Amyloid disease of the kidneys.*

4. *Tumours and infarcts in the kidney* may cause proteinuria, but more usually simple hæmaturia.

4.—ALBUMOSURIA

Albumose, or more correctly proteose, may be found in urine during autolysis of the tissues. It is not of great clinical importance except to distinguish it from Bence-Jones proteinuria. Proteose can be recognised by the fact that although it is precipitated by saturation with ammonium sulphate it is not coagulated by heat. Proteose precipitates disappear on heating and reappear on cooling. It can be separated from albumin by saturating the urine with crystals of ammonium sulphate, boiling and filtering. The precipitate on the filter paper is washed with water, when any proteose will be redissolved and carried through the filter paper. It can then be detected by the pink colour it gives on the addition of strong caustic soda and a drop of dilute solution of copper sulphate. With these reagents native proteins give a violet colour. The Bence-Jones protein, which is found in considerable amounts in the urine of sufferers from multiple myelomata, is not a true proteose though possessing similar solubilities. On treatment as above it yields a violet colour, showing that it has affinities with native proteins. It begins to be precipitated at 40°–55° C., but on approaching boiling-point most of the precipitate is redissolved. This is probably due to the influence of certain salts in the urine, and is not a property of the isolated protein. As Bradshaw showed, it also gives a ring of coagulum on contact with strong hydrochloric acid. Its recognition is of great diagnostic value, as it is pathognomonic of multiple tumours of the bone marrow, and enables them to be detected before there is any external sign, but only pain and tenderness in the bones. At a later stage the tumours may break through the investing bone and give rise to palpable swellings. Sometimes the Bence-Jones protein is spontaneously precipitated, causing the urine to appear milky. Considerable excess of phosphates may be found in this milky precipitate, probably derived from the autolysis of the surrounding bone.

True peptone is exceptionally found in the urine in pneumonia and phthisis, but is of no clinical importance.

5.—HÆMATURIA

When blood is intimately mixed with the urine it is held to be in favour of its renal origin. Bleeding from the bladder is more apt to occur into the last part of the urine voided, while urethral bleeding is said to occur chiefly into the first part. When the quantity of renal bleeding is not great, it imparts a smoky appearance to the urine, owing to the conversion of some of the hæmoglobin into methæmoglobin, which on spectroscopic examination gives an absorption band in the red in addition to the two bands in the green characteristic of oxyhæmoglobin. The chief causes of hæmaturia are best classified as follows :

1. *Prerenal*.—The altered condition of the blood which occurs, for instance, in scurvy, purpura hæmorrhagica and certain hæmorrhagic fevers, leads to the escape of some of the blood through the kidney without any evidence of a definite kidney lesion.

2. *Inflammations of the kidney*, due to (a) Bright's disease, both acute and chronic. Hæmaturia is a constant feature of acute nephritis and of exacerbations of chronic nephritis. It may also occur in the course of chronic interstitial nephritis and arterio-sclerotic kidney without any acute symptoms. "Renal epistaxis" is usually an early sign of an interstitial change, which is sometimes, as shown by Hurry Fenwick, confined to a single papilla where the vessels are dilated. There are a few cases in which no cause for the bleeding, either in the condition of the blood or the urinary tract, can be discovered in spite of the most careful examination of the kidney, the removal of which has been necessitated by the severity of the hæmorrhage. These are true cases of renal epistaxis or essential hæmaturia.

(b) Tuberculosis or a Bacillus coli infection. The latter more usually affects only the pelvis of the kidney.

(c) Certain drugs, such as turpentine, cantharides and carbolic acid, or occasionally hexamine.

3. *Vascular causes*.—Congestion due to heart failure, thrombosis and embolism (e.g. septic endocarditis) are common causes of hæmaturia.

4. *Irritation of the kidney by foreign bodies*, such as

(a) New-growth.

(b) Crystals, such as oxalates or uric acid, and calculi.

(c) Parasites, such as Bilharzia.

Traumatic, vesical and prostatic causes are not considered here.

6.—HÆMOGLOBINURIA

This is due to some hæmolytic agent. It may be—

1. *Paroxysmal*, as in Raynaud's disease and in syphilis. Most cases are syphilitic. The corpuscles are broken down by a hæmolysin which is present in the blood of 5 to 10 per cent. of cases of tertiary syphilis. Those who suffer from paroxysmal hæmoglobinuria are presumed to have some constitutional peculiarity which renders them susceptible to this hæmolysin. The hæmolysin acts as an amboceptor, unites with the red corpuscle in the cold and on return to warmth the normal complement in the plasma causes hæmolysis. In addition to this there are some rare forms of non-syphilitic paroxysmal hæmoglobinuria.

2. *Toxic.* In this group the toxic agent produces the hæmoglobinuria without an additional factor. Striking examples of this are blackwater fever (*q.v.*), poisoning by arseniuretted hydrogen, and transfusion of incompatible blood. Hæmoglobinuria may also occur in Lederer's anæmia. The chemical tests for hæmoglobinuria are the same as for hæmaturia, but the microscope will fail to reveal red corpuscles. Some of the pigment is excreted as methæmoglobin, especially after drugs of the aniline group, nitrites, or potassium chlorate.

7.—PORPHYRINURIA

Sometimes the hæmoglobin molecule is broken down in the blood stream and the pigmentary portion is excreted apart from the protein and iron. This is usually due to poisoning by sulphonal, trional or sulphanilamide, particularly when the drug has been taken regularly for a long time. It is then of grave prognosis; large doses of alkalis should be given. It is commoner in females than in males. Occasionally porphyrinuria occurs apart from these drugs, when it is not of grave import. It has been met with in cirrhosis of the liver, gastric ulcer and as a congenital abnormality of metabolism, when it may be associated with sensitivity to light and with hydroa vacciniforme. Exceptionally toxic symptoms occur even when it is not associated with drugs, as in two cases recorded by Ranking and Pardington, and by one of us. In these, some intestinal toxin with a reducing action appeared to be at work. The intestinal flora has been found rich in yeasts in such cases. Hæmatoporphyrin sometimes imparts a port-wine colour to the urine, but sometimes it is excreted as a porphyrinate. In the latter case the urine is brown, from the admixture of some unknown pigment, and the spectroscope shows two bands closely resembling those of oxyhæmoglobin. On the addition of an acid, however, the characteristic bands of acid hæmatoporphyrin appear.

8.—CHOLURIA

Another derivative of hæmoglobin, bile pigment, appears in all forms of jaundice due to obstruction of the main or intrahepatic ducts. In a true hæmolytic jaundice, such as acholuria family jaundice, as the name implies, bile does not appear in the urine. Bile pigment can often be recognised by noting the tinging of the froth caused by shaking the urine, but is best detected by the addition of a drop of fuming nitric acid to filter paper dipped in the urine, when rings of colour appear, green being the essential one. The green colour given on addition of a solution of iodine to the urine is a less delicate test. Bile-salts are often absent from the urine when bile pigment is present. Matthew Hay's test is the only reliable one for their presence there. On putting flowers of sulphur on the surface of the urine, they sink to the bottom, owing to the lowering of surface tension by the bile-salts.

9.—MELANURIA

Melanin only appears in the urine in melanotic sarcoma. Garrod has shown that in all other diseases in which melanuria has been recorded the test employed has been unsatisfactory. The melanin is excreted as melano-

gen which darkens on standing, and gives a black precipitate on addition of ferric chloride, which is soluble in excess of the reagent, yielding a black solution. A more delicate test is made by the addition of sodium nitroprusside and sufficient caustic soda to render the urine alkaline. The ordinary ruby-red colour, due to creatinin, is developed. The urine is now made acid with acetic acid, and if melanogen is present a prussian-blue colour appears.

10.—ALKAPTONURIA

This is not the manifestation of a disease, but is rather of the nature of an alternative course of metabolism, harmless and usually congenital and lifelong (Garrod). The individual is incapable of completely breaking down the tyrosin in the protein molecule, so that the intermediate product, homogentisic acid, appears in the urine. The urine reduces Fehling solution on boiling, but it does not ferment, and it darkens on standing, or at once on the addition of alkalis. It may stain the linen brown. When a dilute solution of ferric chloride is allowed to fall drop by drop into the urine, each drop produces a transitory deep blue colour. The urine reduces ammoniacal silver nitrate in the cold, giving a silver mirror on the sides of the test tube. Ochronosis—a blackening of the cartilages and ligaments, and sometimes of the conjunctivæ—may occur, and usually there is also a chronic arthritis, which may lead to a curious “goose-gait.”

[For other reducing substances in the urine, including sugar, see article on Diabetes.]

11.—KETONURIA

Ketonuria is a term used loosely to include the appearance in the urine of diacetic acid and its derivatives, acetone and β -oxybutyric acid. Acetone, however, being merely a decomposition product of diacetic acid, is relatively unimportant; β -oxybutyric acid, formerly regarded as the source of diacetic acid, is more saturated and less toxic and has been shown by Hurtley to be formed out of diacetic acid by the liver, as an attempt at detoxication. Diacetic acid is derived from the incomplete oxidation of fats or of the fatty acid groups in protein. It is probably always made in small quantities, but when there is an abundant consumption of carbohydrate, it is completely oxidised. In starvation the store of glycogen is quickly exhausted and the body chiefly lives on its fats; hence ketonuria. Persistent vomiting, advanced carcinoma of the digestive tract and rectal “feeding” also are equivalent to starvation, and will excite ketonuria, though without such a degree of acidæmia as to cause toxic symptoms. In conditions where the liver is thrown out of gear, such as post-anæsthetic poisoning, ketonuria may occur with toxic symptoms, because of the severe disturbance of all metabolic processes. But there are other agents at work besides diacetic acid which may be responsible for those symptoms. Only in advanced diabetes do we find toxic symptoms directly due to diacetic acid. Here there may be complete inability to utilise carbohydrates, so that the body perforce lives on protein and fats. If these are freely given in the food the amount of diacetic acid produced may be very large. But if a diabetic be fasted there is a great drop in ketonuria, showing that most of this is

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exogenous in origin (see Diabetes). The test formerly used for diacetic acid was the mahogany red colour given on the addition of ferric chloride. This has the disadvantage of being masked if the patient is taking any salicyl body. The nitro-prusside test was formerly regarded as showing the presence of acetone, but Piper demonstrated that it is really a much more sensitive test than ferric chloride for diacetic acid. A crystal of nitro-prusside of soda is dissolved in the urine, and then a strong solution of ammonia is poured on the top. A ring, the colour of Condyl's fluid, speedily develops at the junction of the liquids and spreads upwards. The intensity of colour is a rough measure of the degree of ketonuria. The reaction is made still more sensitive by previous addition of crystals of ammonium sulphate to saturation (Rothera).

12.—DRUGS WHICH ALTER THE COLOUR OF URINE

Methylene-blue is used as a colouring matter of sweets and also as an ingredient of certain proprietary pills. It is also given for coli infections of the urinary tract, gonorrhoea and bilharzia, or less commonly as an analgesic in rheumatism, sciatica and migraine. In small quantities it imparts a green colour to the urine, when it may be precipitated with the mucin. In larger doses it turns the urine blue. It can be recognised by its presence in suspension, so that it can be removed by simple filtration. It can be dissolved from the filter paper by chloroform, and is turned pink by the addition of alkalis. Eosin may be used in sweets and turns the urine a fluorescent pink. Prontosil rubrum and pyridium turn the urine a reddish-orange colour, though, if the urine is alkaline, this may not appear until it is acidified. Amidopyrine (pyramidon) may have a like effect. Rhubarb and senna may turn the urine reddish-brown from the chrysophanic acid they contain. The urine turns pink on the addition of an alkali. Santonin turns the urine a vivid yellow, which becomes rose-pink with alkalis. Carbolic acid may turn the urine greenish-black on standing, from the formation of hydroquinone. In carbolic acid poisoning the urine withdrawn by a catheter may even be found olive-green without exposure to the air. Other drugs, which may have this effect are salol, creosote, naphthalene and uva ursi. In chronic carboloria, ochronosis may occur as in alkaptonuria.

Certain drugs can readily be recognised in the urine by some colour reaction. Thus, salicylates are excreted as salicyluric acid, which gives a violet colour on the addition of ferric chloride. Copaiba, which is precipitated by nitric acid, can be distinguished from albumin by the solubility of the precipitate in alcohol. On the addition of hydrochloric acid a urine containing copaiba turns cloudy, the cloud soon becoming rose pink. Iodides in urine give a blue colour with guaiacum, and on the addition of hydrochloric acid impart a violet colour to chloroform shaken up with the urine.

13.—PYURIA

Pus may come from the urethra, prostate, bladder or kidney. The diagnosis of the source is discussed under septic diseases of the kidney. The best test for pus in the urine is the microscope. If the amount of pus be considerable it will yield a ropy mass on the addition of liquor potassæ.

If ozonic ether be shaken with the urine, bubbles of oxygen are evolved. With tincture of gualacum a blue colour may be given even without the addition of ozonic ether.

14.—CHYLURIA

True chyluria is due to blocking of the thoracic duct, most commonly by the *Filaria sanguinis hominis*, but sometimes the result of inflammatory or neoplastic conditions, with consequent rupture of lymphatics of the bladder through back pressure. Fat may be found in the urine in the lipæmia of diabetes, in growths of the kidney, and after fracture of long bones, when fat may be liberated into the circulation. Accidental contamination by an oily lubricant for a catheter and fraudulent addition of milk to the urine must be excluded. Pseudo-chyluria is due to a lecithin compound of globulin, and is sometimes found when there is a great excess of globulin in the urine. Unlike true fat, this substance is not extracted by shaking up with ether.

15.—PNEUMATURIA

Osler gives the following causes for gas in the urine: (1) Mechanical introduction of air in vesical irrigation or cystoscopic examination in the knee-elbow position. (2) Infection of the urine as by the *Bacillus aerogenes capsulatus*. (3) Vesico-enteric fistula.

16.—CRYSTALLINE DEPOSITS IN URINE

These may be:

1. *Uric acid*, which is characterised by multiplicity of forms and the yellow colour due to the urinary pigment they absorb. The chief varieties found are derived from the barrel and the whetstone types. Thus with a small whetstone stuck at either end of a barrel we get the lemon-shaped crystal. If the whetstones at the end of the barrel are larger, we obtain the "bicycle-handle" crystal. A very characteristic form is that derived from two whetstones with their broader ends apposed. The rosette crystal is a group of whetstones joined by their bases. The factors in the excretion of uric acid are considered under renal calculi; the chief factors in the deposit of uric acid crystals as such are high acidity, high percentage of uric acid, and poverty in mineral salts. The first two are the most important, especially the first. Deposits of urates are usually amorphous, but ammonium biurate may crystallise out as spheres with projecting spines.

2. *Oxalate of lime* is found in the urine, usually as small regular tetrahedra, which under the microscope appear as "envelope" crystals. They may arise (a) from ingested oxalates. Rhubarb, spinach, asparagus and sarrisel are the foods most likely to produce oxaluria sufficient to excite symptoms, for each contain more than 2 g. of oxalic acid per kilogram, though many other articles of diet contain some oxalates. Some individuals seem sensitive to strawberries which, however, only contain 0.06 g. per kilogram. (b) In either achlorhydria or hyperchlorhydria; the former permitting fermentation of carbohydrates, the latter promoting absorption of oxalic acid. (c) In crises in neurasthenics, with irritability, lassitude and neuralgic pains, without discoverable cause.

Oxaluria may cause smarting on micturition and may excite both albuminuria and hæmaturia. Its importance as a starting-point for renal calculi is considered later. Paralytic distension of the bowel has also been described in oxaluric crises.

3. *Phosphates*.—See Characters of Normal Urine (p. 1283).

4. *Cystin* is an amino-acid containing sulphur, and is contained in many proteins, being especially abundant in hair. Its presence in more than minute traces in the urine appears to be due to an inborn error of metabolism, affecting only the endogenous protein, since it is not increased by the administration of cystin by the mouth (Garrod). It is deposited as hexagonal plates, and is often accompanied by a variable amount of diamines, such as putrescin and cadaverin, pointing again to an incomplete breakdown of the tissue proteins. If the urine becomes infected, these cystin crystals may aggregate to form a calculus.

5. *Tyrosin* rarely appears in the urine as sheaves of fine glistening crystals. It is then generally accompanied by *Leucin*, which does not appear until the urine is concentrated by evaporation, when it forms spheres with concentric rings. The presence of these substances is sometimes regarded as pathognomonic of acute yellow atrophy of the liver, but they are occasionally seen in other severe disintegrations of the liver, such as cirrhosis.

17.—ORGANISED DEPOSITS

of red blood corpuscles, pus, epithelium, casts and spermatozoa do not call for detailed description here. The first two have already been referred to. For Epithelium and Casts see sections on Inflammatory Diseases of the Kidney.

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CIRCULATORY DISTURBANCES

1. *Active congestion*.—There is no distinction to be drawn between active congestion of the kidney and the early stage of acute nephritis.

2. *Passive congestion*.—Anything which raises the pressure in the renal vein must produce a passive congestion of the kidney. Failing compensation in valvular disease of the heart is the commonest cause of this; but it may also be brought about by respiratory diseases or by pressure on the renal vein by abdominal tumours or ascites. A transient congestion may result from an epileptic fit.

The cardiac kidney, as it is called, is the most typical example of passive congestion. The organ is firm and dark in colour, especially the pyramids. The capsule strips normally. The stellate veins are engorged. The kidney may drip with blood on section, and if placed in a dish after section soon exudes oedematous fluid.

The urine is scanty, high-coloured and of high specific gravity. Unlike the urine of chronic nephritis it is loaded with urates. It contains a variable amount of albumin and hyaline casts, with a few red blood corpuscles, if the congestion is at all considerable. Renal inadequacy does not reach

the high grade seen in true nephritis, nor is death from uræmia likely. The prognosis and treatment are those of the cardiac condition causing it. Stimulating diuretics are of much more service than in nephritis, since there is no primary disease of the secreting structures.

3. *Infarction*.—This, which is a common complication of infective endocarditis, may take two forms—(a) Multiple minute hæmorrhagic infarcts, producing the “flea-bitten” kidney, which may lead to foci of embolic nephritis with fibrinous exudate and leucocytic infiltration. (b) Larger anæmic infarcts, “map-like” areas of coagulation necrosis, roughly wedge-shaped, but with irregular edges and with the base reaching the surface of the organ. Their formation may cause a sudden pain in the loins, if they are large. Either of these conditions will cause both albuminuria and hæmaturia.

4. *Thrombosis of the renal vein*.—This is rare, and is usually significant of a terminal infection, as in a marasmic infant. In thrombosis of the inferior vena cava the process may reach as high as and spread into the renal vein. This would produce the same effects as the cardiac kidney, but in a much more intense form.

BRIGHT'S DISEASE

Bright described an acute inflammation of the kidney accompanied by dropsy and albuminuria, and a chronic form in which dropsy is absent. There has been much controversy as to what should be included in the category of “Bright's disease,” but there is no doubt as to its essential features. It is a bilateral, non-suppurative affection of the kidneys, accompanied by albuminuria and cylindruria. Except in nephrosis, there is generally hæmaturia in the acute or active stages; œdema and effusion in the serous sacs are commonly present. The renal lesion is diffuse in acute and chronic nephritis, but in “chronic interstitial nephritis” it is chiefly localised in wedge-shaped areas, separated by renal tissue which remains relatively normal. The actual lesion in all forms of nephritis is obviously inflammatory, as shown by proliferation of cells, particularly the cells of Bowman's capsule, the layers of which become adherent while the multiplication of their cells leads to crescent formation. There is also small-cell infiltration and œdema of the interstitial tissue of varying degree. Accompanying these inflammatory changes are degenerative changes, chiefly evident in the renal tubules, namely, cloudy swelling, fatty, hyaline and other forms of cellular degeneration, and necrosis. In some forms of Bright's disease the inflammatory changes predominate; in others the degenerative. In one uncommon form the degenerative changes are so marked a feature of the histological picture, while the changes which are without doubt inflammatory are so slight or even absent, that this form is called Nephrosis in contradistinction to Nephritis.

There are other affections of the kidney, such as toxæmic kidney, hyperpietic kidney (benign nephrosclerosis) and senile or atheromatous kidney, which would be better separated from the category of Bright's disease, because in them the disease of the renal parenchyma is neither the first established nor the primary condition of disease. It is, indeed, but part of a widely distributed pathological change in other organs of the body.

They are, however, included in the present classification and description of Bright's disease, because there are intermediate forms which link them to Bright's disease, and because in some cases they develop into Bright's disease.

There is so much overlapping of the various types of Bright's disease that more is lost than gained by pressing distinctions in detail. Volhard, Van Slyke and others recognise three types of Bright's disease which are essentially different in their genesis and pathological nature, namely (1) the hæmorrhagic or glomerular, marked primarily by glomerular inflammation, with hæmaturia and usually diminished renal function (even in the acute stage); (2) the sclerotic disease, marked primarily by pathological changes in the small arteries of the kidneys (and usually other organs), with hypertension as the first sign, and diminished renal function only as a terminal phenomenon; and (3) the degenerative disease or diseases, called nephrosis, marked primarily by degenerative changes in the kidneys, without hypertension or hæmaturia. This classification has long been the accepted basis for the description of Bright's disease, and in our opinion it best harmonises the anatomical, pathological and clinical phenomena.

I. DEGENERATIVE FORMS:

(A) *Toxæmic kidney.*

(B) *Nephrosis.*

II. INFLAMMATORY FORMS.

(A) *Glomerulo-tubular nephritis. (Diffuse nephritis).* 1. Acute nephritis. 2. Chronic nephritis: (a) secondary type; (b) primary type.

(B) *Embolic focal nephritis* set up in infective endocarditis.

III. VASCULAR GROUP.

(A) *Chronic interstitial nephritis, including malignant nephrosclerosis.*

(B) *Hyperpietic kidney* (benign nephrosclerosis).

(C) *Senile or atheromatous kidney.*

TOXÆMIC KIDNEY

Definition.—Certain toxic substances may excite degenerative rather than inflammatory lesions in the kidneys, which are nevertheless capable of complete recovery. Characteristically, as in febrile albuminuria, the affection of the kidneys is dependent, both for its inception and persistence, on some other disease, and, generally speaking, its intensity varies with the severity of the primary disease. In its onset, intensity, course and termination, it simply reflects the toxæmia which causes it.

Ætiology.—The commonest cause is bacterial toxæmia. As fever in itself does not necessarily cause albuminuria, all the so-called "febrile albuminurias" should be referred to this group. Thus the acute specific fevers—pneumonia, typhoid fever, diphtheria, small-pox, tonsillitis and scarlet fever (notwithstanding the fact that the two last often cause a true nephritis)—are common causes of the slighter degrees of toxæmic kidney. More potent are exogenous and endogenous poisons. Mercurial salts, arsenic,

phosphorus and cantharides are important causes clinically, while uranium and bichromate salts are frequently used in the experimental production of the condition. Jaundice and the ketosis of diabetes mellitus are not uncommon causes. The toxæmias of pregnancy belong to this group, but in their tendency in some cases to develop into chronic nephritis and their frequent association with a raised blood pressure and visual disturbance, these cases differ from other members of the group.

Pathology.—The post-mortem appearances are not distinctive. The cardio-vascular system is normal. The kidneys are pale and likely to be increased in size and weight. On section the cut surface of the cortex is pale in contrast to the congested pyramids: it is increased in thickness and its structure is blurred. On microscopical examination the parenchyma shows degenerative changes, particularly affecting the convoluted tubules. Apart from the presence of some swelling of the glomerular tufts, and the presence of an albuminous exudate in the intercapsular space, the glomeruli show little damage. The absence of tissue reaction that is undoubtedly inflammatory and the presence of tissue changes that are certainly degenerative are the distinctive features of the histological picture. Similar changes are to be found in other organs of the body, and especially in the liver, which may show various degrees of damage, namely, cloudy swelling, fatty degeneration and focal or diffuse necrosis.

Symptoms.—When due to bacterial toxæmia the condition does not give rise to symptoms. It is recognised by the presence of a trace or cloud of albumin in the urine on boiling, and by the presence, in the centrifuged deposit, of granular, hyaline and epithelial casts. In addition, there may be a few white blood corpuscles; when red blood corpuscles are present, or when there is frank hæmaturia, the differential diagnosis from an acute nephritis cannot be made with certainty. In the severer types with insidious onset, as in mercurial poisoning or in the toxæmia of pregnancy, the first symptom is often malaise, disturbance of digestion and constipation, accompanied by albuminuria and oliguria. Headache is a prominent symptom, and is often persistent. In the toxæmia of pregnancy a rising blood pressure or œdema may be the initial sign; the œdema is either general or it appears first in the lower extremities, as in cardiac œdema. Eye symptoms are important; there may be dimness of vision and flashes of light before the eyes, or rarely sudden blindness. On examination of the fundus oculi the disc may appear normal, or there may be œdema of the disc or partial detachment of the retina. The vessels are normal and hæmorrhages are rare. These symptoms may be followed by fits (hypertensive crises), but sometimes the fits occur without previous warning. In general, the symptoms of a fully developed case are clinically indistinguishable from those occurring in the uræmia of true nephritis. The urine contains up to 3 or even 4 per cent. protein. Hyaline, granular and epithelial casts may be present, and may be very numerous; white blood corpuscles may be present, though few in number. In severe cases, the urine contains blood-cell casts, the result of capillary thrombosis and extravasation of blood.

Diagnosis.—The diagnosis depends on the recognition of the signs and symptoms of kidney disease in a patient affected by one of the known causes of toxæmic kidney, and on certain biochemical tests by which a true nephritis can be reasonably excluded. In milder cases, the possibility of the symptoms

being due to heart failure must be excluded. Complete recovery is in favour of the diagnosis of toxæmic kidney. Exacerbation of a chronic nephritis can be recognised by a history of previous nephritis and the presence of definite cardiac hypertrophy and arterial changes. The difficulties are sometimes considerable, however, since a marked rise of blood pressure may occur in a toxæmic kidney, while advanced degrees of chronic nephritis may occur without cardiac hypertrophy, increased blood pressure or clinical evidence of arterial disease. The blood urea is normal in the toxæmic kidney; whereas in chronic nephritis the blood urea tends to rise, and may reach 300 mgm. or even more. In the toxæmia of pregnancy, the appearance of albuminuria in the early months of pregnancy is in favour of the condition being one of chronic nephritis, whereas the albuminuria due to toxæmia generally makes its first appearance in the later months.

Prognosis.—The importance of recognising the toxæmic kidney is that both the immediate and ultimate outlook are better than in nephritis of apparently equal severity. The prognosis in the single case depends on the nature of the cause, the degree of its severity, and the possibility of its early and complete removal. Recovery, when it occurs, is complete, but in the severe cases chronic nephritis may supervene.

Treatment.—If not already in bed on account of the condition responsible for the toxæmic kidney, the patient should be immediately confined to bed. Treatment is directed towards eliminating the toxins and resting the kidneys. Barley water and milk and soda should be given. An easy but not loose evacuation of the bowels must be secured daily by the use of magnesium sulphate, jalap, senna, or compound liquorice powder. A simple diuretic and diaphoretic mixture, such as potassium citrate grs. 15, liq. ammon. acetatis min. 60, sp. ætheris nitrosi min. 15, aq. chloroformi ad $\frac{1}{2}$ oz., is given in water every four or six hours. The intake of solids is limited. At the same time, since there is no retention of urea in the blood, it is unnecessary strictly to limit the intake of protein. Soups, meat extractives and condiments are to be withheld. The action of the skin may be stimulated with hot packs or hot baths. The fits are best treated by venesection. The above outline of treatment is for the severer cases; for the febrile albuminurias, special treatment for the renal condition is not required.

NEPHROSIS

This form of Bright's disease is characterised by cedema, marked albuminuria, and two characteristic changes in the blood, namely, a fall in the plasma albumin and increase of cholesterol. It is distinguished from the glomerulotubular type of acute nephritis by the absence of hæmaturia, cylindruria, hypertension and urea retention, and also by the fact that anæmia is less often present in nephrosis or, if present, tends to develop only at a late stage.

Ætiology.—In most cases no ætiological factor is established, and the first evidence is the onset of cedema without previous illness. In the remainder the best established cause is syphilis. Tuberculosis and osteo-myelitis may be ætiological factors. There may be a recent history of chill, of upper respiratory tract catarrh, or of gastro-enteritis. It may be that streptococcal

infection, more especially of the upper respiratory tract, is a cause of the complaint.

Pathology.—The condition of the kidney is that of toxæmic kidney in a more severe form, the fatty changes in the tubules being very marked.

Symptoms.—The disease is generally first recognised by the gradual or rapid onset of œdema, which gradually increases and tends to become massive. The œdema may be generalised, affecting the scalp, hands, trunk and legs. It is often first noticed as puffiness of the eyelids, or it may first appear as a swelling of the feet and ankles extending up the legs. The patient may feel quite well apart from the disability caused by œdema. On the other hand there is more usually complaint of malaise and fatigue, loss of appetite and nausea, and sometimes of epigastric pain. There may be cough and slight shortness of breath due to slight bronchial catarrh, œdema of the lungs or hydrothorax. Swelling of the abdomen may be the result of œdema of the abdominal wall or ascites. The face is pale and the eyelids and cheeks are puffy, but the mucous membranes are of a good colour, and the blood count is normal. (The urine is reduced in quantity, its specific gravity is normal or raised, it contains a large amount of albumin, often amounting to 0.5 or even 1 per cent., and readings of 4 per cent. or even more have been recorded. The urinary deposit contains but a slight excess of cells and few or no casts. Red blood corpuscles are generally absent. The heart and blood pressure are normal, but a pericardial effusion may develop. There is no retinitis and no urea retention. Characteristic changes are found in the blood. The plasma albumin falls more considerably in nephrosis than in other forms of Bright's disease. The normal figures are plasma albumin 4.1 g. per cent., globulin 2.6 g. per cent., total protein 6.7 g. per cent., which gives an albumin-globulin ratio of 1.6 to 1. In nephrosis, plasma albumin may be 2 to 1 g. per cent., globulin 2.6 to 3 g. per cent., so that albumin-globulin ratio varies between 1:1 and 1:3. The blood cholesterol is raised to 300–800 mgm. per cent. (normal 130–200 mgm. per cent.).

Course.—The disease pursues a chronic course. In the first stage there is a gradual increase of symptoms. When the disease is fully developed it may remain more or less stationary for a number of months, at any time during which there may be some exacerbation or remission of symptoms. During an exacerbation the symptoms increase and convulsions may occur. After remaining stationary for some time, even up to 6 or 12 months, there may be a gradual remission of symptoms, and then complete recovery. In other cases an intercurrent infection, such as pneumococcal peritonitis, broncho-pneumonia, or erysipelas, is responsible for a fatal termination. Or the clinical picture of the disease may gradually take the form of chronic nephritis, in which case the œdema tends to become less, lethargy and fatigue increase, anæmia develops, the blood pressure gradually rises, cardiac hypertrophy follows, and death results from uræmia.

Diagnosis.—The differential diagnosis from nephritis is made on the absence of hypertension and hæmaturia, and the presence of a normal or low blood urea. The fall in plasma albumin and the rise in blood cholesterol are both greater in nephrosis than in nephritis. In nephrosis, cylindruria is relatively slight or absent, and anæmia is uncommon. The differential diagnosis from amyloid kidney may not be possible during life. The presence of splenomegaly, anæmia, and especially a chronic infection, such as chronic

osteo-myelitis (a potent cause of amyloid disease), would be in favour of the diagnosis of amyloid change.

Prognosis.—The importance of the recognition of nephrosis depends largely on the fact that complete recovery may occur even after the disease has been present for many months.

Treatment.—The possibility of a toxic cause, particularly syphilis, and to a slight extent bacterial or other toxæmia, should be borne in mind. Syphilitic cases may respond to specific treatment, which, however, must be prescribed with caution. Obvious sources of sepsis should be removed wherever possible. During the first stage of the disease, and indeed as long as there is good hope of recovery, the patient should be kept in bed and nursed between blankets. Fluid intake is limited to that short of causing thirst. During the early stage, when there may be some doubt as to the differential diagnosis from subacute nephritis, the patient should be put on a diet of low protein and salt content. When the diagnosis is established, and the low plasma protein confirmed, adequate protein is given to maintain nitrogen equilibrium. This means something more than 1 gr. per kilo of body weight per diem: in some cases a high protein intake seems to be beneficial, though this treatment has not fulfilled the expectations originally formed. It is not usual now to order a completely salt-free diet, though it is advisable not to permit the addition of salt at the table. If the blood cholesterol is high, the intake of fat should be restricted. The bowels are regulated with magnesium sulphate or some other bland laxative, such as senna pods or compound liquorice powder. Constipation and loose stools are both to be avoided. In some cases the administration of potassium salts is effective in producing diuresis and reducing the œdema, but must be given very cautiously to avoid depressing the heart. The maximum dose given is 120 grs. daily of a mixture containing equal parts of potassium bicarbonate and potassium citrate. Perhaps the best diuretic in such cases is urea by the mouth in doses of 30 to 60 grs. three times a day; considerably larger doses are sometimes given, but in that case the blood urea should be watched. Thyroid has been advocated by some observers. It acts presumably by raising the basal metabolic rate, which is often low in nephrosis. When other means have failed and considerable œdema persists, decapsulation should be seriously considered. It is quite often temporarily or even permanently effective in curing, or at least greatly reducing, the œdema. It has not, in our experience, affected the albuminuria so dramatically as it may the œdema, but on occasion it has seemed to determine a favourable turn in the course of the disease. Opportunities for symptomatic treatment should be looked for, such as the treatment of anæmia with iron, or some slight degree of heart failure with digitalis, or the control of sleeplessness, loss of appetite and dyspepsia, depression and nervous agitation.

ACUTE NEPHRITIS

The classical form of acute nephritis is hæmatogenous in origin and essentially glomerulo-tubular in distribution. Such a definition would exclude an ascending infection of the tubules from the pelvis of the kidney, such as occurs in pyelonephritis. It would also exclude the embolic nephritis of

infective endocarditis, where inflammatory foci are set up in the kidney as the result of septic emboli reaching it from the heart. These produce marked fibrinous exudation and infiltration with leucocytes; but only some capillaries in some of the glomeruli are affected.

Ætiology.—Acute nephritis was formerly not a common disease. Herringham found, at St. Bartholomew's Hospital, where the average number of medical cases is 7000 a year, that there were, in a period of 9 years, only 166 cases, 120 being in males. On the other hand, a large number of cases occurred in the epidemic of acute nephritis in the War of 1914–1918, 1500 being recorded in Flanders alone during 1915. Since the War of 1914–1918 it would appear to have become more frequent.

The causes usually given for acute nephritis are as follows:

1. **ACUTE SPECIFIC FEVERS.**—Scarlatina is undoubtedly the commonest specific fever to produce it. Goodall found nephritis in 8·4 per cent. of all cases of scarlatina. Nephritis is an occasional complication of typhus, small-pox, chicken-pox and mumps. Syphilis, malaria and yellow fever may also cause it. Many cases of nephritis are preceded by tonsillitis, or otitis media, and it is probable that the throat is often the door of entry for the infection.

2. **DISEASES OF THE RESPIRATORY TRACT.**—It may also occur as a complication of other acute infections of the respiratory tract. The commonest bacterial agent is the streptococcus.

3. **DISEASES OF THE SKIN.**—The frequency with which acute nephritis may follow burns or extensive skin diseases is interesting, in view of the physiological connection between the kidney and the skin. It is a not infrequent complication of erysipelas, impetigo, boils, pemphigus and dermatitis. It must be remembered, also, that children who have been burnt are very liable to develop true scarlatina as well as a mere septic rash, and that streptococcal infection may be the responsible agent.

4. **DISEASES OF OTHER SYSTEMS.**—Acute nephritis may also be a complication of acute infections of other systems. Purpura, which is probably toxic in origin, may be accompanied by a true nephritis.

5. **EPIDEMIC TYPE.**—In the American Civil War and in the War of 1914–1918 acute nephritis occurred as a primary disease in an epidemic form, characterised by dyspnoea at the onset, and in general by a benign course. In the fatal cases, inflammatory and thrombotic lesions were found in the lungs and spleen.

It is a very common idea that cold or chill is a cause of acute nephritis. The statistics of the army epidemic go far to disprove this. For, during the first winter, when there was much wet weather, and the men were much exposed, cases were few and far between, and not until the weather was better did the disease assume epidemic proportions. On the other hand, a patient who has nephritis is more susceptible to cold, which may provoke an exacerbation. Where exposure seems to be responsible for acute nephritis, examination will generally reveal some definite evidence of an old-standing lesion of the kidneys. Conformably with that, after the first winter of the war, there was an agreement between the incidence of nephritis in the army and low temperature.

Pathology.—The kidney is swollen, with occasional punctiform hæmorrhages over a pale, greyish surface. The cortex is increased and, on section, its pallor contrasts with the deep red medullary cones. Microscopically, the

glomeruli are swollen, becoming pear-shaped and protruding into the first part of the convoluted tubules, with Bowman's capsule tightly stretched over them. In these glomeruli the nuclei are less distinct, and the capillaries show fatty changes in their walls. The capillary loops become filled with exudate and empty of red blood corpuscles; their lumina contain a fine network of coagulated substance and leucocytes. There is proliferation of the endothelial cells, and mitotic figures are not infrequent. A serous exudate and a varying number of red and white blood corpuscles may be extravasated between the layers of Bowman's capsule. The convoluted tubules have their lumen blocked either by the swelling of their epithelium or by debris, casts and blood. The interstitial tissue is swollen and œdematous, with hæmorrhages here and there, and sometimes lymphocytic infiltration. The arteries of the kidney show little alteration except that some of the afferent arterioles share in the glomerular changes.

Symptoms.—The onset is usually acute, though occasionally it may be rather insidious. In the latter instance the patient may complain of biliousness, nausea, vomiting and abdominal pain, with headache and sometimes diarrhœa before the onset of renal symptoms. In the cases with acute onset, he may have more or less severe pain in the back, and œdema soon develops. It usually starts in the face; the legs and scrotum are generally involved next, and the swelling soon spreads all over the body. Occasionally the dropsy is curiously localised and fugitive. Though dyspnœa is not regarded as a common feature of acute nephritis apart from uræmia or cardiac failure, in the army epidemic it was almost invariable at the onset. As a rule, shortness of breath started at the same time as the dropsy, but did not last so long, having ceased at the end of 2 or 3 days. There is usually only slight fever, though occasionally a temperature of 102° or 103° may be reached. Some irregularity of temperature, however, is common in the first week or 10 days. The pulse may be raised in tension and the blood pressure is generally raised. Occasionally the serum is milky, as was pointed out by Bright. The skin may be dry and itching, with occasionally a papular or erythematous eruption. Retinal hæmorrhages rarely occur.

The urine is greatly reduced in volume, and may be entirely suppressed. Eight to 12 ounces would be an ordinary figure. It is dark in colour and usually contains obvious blood. This may render the urine as dark as porter, but it may be bright red or merely smoky. Sometimes the blood forms a flocculent, reddish-brown precipitate. The urine is usually loaded with albumin, and casts will be found on microscopical examination. At first blood casts and epithelial casts will alone be found; but, at a later stage, granular and hyaline casts will appear. Fatty casts are not found in the first attack of acute nephritis. Their presence suggests a recrudescence of a chronic disease. Isolated renal cells, transitional epithelium and squamous cells from the lower urinary tract are also commonly found. Micro-organisms are not usually observed, and their presence in any number would suggest that the case is more probably one of pyelonephritis. A sudden rise in the secretion of water after a few days is usually a sign of definite improvement.

The sedimentation rate is raised in acute nephritis, and, in favourable cases, its fall is closely related to the reduction in hæmaturia, but when there is continued activity of the disease-process a raised sedimentation rate is the

rule (Oakley). In the presence of nephritic œdema the sedimentation rate is very high in contrast to the low rate of cardiac œdema. This is due to the alteration in plasma proteins in the former condition, and especially to the increase in plasma fibrinogen.

Complications may be due to three main causes.

1. *Renal failure, i.e. uræmia* may develop. Some slight uræmic symptoms are common in acute nephritis, such as headache, dizziness, nausea and vomiting. But any of the forms of uræmia described later may assert themselves. Convulsions are the most common of the severe symptoms, but are not as grave in significance as in chronic nephritis. If treated promptly, recovery may follow.

2. *Extension of the œdema*.—Water-logging of the lungs may occur, producing serious dyspnoea; but this is sometimes chiefly due to cardiac failure. In any case it is serious. A milder degree of bronchial catarrh is quite common. A rare but very dangerous complication is œdema of the glottis, which calls for prompt treatment.

3. *Secondary infections*.—The subjects of nephritis are always liable to secondary infection, and these are particularly apt to affect the serous membranes; therefore pleurisy, pericarditis, and peritonitis are not uncommon complications. The last two are very dangerous.

Sequelæ: If complete resolution does not occur, the patient will develop chronic nephritis of the secondary type (see p. 1309).

Diagnosis.—The combination of dropsy, albuminuria, hæmaturia, casts and scanty urine usually makes the diagnosis quite easy. The differential diagnosis of acute nephritis from an exacerbation of chronic nephritis may be difficult. Definite evidence of cardiac hypertrophy and arterial changes would be in favour of the latter. The presence of granular casts at the outset, or of fatty casts at any time, is suggestive of chronic disease. An infarct in the kidney which causes a pain in the back and hæmaturia may simulate nephritis, but general dropsy is not likely to occur, nor are casts present in the early stages. Great reduction in the volume of urine is not usual. It must be remembered, however, that infarcts may start foci of nephritis.

Signs of septic endocarditis would suggest infarction. In chronic interstitial characteritis there may be a smart hæmorrhage, but the abundant urine of low gravity and the cardio-vascular signs would lead to a correct diagnosis. In the hæmaturia and the cardio-vascular signs would lead to a correct diagnosis. In the hæmaturia and the cardio-vascular signs would lead to a correct diagnosis.

It is a very confusion with acute nephritis is not likely to occur. Moreover, The statistics of the could not be found, though a large blood cast from the first winter, when this is a very characteristic feature. Pyelitis may give rise to exposed, cases were severe may be small hæmorrhages, especially at the beginning better did the disease of micro-organisms in a catheter specimen and abundant a patient who has nephritis only a haze of albumin, in the absence of casts, will an exacerbation. When diagnosis clear. Moreover, general dropsy does not occur examination will general up severe nephritis as a sequel. In any case of hæmorrhage of the kidney when it is associated with profound constitutional disturbance, there was an eight, tachycardia, continued fever and peripheral neuritis, and low temperature, peri-arteritis nodosa must be considered (q.v.). Lastly, in

Pathology.—Especially streptococcal and complicating an ascending infection rages over a pale, glist, the possibility of acute interstitial nephritis should be its pallor contrasts with the diagnosis will be suggested by the presence of albumin-

uria and oliguria, or by symptoms of uræmia complicating septicæmia. The diagnosis is usually made post mortem.

Prognosis.—The prognosis naturally depends on the severity of the disease. It is better in those cases where there is a discoverable cause, an acute onset, and where the patient comes under treatment promptly. Recovery is usually slow, and the criterion of the cessation of the acute stage is the disappearance of red blood corpuscles from the urine.

Volhard distinguished a separate form of acute nephritis under the name of acute focal nephritis. Its onset is sudden, and recovery is the rule. It is recognised clinically by the presence of hæmaturia and albuminuria without œdema, hypertension or urea retention. There is little constitutional disturbance. It is more frequent in children than in adults, and it is said to occur in epidemics. Its ætiology is the same as that of acute diffuse nephritis. It is doubtful if it can be regarded as a distinct clinical entity, but we recognise it to this extent that acute nephritis having these clinical features carries with it a good prognosis, and recovery is to be anticipated in 2 to 4 months. Mild cases recover sooner. When in addition to the above clinical picture there is added hypertension, urea retention, moderate albuminuria and some degree of anæmia, the course of the disease is likely to be longer, and 12 months' duration with complete recovery is not uncommon. We have experience, too, of complete recovery after an illness of 2 years' duration in severe cases of the above type. When in addition to the above there is considerable œdema and massive albuminuria with hæmaturia, and with or without hypertension, the ultimate outcome of the disease cannot be foretold, but in general terms a somewhat better prognosis should be given in acute and subacute nephritis than the present condition of the patient seems to justify, especially for the reason that it encourages persistence with treatment. The disease is rarely fatal in the acute stage. In the subacute stage, namely, after the first 3 weeks of illness, it may enter a stationary phase, or become slightly progressive. The patient may die from uræmia, secondary infections or extension of the œdema to vital structures. The longer the duration of the hæmaturia, even if it be a microscopic hæmaturia, the more likely is there to be some permanent damage to the kidney, and the development of chronic nephritis.

Treatment.—(a) **PROPHYLACTIC.**—The best prophylactic measure is prompt and efficient treatment of any infective process liable to set up nephritis. There is evidence to show that the routine administration of alkalis in scarlet fever diminishes the incidence of acute nephritis. The enucleation of obviously infected tonsils, especially when an attack of tonsillitis has been accompanied by albuminuria and cylindruria, is advisable. The early administration of scarlatina antitoxin serum in a severe case of scarlet fever is prophylactic treatment of nephritis complicating this disease.

(b) **CURATIVE.**—The indications are to remove, if possible, the microbic or toxic cause at work and to ensure such physiological rest for the kidney as is practicable; to promote elimination of nitrogenous and saline waste by other channels; to treat complications as they may arise and to correct the resulting anæmia. In this way much may be done to steer the patient towards recovery, although we can do little to control the course of the inflammatory process. The patient is naturally kept recumbent in bed. To counteract the congestive effects of gravity, it is well to move him from

side to side, and occasionally to put him on to his chest. He should be clad in a flannel nightgown, and be placed between blankets to guard against chills and to encourage free action of the skin. The room should be warm and well ventilated. If suppression of urine threatens, dry cups or poultices should be applied over the loins. This measure is sometimes successfully adopted to diminish hæmaturia.

Diet.—In acute nephritis, the danger of overloading the inflamed kidney with nitrogenous substances is hardly sufficiently recognised; whilst in chronic nephritis the dietetic restrictions are apt to be too severe. The dictum that “in acute affections we concentrate our attention on the diseased organ, whilst in chronic cases we keep the general condition of the patient more in view,” applies particularly to the treatment of nephritis. Nitrogen retention is common and a source of danger, so that the free administration of milk usually recommended is open to objection, since cow’s milk contains 4 per cent. of protein, which equals 0.56 per cent. of nitrogen. It will do little harm to deprive the patient of nitrogen for a time, and von Noorden advises restriction of the diet at the outset to fruit juice, water and sugar. Where there is no nausea, toffee is allowed, which, being composed of butter and sugar, throws no work upon the kidney. It is generally appreciated by children and allays hunger. Barley water, with a little milk added, may also be given, and as the patient improves the proportion of milk may be increased. It is quite unnecessary to give anything else for a few days, and the relatives’ fear of starvation may be allayed by explaining the rationale of the treatment. The excretion of nitrogen is reduced to its lowest level by giving a diet of fats and carbohydrates, when it may fall below that of a fasting person, as was shown by Folin; but excess of fat is inadvisable for reasons given under chronic nephritis. It is well to restrict or withhold table salt and substitute a mixture of formates, citrates and phosphates, such as ruthmol. The fluid intake and urine output should be measured, and a written record kept of total quantity of fluid taken in and excreted every 24 hours. The patient should not be allowed to be thirsty, but generally speaking the amount of fluid allowed in the day should not exceed 3 pints for an adult or 30 oz. for a child of 12. There may be a sudden diuresis after some days, and it is a sign of recovery. It is sometimes termed a “critical diuresis,” and after its occurrence the quantity of water and milk taken may be increased. A drink prepared by adding 1 pint of boiling water to 60 grains of potassium acid tartrate, half a lemon, and some sugar, stirred occasionally until cold and then strained, may be allowed throughout in moderate quantities. The citric acid and the tartrate become bicarbonates in the blood and may render the urine less irritating by making it less acid; apparently it is not as easy to render urine alkaline in a severe case of acute nephritis as it is in the normal individual. Beef-tea, broth and meat juices are all to be condemned as imposing work on the kidney with very little corresponding nutritive advantage.

GENERAL TREATMENT.—The bowels are kept open by a daily laxative such as pulv. jalapæ co., magnesium sulphate or sodium sulphate, or a preparation of cascara. It is important to secure an adequate and easy evacuation of the bowels, but loose stools should be avoided. Occasional constipation is better treated with an enema than by a large dose of laxative. The function of the skin is promoted, and protection from chill is secured by

keeping the room warm and well aired. The patient is nursed between blankets, and wears a flannel nightgown or a vest, preferably with long sleeves, under the night attire to which he is accustomed. By this means the skin is kept warm and at an even temperature. In addition, the patient should be sponged with hot water followed by friction with warm dry towels. More drastic measures are seldom called for in acute nephritis unless uræmia is impending, when the hot-air bath may be of service.

In the acute stage stimulating diuretics are contra-indicated. Saline diuretics, such as potassium citrate, may be given. In so far as saline diuretics, such as potassium citrate, produces diuresis, they do it by raising the osmotic pressure of the blood, and thus drawing water from the œdematous tissues. The following prescription is mildly diaphoretic and diuretic :

Pot. cit., grs. 15.

Liq. ammon. acetatis, min. 60.

Sp. æth. nitr., min. 15.

Aq. camph. ad fl. oz. 1. To be taken every 6 hours.

The addition of 5 minims of tincture of digitalis is advisable if the heart's action becomes weak.

TREATMENT OF COMPLICATIONS.—For the treatment of renal failure, see Uræmia. Pleurisy, pericarditis, or peritonitis should be treated on ordinary lines. Œdema of the glottis may call for scarification of the larynx or even tracheotomy.

AFTER-TREATMENT.—Bed is imperative until red blood corpuscles have disappeared from the urine and is advisable until albuminuria has ceased altogether. This may be impossible, since acute nephritis may go on to chronic nephritis, but there is a considerable advantage in prolonging the rest as much as possible. Bread, butter, vegetables, puddings, eggs and then fish may be gradually added to the diet, according to the scale given under chronic nephritis, as the hæmaturia and albuminuria diminish, but abstention from meat is advisable for some time, and meat extracts had better be altogether avoided. If anæmia develops, iron is given in the form of ferrous carbonate 45 grains, or ferrous sulphate 9 grains, daily in divided doses after food. Chills should be guarded against in every possible way, and the loins may be protected by wearing a well-fitting cholera belt.

CHRONIC NEPHRITIS

(a) SECONDARY TYPE.

It is generally agreed that chronic nephritis involving the parenchyma of the kidney is diffuse from the first, though naturally the interstitial changes take longer to manifest themselves. It is certain that when parenchymatous nephritis has existed for any length of time, there will be interstitial change as well. On the view here adopted, "chronic parenchymatous nephritis" or large white kidney is the subacute stage of a glomerulo-tubular inflammation. If the patient lives long enough, the kidney will pass into the contracted stage, formerly known as small white kidney.

Ætiology.—It is most frequently the sequel of acute nephritis, though

the initial attack may have been so mild as to have escaped notice. Severe forms of toxæmic kidney, such as mercury poisoning, and the kidney of pregnancy, as also nephrosis, develop into chronic nephritis if they fail to clear up.

Pathology.—The kidneys are swollen. The capsule strips easily, leaving a smooth whitish-grey or mottled surface on which the engorged stellate veins are very obvious. On section the cortex is increased in thickness and pale in colour; the normally distinct fine radial markings are blurred; the pyramids are relatively engorged and contrast with the pale cortex. Microscopically, the glomeruli are large and irregular, with an increase in the number of nuclei, and individual endothelial cells have undergone hyaline or fatty degeneration. Proliferation of the cells of Bowman's capsule is found with crescent formation, and adhesion of the visceral to the parietal layer of Bowman's capsule. There is peri-glomerular infiltration of leucocytes, and the capillaries outside the glomeruli are engorged with blood, the glomeruli themselves being relatively bloodless. The cells of the convoluted tubules undergo cloudy swelling and fatty degeneration to a greater or less extent. Desquamation of the cells occurs, and the tubules contain hyaline and epithelial casts, or red and white blood corpuscles. In some cases of chronic nephritis the tubule changes are most marked, and the glomeruli are relatively little affected in the earlier stages of the disease. As time goes on fibrosis increases, the kidneys shrink in size, their surface becomes granular, and the capsule thickened and adherent. The kidneys are tough on section; the whole surface is a more uniform brownish colour, or in extreme cases of fibrosis, whitish-grey. The cortex is narrowed; the cut vessels may be a little prominent. Microscopically, many of the glomeruli may have undergone hyaline degeneration and fibrosis. In others, crescent formation is marked; there is increase of fibrous connective tissue around the capsules, and small cell infiltration. The tubules tend to dilate and become tortuous, and their lining cells flattened. In others there is hypertrophy of the tubules to compensate for units which atrophy and disappear completely. In some cases the vasa afferentia undergo intimal hyperplasia and fatty degeneration, and there may be hypertrophy of the media. When the blood pressure was persistently raised during life, the heart, especially the left ventricle, will be found hypertrophied. The aorta is thickened, and ordinary atheromatous changes may occur at an unusually early age.

Pathology of œdema in chronic parenchymatous nephritis.—Dropsy is one of the most characteristic features of the disease. Various explanations have been given of its causation. One of the earliest was that it was due to hydræmia from retention of water which the kidney could not excrete. But even total anuria need not cause dropsy, and Rowntree has shown that in glomerulo-nephritis the blood volume may be within normal limits. The next hypothesis was that the capillary endothelium was damaged by toxins, and therefore became unduly permeable (Cohnheim). It has been shown experimentally, however, that such damage may actually hinder the passage of fluid from the blood to the tissues. Widal attributed œdema to the defective elimination of salt by the kidney, which led to accumulation of water by raising the osmotic pressure of the tissues. But if water is retained, salt must also be retained, and when diuresis is produced, salt is excreted also. An important observation was made when Epstein showed that a feature peculiar to nephrosis and to chronic nephritis with marked

cedema was a great reduction in the protein content of the blood and exudates, almost entirely affecting the albumin, so that the amount of globulin is always increased relatively and sometimes absolutely. The daily drain on the protein may even amount to 10 per cent. of the total protein in the blood. This causes a fall in the osmotic pressure of the blood, giving the tissues the controlling power to absorb and retain fluid. In support of this view it may be mentioned that the cedema produced in perfusion experiments with normal saline or Ringer's solution is prevented by the addition to the perfusing fluid of colloids which are in osmotic equilibrium with the colloids of the lymph and tissues. A weak point in this hypothesis is its failure to explain the dropsy of acute nephritis, which comes on long before any depletion of the proteins of the blood can occur.

But the most usually accepted explanation of renal dropsy to-day is an alteration of the affinity of the tissue cells for water as the result of an altered salt metabolism, especially in respect of the sodium ions. In other words, the cedema is regarded mainly as a result of damage to the extra-renal tissues by the same agent that damaged the kidneys, rather than as a consequence of the failure of renal function.

That the blood serum in nephritis may be milky was noted by Bright, and subsequent observers have called attention to pseudochylous ascites in this disease. This is due to the increased cholesterol content, which is more marked in this type of Bright's disease than in any other, except nephrosis.

Symptoms.—These may be continued from those of acute nephritis. More usually there is an interval of apparently normal health. Then the patient begins to suffer from languor and digestive disturbances, followed by the combination of anaemia and dropsy, which gives rise to a very characteristic aspect. Hence the saying "large white kidney, large white man." The dropsy may extend to the serous sacs. The urine is scanty, probably 20 ounces or less in the day; its specific gravity is high, but urates are not so abundant as in the urine of the cardiac kidney. It contains a large amount of protein, usually about 0.5 per cent., as measured by Esbach's method. Numerous tube casts will be found on sedimenting the urine, epithelial, fatty, granular and hyaline forms all being present. Red blood corpuscles may be found from time to time. Examination of the blood may show no increase in the blood urea.

Vomiting and diarrhoea are common and troublesome. Ulceration of the colon, probably due to the vicarious elimination of toxins by the bowel, is an occasional and dangerous complication. Areas of exudate, known as "cotton-wool" patches, and cedema of the optic disks—constituting albuminuric retinitis—may be found in severe cases. There may be dyspnoea, due to acidæmia, the result of diminished excretion of acid sodium phosphate. Secondary infections of the lung, pleura, pericardium or peritoneum may occur.

If hypertrophy of the heart and a rise of blood pressure fail to take place, the outlook is very grave, and death from uræmia or secondary infection is likely to close the scene. If, on the other hand, the blood pressure rises, the heart hypertrophies, the dropsy subsides, and a more chronic stage supervenes. The output of the urine then increases, probably up to 80 oz. or more; the specific gravity being persistently low. This is due to failure of the kidney's

capacity to concentrate the urine. The quantity of albumin is very variable, but is always more than that of "chronic interstitial nephritis." Epithelial, fatty, granular and hyaline casts continue to appear unless they are disintegrated by the extreme dilution of the urine. Towards the end the secretion is sure to fail, and uræmia is likely to follow. Signs of cardiac hypertrophy can be detected, and the blood pressure is generally raised to something between 160 and 220. Albuminuric retinitis is more likely to occur now. Later, silver wire arteries, retinal hæmorrhages, which are often flame-shaped, and even glistening white patches are sometimes to be noted. Infarction of the lung may occur, causing pain, dyspnœa, hæmoptysis with signs of consolidation, and perhaps pleural friction. It results from detachment of a clot in the right auricular appendix, and, being generally due to a secondary infection, marks a definite step downwards. There may be other signs of infection, such as pericarditis or peritonitis. But in the absence of complications life may be prolonged for several years.

Diagnosis.—The combination of dropsy, anæmia, albuminuria and cylindruria generally makes the diagnosis of chronic nephritis easy. In the dropsy with albuminuria of failing heart the œdema first occurs in the most dependent parts, while in nephritis the eyelids are first affected. In cardiac dropsy the liver will probably be enlarged and tender, and the urine will be high in colour and loaded with urates; the only casts it will contain are hyaline; renal function is not seriously impaired. Amyloid kidney may be accompanied by cachectic dropsy; but the heart will not be hypertrophied and the blood pressure is not raised. Moreover, a cause for amyloid disease, and the presence of amyloid disease elsewhere, are usually obvious.

If there is no œdema the diagnosis has to be made from functional albuminuria, residual albuminuria and chronic interstitial nephritis. Functional albuminuria only occurs before thirty and generally about puberty, albumin is absent from the urine secreted in the recumbent posture, casts are absent, with the possible exception of the hyaline variety, and calcium lactate may clear up the albuminuria for a time. Residual albuminuria, an uncommon condition, is not an indication of a progressive disease. The albuminuria is detected accidentally, there being no symptoms. The blood pressure may, however, be slightly raised. There are no cells in the centrifuged deposit of urine. In "chronic interstitial nephritis" the specific gravity of the urine is very low, and there is little albumin. Unless the heart is failing there will be no œdema. The estimation of blood urica should be carried out whenever there is a question as to renal efficiency. As in acute nephritis the erythrocyte sedimentation rate is always raised.

Prognosis.—The outlook in chronic nephritis is always serious. It is essentially a progressive disease, but with care life may be prolonged for several years. Death may occur from uræmia, heart failure or secondary infections. Retinal changes make the prognosis more serious, and "woolly" exudate with swelling of the optic disc generally foretells death within two years. Retinal hæmorrhages and discrete white patches of degeneration in the retina are, however, of less serious significance.

Treatment.—It is essential, as a prophylactic measure, that the treatment of all cases of acute nephritis should be thorough and prolonged. Septic foci, especially in the tonsils, should be looked for and thoroughly treated, as also should any syphilitic or malarial infection. Confinement to

bed is only advisable during exacerbations, when dropsy is extreme, or when uræmia is threatening. The skin should always receive attention, and patients should sleep between blankets and be careful to avoid exposure to cold and wind.

Diet.—There has been a tendency to restrict the protein intake too much, since there is no evidence that the albuminuria is influenced by the amount of protein in the food. Epstein has urged, indeed, that a high protein diet is indicated in order to raise the low protein content of the blood, while fats should be avoided to diminish lipæmia. That such a diet may markedly reduce œdema is true, but not necessarily by raising the protein content of the blood. Probably the diuretic action of the urea formed from the high protein diet is partly responsible. It is, therefore, wise to estimate the blood urea and, if it is not raised, to carry out the urea concentration test (p. 1286), and only to make use of the high protein diet if this test shows at least 2 per cent. of urea. If it is below that figure the protein intake may be calculated on the basis of 1 G. of protein a day for every kilo of body-weight. Naturally, meat extracts and cellular organs, such as liver, kidney and sweetbread, should be avoided, because they contain a large amount of purin; that has to be excreted by the damaged kidney, which eliminates uric acid with difficulty. This is contrary to the principles of physiological rest but, equally, such restrictions of diet must be avoided as would lead to failure of appetite and consequent wasting, while incapable of diminishing the albuminuria. A much greater variety of diet than is usually allowed might be permitted; cooked eggs and dishes made from eggs may certainly be taken. Raw eggs, however, contain certain indeterminate substances which may irritate the kidney. The distinction drawn between red and white meat is fallacious. Red meat is assumed to be more injurious, presumably because it is supposed to contain more purin, whereas the reddest meat contains far less than sweetbread. Chronic nephritis should not be restricted to milk, which is too dilute a form of food for them, and may increase the dropsy. An entirely salt-free diet is not to be recommended, though moderate restriction in this respect is probably wise. Salt can be replaced by lemon juice or ruthmol. In this way we can avoid increasing the miseries of an incurable disease by unnecessary restrictions. If nitrogen retention exists as shown by estimations of blood urea, a diet poor in protein should be taken on one day in each week. Indeed, a day when the diet is restricted to fruit and sugar is often as useful in chronic nephritis as is the day of vegetable and egg diet in diabetes. But prolonged nitrogen starvation is as bad for the nephritic as for any one else, and in some cases increases the water-logging of the tissues.

Generally speaking, alcohol is inadvisable in any form, and should never be ordered to those unaccustomed to it. In those who have been taking it regularly, deprivation may interfere with appetite, in which case a little well-diluted whisky is probably as innocuous as any form of alcohol can be. But the strictest moderation must be enjoined. Tea and coffee used to be forbidden, because of the methyl-purins they contain, but in our opinion this restriction is unnecessary.

Diuretics.—Diuretics should be used with caution in chronic nephritis. When there is marked œdema the fluid intake should be limited to a litre or perhaps 2 pints in the 24 hours, but the patient should not be allowed to be thirsty. In some cases the administration of potassium salts by mouth acts

as an efficient diuretic (*vide* treatment of nephrosis). Saline diuretics in the form of citrates and acetates may be safely given, provided that the dose is moderate, and that the possibility of alkalosis developing in severely damaged kidneys is remembered. Urea may be given in those cases in which the blood urea is low, and in which the danger of a rising blood urea is not anticipated. The caffeine group of drugs should be employed with caution, and when used they are best given in small doses, and then withheld if diuresis does not result. Theophylline and sodium acetate grs. 4, or theobromine and sodium salicylate (diuretin) grs. 10, administered twice or three times daily may be prescribed if there is no hæmaturia. In general terms organic mercury preparations are contra-indicated in chronic nephritis on account of the danger of mercury-poisoning. Nevertheless, in cases in which œdema is persistent, renal function is adequate and there is an absence of anæmia, injection of mersalyl (salyrgan) may sometimes be used with advantage. Sixty or 120 grains of ammonium chloride are given on each of 2 days before $\frac{1}{2}$ to 1 c.c. of the drug, and, if tolerated, this treatment is repeated at intervals of 5 to 9 days. Œdema in chronic nephritis may be in part due to heart failure. In this case digitalis may prove a valuable and efficient diuretic. A raised blood pressure is not a contra-indication to its use.

A marked feature of chronic nephritis is the defective adjustment of the kidneys to varying water supply. As in acute nephritis, the drinking of large amounts of fluid may, therefore, merely increase the œdema.

As explained under Acute Nephritis, the saline diuretics are the least open to objection; since they draw the extra water from the tissues they cannot increase and may diminish the œdema. Urea as the natural diuretic of the body is often given in doses of 45–60 grains three times a day, when there is no nitrogen retention.

Diaphoretics.—The arguments for and against diaphoretic measures will be found under uræmia. Diaphoretic drugs are not suitable for the routine treatment of chronic nephritis, as a moist perspiring skin renders the patient more liable to chills—always a danger in this disease. A course of hot-air baths may sometimes be helpful when there is evidence of salt retention. If they are followed by diuresis they are doing good.

Purgation.—Although efficient action of the bowels must be maintained, habitual loose stools are to be avoided, because they weaken the patient and promote the absorption of intestinal toxins. The special liability to mercurialism renders calomel unsuitable for routine treatment.

Acupuncture.—The patient is placed in a cardiac bed with the head raised and feet lowered for a day or two before acupuncture is performed so that the fluid gravitates into the lower limbs. The preparation for acupuncture consists of a mackintosh sheet placed under the lower limbs, and arranged to form a chute leading into a pail on the floor: the lower limbs from the knees to and including the toes are cleaned with ether soap, and then with spirit, and placed on sterile towels: the skin is covered with a coating of Lassar's paste. Acupuncture is performed with a medium-sized trocar from a Southey's tube set, the trocar being stabbed through the paste and skin to the subcutaneous tissues. Beginning at the side of the tendo Achillis, 15 punctures are made in line posteriorly in the lower two-thirds of the leg. Two lines of punctures about one inch apart are made in each

leg. Each leg is then wrapped in a piece of sterile jaconet, wrapped firmly round the thigh immediately above the knee, and secured with adhesive tape. Below the jaconet is wrapped loosely round the limb forming a tube which conducts the fluid into the mackintosh gutter at the foot of the bed. Drainage may continue for a week. By this method fluid may even be drained from serous cavities when there is marked dropsy.

Decapsulation.—This procedure is now reserved for the treatment of œdema in the nephrotic type of Bright's disease if adequate treatment on conservative lines has failed to relieve it.

Climate is a valuable help. In this country, Ventnor or anywhere on the south coast from Bournemouth westward is the most suitable climate that can be obtained. Egypt generally suits such patients particularly well. Madeira or California are also quite suitable. The wind and the more violent fluctuations of temperature on the Riviera render it much less advisable.

Treatment of complications.—These are uræmia, heart failure and secondary infections, such as pericarditis, pleurisy, colitis and peritonitis. Their treatment is discussed under those headings.

In conclusion, it must be recognised that the kidney, once chronic nephritis is established, cannot completely recover, and the main thing is to attune the mode of life to a low key, subjecting the patient to as little strain as possible. He may have a considerable variety of food, provided that the intake of protein is regulated in the way described above, and that he takes very little purin and salt. He can be helped by saline diuretics and un-irritating preparations of iron, such as liquor ferri acetatis. He will do all the better if his medical man realises that many of the methods recommended in the treatment of this disease are impotent, where not actually harmful.

(b) PRIMARY TYPE—RENAL DYSBIOTROPHY

Chronic nephritis may develop without any known cause. In such a case there is no past history of acute nephritis; of symptoms of Bright's disease, such as œdema, hæmaturia or pain in the back; nor indeed any history of infection, such as scarlet fever or tonsillitis to which the onset of the disease can be attributed. The complaint may be found accidentally by the discovery of albuminuria in the course of routine examination. More often the diagnosis is first made at a later stage of the disease when there is complaint of asthenia, anæmia, or liability to fatigue. Sometimes, indeed, there are no symptoms of the disease until it has reached the terminal stage of uræmia.

Ætiology.—This form of chronic nephritis probably belongs to the group of congenital-developmental diseases of inborn and familial type, as defined by Parkes Weber. According to this view certain forms of chronic nephritis are inborn constitutional diseases which, though they may manifest themselves soon after birth, may sometimes be delayed in their appearance until years later. When the disease runs its course and ends fatally within a few weeks or months of birth, there can be no doubt of its having been present in utero. When it does not manifest itself until after many years of antecedent good health, we may assume that the disease was only potentially present at birth, the inborn tendency to its development being due to a congenital tissue inferiority or dysbiotrophy. The reasons for this view are : (1) the absence of a discoverable ætiology already referred to ; (2) the not

uncommon familial occurrence of the complaint; (3) the finding post mortem of congenital abnormalities in the kidneys or urinary tract; and (4) that in its course, which is latent, progressive and invariably fatal, the malady resembles many other diseases which belong to this congenital-developmental class.

Familial hæmorrhagic nephritis, which is also hereditary and apparently congenital, is a rare condition. In one family 16 cases occurred among 28 individuals in 3 generations, 8 being in males, 7 of which ended fatally in early life, and 8 in females with only 1 death. The disease evidently runs a more benign course in the female. In one patient the first symptom occurred when only 3 weeks old, in another when 2 years old, while in the remaining cases the onset was quite early in life. Most of the cases have been characterised by recurrent attacks of hæmaturia, sometimes considerable in amount. As such attacks may be preceded by an increased nitrogen output, some inherited form of protein sensitivity seems a probable factor. As deafness may also be familial, it is interesting to note that it was marked in 5 of the 16 patients, and one otherwise healthy member of the family was also deaf. The condition usually terminates in uræmia.

Pathology.—The kidneys are small, pale and fibrosed. One kidney may be smaller than the other. Congenital deformity is not uncommon: occasionally one kidney is absent or represented by only a nodule of fibrosed tissue, from which an atrophied ureter has its origin. On microscopic examination there is diffuse fibrosis of the kidneys; many glomeruli are atrophied, others show varying degrees of inflammatory reaction.

Symptoms.—In the rare cases that appear in infants of a few weeks or a few months old, the symptoms are those of uræmia, namely, dyspepsia and loss of weight leading to diarrhoea, vomiting and cachexia. In those that develop about the age of puberty the disease may cause infantilism (renal dwarfism); and bone deformities resembling rickets often develop, and may be associated with a low calcium content of the blood, leading in some instances to a compensatory enlargement of the parathyroids. In one group of cases there is cardiac hypertrophy, a considerably raised blood pressure, and retinitis. In these arteriolar changes (diffuse hyperplastic sclerosis) are found post mortem. In others the blood pressure is normal or subnormal. In another group (first described by Rose Bradford in 1904) the disease makes its first appearance between the ages of 20 and 30 years. It may appear suddenly in the form of uræmia without warning, and become rapidly fatal. Even in those cases in which the kidneys are found post mortem to be white, fibrotic and greatly reduced in size, with diffuse inflammatory changes of long standing, early adult life may have been apparently perfectly healthy, and symptoms of the disease may have been present for only a few months.

Differential Diagnosis.—Loss of weight, a sense of fatigue, anæmia and dyspepsia, may have been so marked as to raise the question of tuberculous disease or neoplasm. When there is marked asthenia and pigmentation, Addison's disease may be suggested, or the differential diagnosis may lead to the suspicion of pernicious anæmia. In cases with high blood pressure a juvenile form of hyperpiesia must be excluded. When there is marked polyuria with urine of low specific gravity, the possibility of diabetes insipidus arises.

Treatment.—The disease is essentially progressive, and treatment can only be symptomatic.

CHRONIC INTERSTITIAL NEPHRITIS, INCLUDING
MALIGNANT NEPHROSCLEROSIS**Synonym.**—Granular Kidney.

When this term was first used it included a number of clinical entities, which together formed a somewhat motley group. Among these certain forms of chronic nephritis have now been identified as such, particularly the type recognised by Rose Bradford as the small white kidney, and classified by us under chronic nephritis, primary form. Clifford Allbutt identified hyperpiesia, Samuel West and others recognised the senile kidney. Some authors are inclined to think that after excluding these clinical forms only a single entity, malignant nephrosclerosis, remains. "Chronic interstitial nephritis," however, probably still includes more than one clinical entity, and it is for this reason that we have retained the term.

Ætiology.—The causation of malignant nephrosclerosis is unknown, but probably those factors which apply to hyperpiesia already described are concerned. Malignant hypertension, however, tends to make its appearance at an earlier age, and is not uncommon at any age after 35 years, and is frequent between the ages of 40 and 55. According to Ellis it is more common in men than in women. As to the other cases included under the term "Chronic Interstitial Nephritis," the ætiology is unknown, but gout, lead poisoning and alcohol are thought by some to be factors. Syphilis is not a cause.

Pathology.—The kidney tends to be reduced in size, is tough, and red in colour. The capsule is adherent, leaving a finely granular surface on stripping. Sometimes the capsule is thickened and splits on attempting to strip it, thus giving an erroneous impression of a smooth surface. Retention cysts may be seen, both on the outside and the inside of the organ. On section the cortex is reduced; not only is it shrunk from without inwards, making the organ smaller, but the increase of intrapelvic fat shows that it has also shrunk from within outwards. The vessels are unduly prominent. The glomeruli show signs of inflammatory reaction, and the interstitial tissue in their neighbourhood is increased and infiltrated with small cells, generally of the mononuclear type. These areas of disease are patchy, and form wedge-shaped areas, with their apex towards the cortico-medullary zone. The intervening areas of renal tissue show little or no change. The histological lesions in the arteriæ interlobulares and vasa afferentia, as also the patchy small-cell infiltration in the kidneys and evidence in some glomeruli of proliferation of the cells of Bowman's capsules, provide evidence of inflammatory reaction. The cardiovascular changes are those described under hyperpiesic kidney (benign nephrosclerosis). In addition a characteristic lesion is acute arteriolar necrosis, while focal necrosis of the capillary tuft is common. Hæmorrhagic infarction of some glomerular tufts may be found. The distribution of the renal lesion in chronic interstitial nephritis is distinct from that in chronic nephritis, for in the latter the lesion is diffuse and every glomerulus is more or less altered in structure. In advanced cases of chronic interstitial nephritis, however, the extent of structural alteration extends and becomes more diffuse.

Symptoms.—The subject of chronic interstitial nephritis often fails to seek medical advice until the condition has become well advanced, and this

is partly because the body adjusts itself to the changes which are taking place in it as a result of the disease, and partly because the disease may develop very rapidly in its early stages. Common early symptoms are loss of energy, proneness to fatigue, lack of power of concentration, headache and dyspepsia. In other cases the patient feels well, and consults his doctor on account of hæmorrhage. The source of the bleeding is most commonly from the nose (epistaxis), or frank hæmaturia. In other cases it may be a cerebral vascular accident, thrombotic or hæmorrhagic: less often it takes the form of hæmamestesis or melæna. Disturbance of vision due to retinal hæmorrhages, or sudden blindness due to hæmorrhage into the vitreous are not uncommon early symptoms. A woman may complain of bleeding from the uterus, and rarely a blood-stained seminal discharge is the initial symptom in a man. In other cases the initial symptoms are cardiac, and the patient complains of cardiac pain, shortness of breath, or consciousness of the heart's action. Nocturnal frequency of micturition may be an early symptom. On examination the patient will be found to have an increased blood pressure, the systolic ranging between, say 200-240 mm. or even higher, and the diastolic over 120 mm. There is also cardiac hypertrophy, but the case differs from a hyperpietic kidney (benign nephrosclerosis) in one or more of the following ways. Thus, the complexion tends to be sallow, and often there is loss of weight and some degree of secondary anæmia. The urine may contain only a trace of albumin, but more frequently it contains an appreciable quantity, namely 0·1 per cent. or more. The urinary deposit characteristically contains red blood corpuscles and granular and hyaline casts. When the disease is fully established, the urine has a low fixed specific gravity, varying between 1008 and 1012. Further examination may show impairment of renal function, and the blood urea may be raised to 70 mgm. per cent. at least. On examination of the eyes, in addition to arterio-sclerosis of the retinal vessels, retinal hæmorrhages and exudates, cedema of the disc and retina, and some detachment of the retina may be found. Ellis has emphasised the diagnostic importance of papilledema in malignant nephrosclerosis.

There are cases of chronic interstitial nephritis which run a most rapid course in the initial stage. The clinical features are hypertension, cardiac hypertrophy and hæmorrhage. Toxic symptoms make their appearance within 3 to 6 months of the onset, and by this time some degree of failure of renal function may be demonstrated by clinical tests, or the patient's symptoms may already be those of uræmia. The whole evolution of the disease and its fatal termination in uræmia, cerebral hæmorrhage or cardiac failure, may take place within a period of 12 to 18 months.

There are other cases in which there may be a preceding period of five or more years during which time the patient is known to have hyperpiesia, and complains only of cardiac symptoms which are readily explained in terms of it. Meanwhile the disease-process seems to be more or less stationary, and then for no obvious reason the disease takes a progressive form, and a terminal acute phase lasting only 3 to 6 months is ushered in by an attack of hæmaturia or epistaxis, by retinitis, or by symptoms of renal failure, and the toxic picture of chronic interstitial nephritis rapidly develops.

On the other hand, there are chronic forms of the complaint which pursue a varied course different from the grave progressive course of malignant hypertension. The variety of the clinical picture which these cases present

makes their description difficult, and it is to allow of their recognition, and in order to leave the subject open for further clinical and histological observation, that the term "chronic interstitial nephritis" has been retained. Thus, a man complains of malaise, loss of health and strength, undue fatigue or loss of weight. On examination, the chief finding is an appreciable albuminuria. At this stage there may be no abnormal cellular deposit, or there may be a few red blood corpuscles and hyaline casts in the urinary deposit. Renal function may be normal. With limitation of his activities and general measures of medical treatment, the patient may recover his health to a considerable extent, though the albuminuria persists. From now on, over a period of 2 to 5 years, or perhaps longer, reasonably good health is maintained. Sometimes the blood pressure is but little raised. There may be an attack of hæmaturia or epistaxis from time to time. The blood urea may be a little raised, and may even remain more or less stationary at a level of 60 to 80 mgm. urea per cent. for several years, until finally physiological adjustments fail, and uræmia, a vascular accident, cardiac defeat or an intercurrent infection determines the fatal termination.

Complications.—The principal complications are due to failure of the pump, the tubing, or the filter. In other words, the heart may fail, causing venous congestion; the artery may give way, as in cerebral hæmorrhage; or the renal excretion become so inadequate as to lead to uræmia. Glycosuria is sometimes found. This may be due to alcoholic excess, a factor in the causation of the interstitial nephritis.

Diagnosis.—This rests on the combination of urinary and cardiovascular signs. The differential diagnosis from hyperpiesia is discussed on p. 1068, and that from secondary contracted kidney on p. 1312.

Prognosis.—The disease is usually progressive. Its course may be very slow, but after the development of retinitis a fatal ending occurs in the great majority of cases within 2 years. In some cases, however, even including those in which all the characteristic signs of the disease are present, including microscopic hæmaturia, persistent hypertension and retinitis, the disease may reach a stationary phase, and considerable recovery may occur, the fatal termination being delayed on occasion for as long as 6 years. For this reason a definite prognosis should not be given until a late stage in the disease. Tests of renal function afford useful prognostic evidence. Any evidence of cardiac dilatation, or of uræmia, even of the chronic variety, makes the outlook much less satisfactory. Retinal changes are of ill-omen.

Treatment.—In general terms, this should be carried out on similar lines to that for hyperpietic kidney (*q.v.*), though the response is not likely to be satisfactory. In addition, the anæmia often calls for treatment.

HYPERPIETIC KIDNEY

Synonym.—Benign Nephrosclerosis.

In this form of renal disease the vascular changes are of greater importance than the renal, and it will only be necessary to make a brief reference to it here.

Ætiology.—This is the same as that described under Hyperpiesia (see page 1065).

Pathology.—The chief kidney changes are in the small vessels. They consist of thickening of the intima and medial hypertrophy. The former may go on to fatty degeneration and obliteration of the lumen of the vasa afferentia, and so cause ischæmic fibrosis of the glomeruli with atrophy of the associated tubules. Thus, the changes in the renal parenchyma are largely degenerative in character, rather than the inflammatory changes seen in chronic interstitial nephritis. The kidney is slightly reduced in size and is somewhat firmer than normal. On section, the fine radial striation in the cortex is preserved, and throughout the organ the small arteries are prominent. Histologically the essential lesion is a thickening of the intima of the vasa afferentia and the interlobular arteries, with hypertrophy of the media. In the early stages there is cellular proliferation in the intima and increase of hyaline material. At a later stage there is fatty degeneration in the terminal arterioles in contrast to their parent vessels, in which little or no fatty degeneration is found. The thickening of the intima may lead to obliteration of the lumen, with fibrosis and atrophy of the glomerulus and its tubules. At a later stage, too, owing to fatty degeneration and atrophy of the muscle fibres of the media, the media may be actually thinner than normal. These changes, like those described in chronic interstitial nephritis, have a patchy distribution in the organ. The fibrous connective tissue in the immediate neighbourhood is thickened, but there is no glomerulitis and little or no small-celled infiltration, in contrast to the inflammatory reaction found in chronic interstitial nephritis. The vascular changes described above were originally termed arterio-capillary fibrosis by Gull and Sutton. They were first accurately described by Jores under the term diffuse hyperplastic sclerosis.

Symptoms.—These are those of hyperpiesia (see pp. 1066, 1067).

Treatment.—See that of hyperpiesia (pp. 1068–1070), the heart in hypertension (p. 996), and renal uræmia (pp. 1327, 1328).

SENILE OR ATHEROMATOUS KIDNEY

In this form of kidney disease also the vascular changes are of greater importance than the renal, and it is only necessary to deal briefly with the affection.

Pathology.—The kidneys show depressed red areas, which are due to contraction of fibrous tissue along the distribution of particular interlobular arteries, and, therefore, tend to be conical in form, with their base to the surface of the organ. There is an absence of cardiac hypertrophy; the pressure in the diseased arteries falls below that necessary for glomerular excretion. The affected glomeruli accordingly shrink, and the connective tissue around them becomes condensed and thickened. The degenerate glomerulus and its capsule fuse together, and undergo fatty and fibrotic changes. The atheromatous kidney is, therefore, generally due to atrophy following insufficient circulation, with consequent fibrosis.

Symptoms and Diagnosis.—There may be gradual failure of the physical and mental powers—described by Allbutt as “contraction of the spheres of bodily and mental activity”—rather than the more dramatic events of chronic interstitial nephritis. There is a trace of albumin in the urine. The radial artery is thickened and tortuous. The blood pressure is not high, and

there is an absence of cardiac hypertrophy. Death by cardiac failure or intercurrent affections is the commonest ending, while cerebral hæmorrhage and uræmia are unlikely.

URÆMIA

Uræmia is the name which has been given in the past to the toxic state which complicates or terminates severe kidney disease, and in which urea retention occurs. More recently it has been recognised that a high grade of urea retention may develop as a result of extra-renal factors. It is proposed to group these conditions under the name of Extra-renal Uræmia in order to emphasise the fact that although the kidney fails in its function, organic disease of the kidneys is not the primary fault.

EXTRA-RENAL URÆMIA

ALKALOSIS (see also Alkalosis, pp. 405-408).—This is a toxic state characterised by malaise, gastro-intestinal disturbance, and a variety of nervous symptoms due to an increase of the CO_2 combining power of the blood plasma.

Ætiology.—Alkalosis is caused by giving too large doses of sodium bicarbonate or other alkali. The minimum normal tolerance of alkali by mouth seems to be the equivalent of 15 g. of sodium bicarbonate in 24 hours. If a patient develops alkalosis when taking this quantity of alkali or less, some predisposing factor will be found. Such factors are anæmia, kidney disease, or vomiting. Of these factors anæmia is important, because of the action of hæmoglobin as a buffer helping to maintain a constant pH in the blood. With a low hæmoglobin content in the blood an extra tax is thrown on the kidneys, so that in anæmia a smaller quantity of alkali given by the mouth may be the cause of alkalosis. Impaired renal function, such as results from kidney disease, is another factor predisposing to alkalosis, because the kidneys are unable to excrete the excess of alkali. As alkalosis may itself be responsible for kidney damage a vicious circle is set up. It has been suggested that chloride loss and the consequent hypochloræmia is the cause of the rise in blood urea. This explanation cannot, however, account for the whole condition, because there are other conditions in which there is a considerable loss of blood chlorides without uræmia. The uræmia which develops in repeated vomiting, however, is probably due to loss of hydrochloric acid in the vomit.

Symptoms.—The toxic symptoms caused by giving too large doses of sodium bicarbonate or other alkali appear from 4 days up to 4 weeks from the beginning of treatment (Cooke). The patient complains of malaise, dizziness, constipation and headache, which commonly takes the form of a sensation of pressure on the vertex. He becomes nervous and irritable, often resentful. There is loss of appetite, distaste for food, nausea and vomiting. The patient becomes drowsy in the day and sleepless at night. Respiration is slowed, the pulse rapid, the face flushed, and the body perspiring. There is aching or actual pain in the trunk and limbs, with tenderness of the muscles on pressure, and increased muscular irritability. In the severest cases there may be tetany or epileptiform convulsions, and if the condition is unrecognised

the patient may become comatose, with incontinence of urine and *fæces*, and die. In addition to the above symptoms, a dry furred tongue, thirst, and at a later stage diarrhoea are common. The urine is alkaline, except sometimes in the early stages. It contains a trace or cloud of albumin. The deposit may contain hyaline or granular casts, and a few red and white blood corpuscles. Renal function is impaired, as is shown in nearly all cases by urea retention, and in some by failure of the power of urine concentration, which results in the excretion of urine of a constantly low specific gravity. There may also be a considerable polyuria, amounting to 2 or 3 litres in 24 hours. In most cases reported the dose of alkali which has caused alkalosis has been the equivalent of 20 g. of sodium bicarbonate given daily for 4 days or more. More usually the toxic dose has been the equivalent of 30 to 60 g. of sodium bicarbonate daily, and larger doses than this are naturally all the more likely to produce the condition.

Diagnosis.—Alkalosis must be suspected whenever a patient taking alkali develops symptoms of malaise, headache, constipation, digestive disturbance, or change in the personality. The diagnosis is established by an examination of the blood. The blood urea is commonly raised to 60 or 80 mgm. per cent., and may reach the high figures found in renal uræmia. The alkali reserve, which normally varies between 50 and 75 c.c. CO_2 per 100 c.c. of plasma, increases to 90 c.c. or more. The chloride content of both blood and urine may be low. The CO_2 combining power of the plasma is the most important observation to make, because it precedes the rise of blood urea.

Treatment.—The administration of alkalis is immediately discontinued. Acid sodium phosphate, gr. 10, three times daily after food is administered. If the case is under close observation, and if repeated estimations can be made of the alkali reserve, larger doses may be prescribed, but on account of the danger which exists, owing to renal damage, of a rapid change from alkalosis to acidæmia, some authorities are opposed to giving acid in any form. In any case, it is unwise to prescribe ammonium chloride.

GASTRO-RENAL URÆMIA

Repeated vomiting from any cause may induce alkalosis and thus be responsible for uræmia. In acute cases with albuminuria the differential diagnosis from renal uræmia is made clinically on the high specific gravity and high urea content of the urine. In chronic cases a complete blood examination and the observation of the degree of alkalosis may be required to exclude renal uræmia.

Diarrhoea may be responsible for uræmia, both on account of the loss of fluid and the resultant loss of chloride. But in this case the uræmia is associated with the opposite condition of acidosis, as may be established by the estimation of the alkali reserve. In acute cases, treatment is by intravenous injection of 200 to 400 c.c. of a 2 per cent. sodium bicarbonate solution.

RENAL URÆMIA

This type of uræmia belongs to a different category from that of Extra-renal Uræmia, because it is primarily due to severe kidney disease of which it is a complication or a terminal phase. A raised blood urea is the distinguish-

ing feature of uræmia. Nevertheless, the term renal uræmia is used in a clinical sense, both because the diagnosis can be made without an estimation of the urea in the blood, and because, on occasion, the patient may suffer from uræmia before the blood urea is appreciably raised. Renal uræmia varies greatly in its symptomatology, but, as Clifford Allbutt pointed out, it is generally characterised by anæmia, headache, nausea, lethargy, retinitis, convulsions or coma.

Although there are many factors in support of the so-called retention theory of uræmia, its complete explanation in biochemical terms is not yet clear.

In view of the high blood urea in severe uræmia, urea retention might be regarded as the simplest explanation of the condition. It has been shown experimentally that the administration of massive doses of urea causes headaches, giddiness, apathy, drowsiness, bodily weakness, nausea and diarrhœa—a group of symptoms characteristic of chronic uræmia. But that this is not the whole explanation of uræmia is shown by the fact that symptoms of uræmia may be present before the blood urea has risen to a level proportionate to the symptoms. It has been suggested, therefore, that other renal excretory products, such as indican, uric acid and the salts of urine, may contribute to the uræmic state. When the hypobromite method is used for the estimation of urea in the blood or cerebro-spinal fluid other nitrogenous products than urea are estimated. The urease method gives the amount of urea only, so that the difference between the results obtained by these two methods is an indication of the quantity of amine bodies other than urea, and to these bodies some at least of the clinical syndrome is probably due. But even this expansion of the retention theory to include nitrogenous bodies other than urea does not explain uræmia, because in complete suppression of renal function, such as occurs when the ureter of a single kidney is completely destroyed by any cause (Ascoli's *urinæmia*), the clinical picture is entirely different from that of uræmia complicating acute and chronic Bright's disease. Ascoli says: "Severe *urinæmia* in man is chiefly manifested by bodily weakness and languor, which often appear before any other symptoms, but generally lead to progressive mental weakness and exhaustion, often terminating with great suddenness. The greater part of the most prominent symptoms of uræmia are, however, lacking, especially the severe and acute mental disturbances, the sudden amaurosis, and the epileptic phenomena in general. Only in occasional cases do the symptoms resemble uræmia." The name of *latent uræmia* is sometimes given to this condition, but it is hardly suitable. It has also been held in the past that uræmia may be due to some precursor or derivative of urea or other nitrogenous body normally secreted by the kidneys, but there is no convincing evidence of the accumulation of such bodies in the blood at least in such quantity as to cause uræmia.

It is evident that the retention theory and a failure of the excretory function of the kidneys do not provide a complete explanation of renal uræmia. But the kidneys have two other important functions, namely, the regulation of the osmotic pressure of the blood and the regulation of its hydrogen-ion concentration. Now, in uræmia the acid-base equilibrium is disturbed, and there is a fall in the CO_2 combining power of the blood plasma; that is to say, *acidæmia*. If this state of *acidæmia* is corrected by the administration of alkali in such dosage as to restore the CO_2 combining power of the

plasma to normal, the patient may be relieved of the more acute symptoms of uræmia, and may be maintained symptom-free for several weeks or months. Or, if the uræmic state is the complication of a recoverable disease, as in the uræmia of acute nephritis, or the uræmia complicating or following operations on the urinary tract, the restoration of the acid-base equilibrium in the blood may save the patient's life. The fact is that when diseased kidneys fail in their function of maintaining acid-base equilibrium, and even when the disease is not so severe as itself to have determined the onset of uræmia, the administration of alkali or acid in excess readily causes uræmia. Further, both acidæmia and alkalosis in themselves cause renal damage, and thus aggravate pre-existing renal disease. In this way a vicious circle is set up.

In order to realise fully the way in which a grave disturbance of the acid-base equilibrium of the blood caused by giving too much alkali to a patient with presumably previously healthy kidneys, the reader is referred to alkalosis. In a severe case of alkalosis the clinical syndrome may be indistinguishable from renal uræmia, including albuminuria, low specific gravity urine, cylindruria, high blood urea, polyuria, thirst, lethargy, loss of appetite, nausea, vomiting, constipation, diarrhœa, coma, convulsions and death.

There is another approach to the interpretation of some of the symptoms of uræmia. Symptoms similar to those occurring in the cerebral type of uræmia may be produced by a disturbance of the cerebral circulation due to œdema of the brain, or to spasm of one or more of the cerebral arteries without a rise in the blood urea. In either case a raised blood pressure is the determining factor. In the acute cases, headache, vomiting and bradycardia are the important clinical features, and because of the rise of blood pressure which characterises the attack, it is described as a hypertensive cerebral attack. Even in cases with a high blood urea the cerebral symptoms may be due to disturbance of the cerebral circulation, and treatment by lumbar puncture or venesection, according to the indications provided by the particular case, may abort or cut short the attack. (*Vide* Diagnosis.)

It may be concluded that some of the symptoms of renal uræmia are due to a toxæmia acting on the nervous system caused by the abnormal metabolic products resulting from inadequate excretion by a diseased kidney. An important part of the clinical syndrome is due to a failure of the kidneys to maintain the normal hydrogen-ion concentration of the blood. The acidæmia that results is in part due to a failure of the kidneys to excrete acid phosphates (Marriott and Howland). Other symptoms, such as increased nerve excitability and localised muscular twitchings, have been attributed to a fall in the blood calcium by de Wesselow, and Izod Bennett compares such twitchings with those of tetany. Lastly, the disturbance of cerebral circulation, whether due to cerebral œdema or vascular spasm, may play a part. On these several lines we are approaching an adequate explanation of the uræmic syndrome. Lastly, account must be taken of the possibility of the secretion of a pressor substance by ischæmic kidneys being a factor in the causation of the uræmic state.

Symptoms.—A convenient clinical classification of the types of uræmia is : (1) *Cerebral* in the fulminating and acute cases ; (2) *Respiratory* where acidæmia is predominant ; and (3) *Gastro-intestinal* in the chronic cases. The terms acute and chronic apply to the uræmia and not to the disease responsible for it. But each of these types is really nervous in origin. Usually the first

type begins with severe headache. Drowsiness and twitchings of the face and hands follow. The twitchings may become aggravated into epileptiform convulsions, and the drowsiness may deepen into coma, ending in death. But several important departures from this course may occur. Sudden loss of vision, amaurosis, is not infrequent, although the fundi may not show the changes characteristic of albuminuric retinitis. Local palsies, hemiplegia or monoplegia, may come on spontaneously or after a convulsion, and are frequently due to small vascular lesions. Intense itching of the skin, tingling and numbness of the extremities, muscular cramps or insomnia may usher in the more serious symptoms. Sudden mania or delusional insanity may be the first and a very misleading symptom. The cerebral type is often rapidly fatal, but convulsions and amaurosis, though more striking, are less grave than the other symptoms. In the epidemic of war nephritis we saw seven instances of uræmic convulsions with complete recovery from the nephritis.

The commonest respiratory symptom is dyspnoea, often paroxysmal, to which the name of uræmic asthma is given. It is associated with a fall in the CO_2 of the alveolar air from the normal 5 per cent. to 3 per cent. or lower. There is diminished alkalinity of the blood, from the presence of some non-volatile acid. Addison called attention to the hissing character of the respirations in this condition. In all types of uræmia there is a tendency to stomatitis, and this is perhaps particularly so in uræmic asthma. This combination of dyspnoea of a hissing character in a drowsy patient with bleeding gums often characterises the terminal phase of uræmia. At first there may be no signs in the chest except the ordinary cardiovascular signs of chronic nephritis, but as the attack proceeds there are usually abundant moist sounds from the onset of cedema of the lungs. The heart fails, the patient becomes steadily waterlogged, slipping down into the bed from the orthopnoæic position as he becomes more and more drowsy. The fatal issue may not occur in this way, however, but from development of some of the more acute nervous symptoms.

Less common than this type of dyspnoea is Cheyne-Stokes' respiration. The whole of the cerebral functions may then show a curious periodicity; thus the pulse quickens during the noisy breathing, the pupil dilates, the patient becomes more conscious and restless. As the apnoæic pause succeeds, the pulse slows down again, the pupil contracts and the patient becomes quieter or even comatose.

The gastro-intestinal symptoms are nausea, hiccough, vomiting and diarrhoea. The gastric part of these symptoms may be very chronic. Any practitioner who neglects systematic examination of the urine will sooner or later treat a case of uræmia as one of simple dyspepsia. Apart from the urine, there is, however, one significant point: the dyspepsia may improve under treatment while the vomiting persists. In simple dyspepsia vomiting is never the last symptom to clear up. It is stated that this vomiting has no relation to meals, but this is far from being invariably true. Vomiting may occur only then, and so the mistake is made. In severer cases the vomiting may be quite uncontrollable, when the prognosis becomes correspondingly grave.

Attacks of diarrhoea are not uncommon in chronic nephritis and are not in themselves significant of uræmia. The amount of nitrogenous excretion

occurring by the bowel, when urinary elimination is inadequate, irritates the intestine and leads to the so-called albuminuric ulceration. Another explanation of this condition is that hæmorrhages which occur here as elsewhere in chronic nephritis are the precursors of the ulceration. There may also be an intense catarrhal or even "diphtheritic" colitis. Here, therefore, there are local lesions sufficient to account for symptoms usually referred to uræmia, for such lesions are conspicuously absent at least in the asthmatic and gastric syndromes. It is accordingly inadvisable to call these symptoms uræmic, as is generally done. At any rate the term should be confined to those violent choleraic attacks which are out of all proportion to the local lesions. Both the vomiting and diarrhœa are sometimes regarded as an attempt at vicarious elimination of toxins. The fact that the vomit may contain a higher percentage of amines than the blood certainly suggests this, but it must not be forgotten that either vomiting or diarrhœa may so alter the pH of the blood as to aggravate existing renal damage. It is this alteration which may actually determine the onset of uræmia.

Diagnosis.—This brief account of the symptoms of uræmia will indicate also some of the pitfalls besetting diagnosis. When the patient is known to have had Bright's disease, or indeed any disease or injury to the urinary system, the possibility of uræmia will arise. The finding of hæmaturia, albuminuria, cylindruria, low specific gravity urine, bacilluria or pyuria, will indicate urinary disease. The estimation of the blood urea and CO_2 combining power of the plasma will often be necessary to establish the diagnosis.

The first differential diagnosis to make is between extra-renal and renal uræmia. The causes of extra-renal uræmia must be inquired into, particularly as to whether the patient is taking alkali. Anæmia is a predisposing factor. The importance of vomiting and diarrhœa in causing extra-renal uræmia must be taken into account. In acute extra-renal uræmia, such as may be due to vomiting, the concentration of urinary urea is high. In chronic cases, however, secondary kidney damage may lead to polyuria, low specific gravity urine, and low urinary urea even in extra-renal uræmia.

Certain conditions which clinically resemble uræmia are separated from it under the term *pseudo-uræmia*, because they have a different pathology and belong to a different order of clinical events. The commonest cause of pseudo-uræmia is cardiovascular disease. Under this heading are to be included the convulsive seizures and varied evidence of cerebral disturbance in arterio-sclerotic subjects in whom the symptoms are due to vascular lesions, sometimes limited to capillary areas, in the brain. Heart failure, when responsible for cerebral disturbance, nocturnal dyspnœa, Cheynes-Stokes' breathing, and on occasion psychosis, belongs to the same order of events, and has to be distinguished from true uræmia. It may be a matter of considerable difficulty to distinguish between cardiovascular disease in which the right side of the heart is failing, and in which there is renal congestion with albuminuria, microscopic hæmaturia and cylindruria—a difficulty which may be increased by the fact that the blood urea may rise to 80 or 100 mgm. per cent. in congestive heart failure. Further, in true or renal uræmia cardiac failure may be an important complicating factor. In simple congestive heart failure the urine is loaded with urates, its specific gravity is raised, twitches do not occur, and the blood urea is normal or inconsiderably raised. Disorder of the cerebral circulation, whether due to cerebral thrombosis, capillary

hæmorrhages, angio-spasm, or cerebral œdema, may be responsible for transient monoplegia or hemiplegia, convulsions and coma. In some cases these and other symptoms of central nervous origin are associated with a sudden rise in blood pressure and constitute hypertensive cerebral attacks. In other cases the intracranial disturbance is due to œdema of the brain, as was first suggested by Traube in 1860. In this type of attack the patient is usually under the age of 40. There is complaint of sudden severe headache. Drowsiness is common, and motor weakness with focal signs or loss of vision may be transient phenomena. Retinal hæmorrhages, exudates and papilloedema appear within a few hours. The blood pressure rises and the cerebro-spinal fluid pressure is also raised (McAlpine). The blood urica is normal unless the attack occurs as a complication of uræmia. Œdema of the brain is found post mortem.

Other diseases which may simulate uræmia are cerebral tumour and meningitis. Some cases of cerebral tumour without localising signs, but with the classical symptoms of headache, vomiting and optic neuritis, may be very difficult to distinguish from uræmia, if there is chronic nephritis as well—a not very uncommon complication in syphilitic tumours of the brain. But such cases are more chronic in their course than uræmia. If the cerebral type of uræmia be accompanied by pyrexia, as it sometimes is, the question of meningitis must be considered. Lumbar puncture may then throw light on the case by the cytology, bacteriology and urica content of the fluid. Lastly, when the uramic state has reached the stage of coma the differential diagnosis from alcoholic poisoning, status epilepticus, trauma, opium, diabetic coma, the apoplectiform onset of general paralysis and cerebral hæmorrhage must be made. In all such cases a careful examination of the urine is essential, because comparatively slight renal inadequacy may lead to toxic symptoms by the retention of some poison which would otherwise have been promptly eliminated. Thus salicylates, iodides, opium and mercury are badly excreted by the nephritic.

Treatment.—As in the case of extra-renal uræmia the maintenance of the normal acid-base balance of the blood is of first importance. In most cases of renal uræmia there is acidæmia, and this is treated by the cautious administration of alkali by mouth, or by the intravenous injection of a 2 per cent. solution of sodium bicarbonate 200 to 400 c.c. Anæmia renders the acid-base balance unstable. It is treated by the administration of iron, and 60 to 90 grains of iron and ammonium citrate are given in divided doses daily, provided the salt does not upset the digestion. Vomiting and diarrhœa should be checked, because they weaken the patient, prevent assimilation of food and cause dehydration. Vomiting causes alkalosis, and diarrhœa may cause acidæmia. A bismuth mixture or 3 minims of dilute hydrocyanic acid and 10 minims of solution of adrenaline hydrochloride 1 in 1000 in half an ounce of water given every 3 or 4 hours may afford relief.

The retention of urica is treated by a low nitrogen diet. The diet advised for acute nephritis may be given with advantage for a week or 10 days, and after this for 1 or 2 days in each week. If a low nitrogen diet is continued it must at least be adequate in its protein content to retain nitrogenous equilibrium, for which purpose 1 G. per kilo body-weight is required. Elimination is promoted by maintaining bowel function, avoiding constipation on the one hand, and diarrhœa on the other. According to von Noorden, 8 G. of

nitrogen can be excreted by the bowel in the day and 3 G. by the skin. Strong aperients and mercurial preparations should not be employed. A good evacuation may be secured by magnesium sulphate 30 to 60 grains repeated as necessary, 60 to 90 grains of pulv. jalapæ co., 1 oz. of mist. sennæ co., or 60 to 90 grains of compound liquorice powder. The function of the skin is promoted by suitable clothing, wearing wool or flannel next to the skin, and sleeping between blankets. Much sweating may have disadvantages that it causes dehydration, and gives the kidneys a more concentrated and therefore a more irritating urine to secrete. The vapour bath or hot pack is more trying to the heart than the hot-air bath. None of these measures should be continued more than a quarter of an hour after sweating has begun, and a careful watch must be kept on the pulse; the procedure should be stopped at once if there are any signs of collapse, and stimulants should be at hand. If the treatment is having a good effect, sweating will begin at a lower temperature with successive baths. A nightly hot bath, containing 4 tablespoonfuls of mustard, followed by wrapping in hot blankets until sweating has ceased, is useful in some chronic cases, even where there is no œdema. Pilocarpine is no longer advised to provoke sweating.

Headache and other symptoms of cerebral irritation may be relieved by lumbar puncture when accompanied by an increase in cerebro-spinal fluid pressure. It is safe to draw off 10 or 20 c.c. if the pressure is raised, but if with the drawing off of the fluid there is increase of headache the needle should be withdrawn immediately. If the headache is due to a hypertensive attack, particularly if there is evidence of congestive heart failure, venesection may give relief. The withdrawal by venesection of 10 to 20 oz. of blood may cure the headache, and it may stop convulsions in acute nephritis with an overburdened heart. Venesection is contra-indicated in chronic uræmia with anæmia. Bromide and chloral hydrate will often relieve headache. Twenty to 30 grains of sodium bromide with 15 to 20 grains of chloral hydrate are given by mouth. Aspirin, phenacetin and codeine are other useful drugs best prescribed in combination in severe cases. Morphine, papaveretum (omnupon) and dilaudid are reserved for intractable cases, and are to be used with caution.

LARDACEOUS DISEASE

Synonyms.—Amyloid or Waxy Kidney.

Definition.—A pathological condition in which the blood vessels of the kidney, in more advanced cases the tunica of the tubules and the interstitial tissue also, are the seat of waxy degeneration.

Ætiology.—This affection is now rarely met with. It attacks men more than women, and although occasionally seen in children it is more likely to occur in adolescence and earlier adult life, being uncommon after fifty years of age. It is usually due to chronic suppuration, especially in bone, chronic tuberculosis and syphilis. It rarely occurs in other chronic infections, but it has been described in severe rheumatic heart disease, and a certain amount of amyloid change has sometimes been found post mortem in patients suffering from chronic cardiovascular disease and chronic nephritis in the absence of chronic suppuration. As it is a degenerative change it has however more affinity with nephrosis than with nephritis.

Pathology.—Amyloid material or lardaccin is a product of protein degeneration, and consists of protein linked with chondroitin-sulphuric acid. The latter substance is a normal constituent of elastic tissue and cartilage. In uncomplicated cases, the affected kidney has the appearance of a large white kidney with a smooth surface and a capsule that strips easily. The organ is firmer than it otherwise would be. On section, the cortex is thicker than normal and has a yellowish white appearance; the glomeruli may be visible as minute translucent spots. The pyramids are dark red, in contrast to the pale cortex. If a solution of iodine in potassium iodide is poured over the surface, some of the glomeruli stand out as mahogany-brown spots and the vasa recta as brown streaks. In histological preparations stained with methyl-violet, amyloid material takes a pink colour. The disease tends to appear first in the capillaries of some glomeruli, while others are normal, and its incidence is often partial within a single glomerulus. The afferent arterioles, vasa recta and capillary plexus are next affected; in more advanced cases there is amyloid degeneration of the tunica propria of the tubules with amyloid deposits in the interstitial tissue. In most cases there is an associated nephritis, interstitial rather than parenchymatous. The kidney lesion is generally the most striking part of a widespread lardaceous degeneration which also involves the liver, spleen and intestine; less commonly the blood vessels of the thyroid, suprarenals, pancreas, heart and brain may be affected as well. Occasionally only the kidney is implicated.

Symptoms.—The onset is insidious and the symptoms are not likely to occur unless chronic suppuration has existed for at least 3 months.

The urine is copious, of low specific gravity (1003 to 1010). The amount of albumin is variable; when abundant there is probably coincident nephritis. The amount of urine and its specific gravity may also be affected by the presence and degree of coincident nephritis, and the state of the heart. Hyaline and granular casts are present in the urine; casts staining brown with iodine are not evidence of amyloid disease, and may occur in other diseases of the kidneys. True waxy casts are not found. In later stages there is oedema, with diminished excretion of urine. The blood pressure is not raised, nor is the left ventricle hypertrophied, unless there is coexistent chronic nephritis.

Diagnosis.—The diagnosis is indicated by the nature of the urine. It is made (1) when there is a sufficient cause in the past history or present condition, namely, chronic suppuration or syphilis; (2) on the general condition of the patient, namely, a secondary anaemia, which may reach an extreme grade, with a pale or "alabaster" facies and cachexia; (3) on signs of lardaceous disease in other organs, such as enlargement of the liver or spleen and diarrhoea.

Course and Prognosis.—This depends on that of the primary cause. If the latter is unchecked, the disease is slowly progressive and death occurs from exhaustion due to the original disease, less often from uræmia. Where the original disease can be cured, recovery may occur. Complete recovery of the kidneys is less likely than is recovery of the liver, spleen and intestines.

Treatment.—The treatment is that of the original disease. In suppuration of the bones or joints, empyema, etc., it is surgical; but it must be recognised that in advanced cases surgical treatment may be too late, even though it is successful in eradicating the septic focus. In all cases fresh air

and sunlight and a nourishing diet are essential. Iron, arsenic and cod-liver oil should be given. Cases of syphilitic origin should be treated with bismuth and arsenic, while mercury and iodides should rarely be given, and then only with caution since even therapeutic doses have produced serious reactions.

PYELITIS

Definition.—Pyelitis is inflammation of the renal pelvis. The changes in the renal parenchyma are those described under Toxæmic Kidney. Pyelitis may be complicated by nephritis, and the condition is then termed pyelo-nephritis.

Ætiology.—Most cases are due to a blood-borne infection of the renal pelvis, and it may be noted in this connection that it is a normal function of the kidney to excrete micro-organisms present in the blood stream; whether the renal parenchyma is, or is not, of necessity damaged in the process is a point on which there is not as yet exact information. The pelvis may also be involved by ascending infections—(a) via the lumen of the ureter when there is ureteral obstruction; it is probable that infection does not spread by this channel when the lumen is normally patent. (b) By way of the peri-ureteral lymphatics from local foci in lower parts of the urinary tract, such as the bladder, urethra, prostate, seminal vesicles and epididymis. Lastly, there is the possibility of direct spread of infection from the bowel, and by cross lymphatic channels from one kidney to the other. In those cases in which a pyelitis occurs secondary to appendicitis, cholecystitis, ulcerative colitis, etc., the spread of infection may be by the lymphatics or the blood stream.

Pyelitis is more common in females than in males. Its age incidence depends on the determining cause. Thus, it is common in female infants, as a result perhaps of urethral infection, to which they are more liable than male infants. It is not an uncommon complication of pregnancy, occurring especially in the fifth month of gestation. It is common in males at a later age, associated with enlarged prostate and cystitis.

In general terms any injury or disease of the renal pelvis, or any condition which interferes with the normal flow of urine, may be the determining cause of pyelitis. Thus it is a common complication of hydronephrosis from whatever cause. It often complicates stone in a kidney, tuberculosis of the kidney and new-growths of the renal pelvis. The frequency of pyelitis as a complication of intestinal catarrh (whether due to infection or the habitual use of laxatives) and ulceration is probably due to the increased virulence and excessive numbers of bacteria that reach the kidney in such conditions.

Pathology.—The mucous membrane of the pelvis is swollen, cedematous and hyperæmic, and the submucous venules are engorged. Where there is obstruction, the pelvis is dilated and contains a slightly turbid or opalescent fluid. In these circumstances the ureter above the obstruction is dilated and tortuous and its walls are thickened. The kidney is swollen and pale, from cloudy swelling, and in severe cases there may be multiple small abscesses in the renal parenchyma.

Bacillus coli is by far the most common infecting micro-organism. Streptococci, staphylococci, gonococci and bacilli of the proteus and typhoid

groups may be found. The infecting micro-organism is readily recovered from the urine.

Symptoms.—The clinical types of pyelitis differ greatly from one another, and the condition may be responsible for an acute fulminating illness or for chronic malaise of indefinite nature.

LOCAL SYMPTOMS.—Pain is the most important, especially as a diagnostic indication in acute cases. It is a dull ache in the loin or flank, at first slight and intermittent, later, or in other cases at once, constant and sometimes intense. Occasionally it takes the form of renal colic. At its onset the pain may be diffuse and abdominal. Increased frequency of micturition is a common symptom. There may be strangury.

GENERAL SYMPTOMS.—In acute cases there may be sudden onset with rigors, vomiting, headache and the general constitutional disturbance of profound toxæmia. These cases may simulate septicæmia (in fact there may be septicæmia), appendicitis, or, when associated with abdominal distension, constipation and vomiting may even simulate intestinal obstruction. In other cases, with cerebral symptoms, meningitis may at first be difficult to exclude.

In subacute cases, without marked pain or rigors, there is general malaise, fever, anorexia, wasting and a secondary anæmia associated with some degree of polymorphonuclear leucocytosis (W.B.C. = 10 to 15,000).

In relapsing cases there are periods of exacerbation with acute symptoms, and intervening periods of fair health or general malaise. Fever is commonly present; in acute cases with rigors it may rise to 105° or 106° F. In general the temperature is irregular, remittent or intermittent, varying between 102° and 104° F. in acute cases, and 100° and 102° F. in subacute cases. The pulse is raised in proportion to the temperature, and there is a corresponding slight increase in the respiration rate. Of other general symptoms constipation or diarrhœa frequently precedes the disease, and constipation generally accompanies it. Toxæmia is often marked.

Deep tenderness on palpation of the renal region is the most important sign to determine. There is some degree of abdominal rigidity, and it may be possible to determine enlargement and tenderness of the kidney. The urine is passed in small quantities at frequent intervals. It has the usual characters of febrile urine and is turbid. The turbidity or an opalescence is still present after filtration. When an appreciable quantity of pus is present it settles at the bottom of a specimen glass in a thick whitish deposit. Examination of the deposit (catheter specimen in women) shows pus cells and epithelial cells from the urinary tract. There may be hæmaturia.

Bacteriuria.—In this condition bacteria are present in the urine in such quantity as to make it hazy to the naked eye, but there is little or no inflammatory reaction in any part of the urinary tract. Hence there are no localising symptoms and few pus cells. The urine when freshly passed has a hazy appearance. In a test-tube, when the tube is rotated, the urine has a "satiny" appearance or shimmer. It is not cleared by filtration. It often has a fishy smell in *B. coli* infection, and is ammoniacal in smell in *B. proteus* infection. Its reaction is acid, unless due to staphylococcal or *B. proteus* infection. It generally contains a trace of albumin, and often may contain a few white blood corpuscles and epithelial cells. A catheter specimen grown in broth, in dilutions of 1 c.c., $\frac{1}{10}$ c.c. and $\frac{1}{100}$ c.c. urine in

10 c.c. broth, gives a growth in all dilutions, and in *B. coli* infections there is generally a growth in greater dilutions. Streptococcal and staphylococcal infections are less common.

There may be no other symptoms. On the other hand, there may be indefinite malaise, fever, gastro-intestinal disturbance, especially indigestion, constipation and abdominal pain; in other cases headaches, rigors and even meningism may occur. There may be local symptoms, such as enuresis in children and frequency of micturition in adults. When the symptoms point to inflammatory reaction in one part of the genito-urinary tract, such as pyelitis, cystitis, prostatitis, urethritis, or epididymitis, the condition is better diagnosed accordingly.

The recognition of bacilluria may be of great importance, not only because of the ill-health, acute disease or complications for which it may be responsible, but also because it may be a valuable pointer to other disease. For instance, a patient may complain of loss of energy and indefinite malaise. On clinical examination the only clinical finding may be some degree of secondary anæmia or bacilluria. A further examination of the urine bacteriologically, or X-ray examination of the urinary tract, may reveal previously unsuspected tuberculous disease, stone or neoplasm, even in the absence of urinary symptoms.

Diagnosis.—When there is fever and constitutional disturbance without localising signs or symptoms, the differential diagnosis is from those diseases which come in their early phases under the category of indeterminate fever. The diagnosis is established by examination of the urine. Pyonephrosis is diagnosed by the presence of a tumour. Calculus is recognised by its clinical features and by X-ray photograph. Perinephric abscess in its early stages is not accompanied by pyuria or frequency of micturition. Cystitis is generally afebrile; and it is accompanied by suprapubic discomfort and pain, particularly at the end of micturition; the diagnosis can be established by cystoscopy. Urethritis is recognised by local tenderness, urethral discharge and urethroscopy, and prostatitis by swelling and tenderness on rectal examination.

Prognosis.—The natural course in the majority of cases is to recovery in a few weeks. With modern remedies the urine can generally be sterilised in 7 to 14 days unless the urinary infection is a complication of some other condition or disease. The prognosis depends very largely on this sterilisation of the urine, because the persistence of even a minimal infection is likely to lead to relapse, or apparent recovery may be followed by a recurrence after a variable length of time. The disease may progress to pyelo-nephritis, ascending suppurative nephritis, pyonephrosis or perinephric abscess. A fatal termination is rare, except when the condition complicates other disease, such as paraplegia, or in elderly persons with obstruction to the outflow of urine.

Treatment.—Prophylaxis is important in nurseries and children's hospitals, since there is evidence of spread of infection via the urethra, at any rate in females. Here it is a question of cleanliness. In general terms exposure to cold, over-fatigue, and loose stools are to be avoided when there is susceptibility to coli infection of the urinary tract.

The treatment of an acute attack consists of absolute rest in bed, flushing out the kidneys with large quantities of fluid, and regulation of bowel

function. It is important, especially when there is fever, to avoid exposure to cold and any possibility of chill. Particularly when there is fever the patient should wear wool next to the skin, lie between blankets and be nursed in bed. Five to 8 pints of fluid are given in every 24 hours in the form of water, barley water, imperial drink, lemon drink, weak tea and thin soups. Milk as such is unsuitable, but junket, buttermilk, whey and cream are good. As the temperature subsides the diet is increased by the addition of carbohydrates, fruit, vegetables and fat. Cooked milk in the form of milk puddings is allowed. Alcohol is withheld. The bowels are emptied with an initial laxative, followed by an enema if necessary. After this the action of the bowels is regulated with paraffin, salts or mild laxatives, such as liquorice powder, senna pods or rhubarb, so that constipation is avoided on the one hand and loose stools on the other.

In the initial febrile stage when there is bacterial toxæmia, sufficient alkali is given by mouth to make the urine alkaline. A mixture containing 30 grains each of potassium citrate and sodium bicarbonate is given 3-hourly until the urine is alkaline. Every specimen of urine passed is tested with litmus paper. When the urine is alkaline the quantity of alkali by mouth is reduced by giving it 4- or 6-hourly, but always in sufficient quantity to keep every specimen of urine alkaline, until the temperature is normal. One of the sulphanilamide group of drugs is then given 4-hourly. A full dose is sulphanilamide 1 gramme 4-hourly (6 grammes in 24 hours). This may be reduced to 4 grammes in 24 hours after 3 or 4 days, and later to 3 grammes (*i.e.* 0.5 gramme 4-hourly). Alternatively sulphapyridine (M. & B. 693) may be prescribed, or rubiazol (Roussel) 12 to 8 tablets in 24 hours (one tablet contains 0.2 gramme of the azo-compound). In some cases, especially if these drugs are not well tolerated, a mandelic acid salt may be given. The usual dose is 45 grains of ammonium or calcium mandelate thrice daily.

Flushing out the kidneys with large draughts of water must always dilute the antiseptic drug. Flushing and antisepsis should therefore be attempted consecutively and not simultaneously.

In uncomplicated cases a sterile urine may be expected in 7 to 10 days. In some cases the drug may have to be administered for a longer period, or the course of treatment may have to be repeated after an interval of a fortnight or 3 weeks. Occasionally in resistant cases, or in those which continually relapse as soon as the drug is withheld, good results have been obtained by giving ammonium mandelate for several weeks, provided that the patient is under good observation, so that the occurrence of kidney damage and acidæmia can be prevented. In acute cases with fever it may be better to initiate treatment with a sulphanilamide drug without the initial course of alkali therapy, provided that a close observation of the case can be maintained, including estimation of the blood urea as required. During the acute phase of the disease the kidneys are more susceptible to the toxic effects of these drugs.

It is important to remember that, like simple bacilluria, infections of the urinary tract are often a complication of organic disease of either the urinary tract or bowel. A urinary infection, which at first sight appears to be a simple coli infection of the urinary tract, may be only a complication of tuberculosis of the kidney calculus, hydronephrosis or neoplasm. Equally it may be a complication of organic disease of the digestive tract, such as chronic appendicitis,

especially if the right ureter is involved, diverticulosis, or even cholecystitis. In any case of urinary infection that is resistant to treatment or presents any unusual symptom, a detailed investigation of both urinary and digestive tracts is required in order to determine or exclude a change in structure which may be the underlying and determining cause of the urinary infection.

In the uncommon fulminating cases with unilateral suppurative nephritis, nephrectomy may save the patient's life.

Chronic cases.—An initial course of treatment such as that outlined for acute infection with rest in bed is advisable. In some cases, perhaps on account of long-standing infection, or some other disease of the urinary tract, such as calculus diverticulum of the bladder, enlargement of the prostate, etc., it may be impossible to sterilise the urinary tract. Every effort should then be made to build up the patient's resistance by living in fresh air, avoiding chill and over-fatigue, a generous and nourishing diet, and by so arranging the diet that the bowels are open regularly without taking purgatives other than a simple saline in the morning, paraffin and agar, or other laxative which determines the evacuation of a formed stool. It is important so far as is possible to restore integrity of structure to the body as a whole by the cure of anæmia, for instance, and by the eradication of obvious sepsis elsewhere in the body. In incurable cases the infection may be controlled to a greater or less extent by long-continued use of hexamine, which may often be given with advantage with methylene blue.

PERINEPHRITIS AND PERINEPHRIC ABSCESS

1. Perinephritis without suppuration is really a part of some cases of chronic nephritis. Its clinical importance is not generally recognised, but it may be a cause of lumbar pain in that disease. The capsule of the kidney is thickened and adherent to the perirenal tissues, many of the adhesions being vascular.

2. Perinephritis proceeding to suppuration may be primary or secondary.

Ætiology and Pathology.—The primary form may follow injury, but more frequently it results from boils, carbuncles and tonsillitis, or complicates an acute specific fever. Soon after the War of 1914-1918, cases were so common as to be described under the name of *epidemic perinephric suppuration*. The infecting organism is *Staphylococcus pyogenes*. J. Koch has shown experimentally that intravenous injection of staphylococci is followed by their excretion in the urine after an interval of 4 to 6 hours. In the process of excretion, according to Koch, they may give rise to multiple cortical abscesses, cylindrical medullary abscesses, or, passing along the cortical lymphatics, may gain access to the perinephric tissues and there cause abscess formation. In these circumstances perinephric abscess is an example of the mildest form of staphylococcal pyæmia with single metastatic abscess formation. The secondary form may complicate suppuration in the neighbouring organs, such as the kidney, liver, gall-bladder or appendix. It may be secondary to caries of the spine. In other cases the infection may be carried by lymphatics from a focus in or around the bladder, rectum or female pelvic organs.

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Such is the ordinary terminology, but it will be observed that the "primary" form is really due to infection from a distant focus through the blood stream, while the "secondary" is due to direct extension or infection through the lymphatics from some focus in the neighbourhood of the kidney.

Symptoms.—The onset is generally gradual. It is characterised by fever and malaise as in typhoid fever. There may be no local symptoms for the first 7 to 14 days, and during this period there is increasing toxæmia, general abdominal discomfort or pain, slight fullness and resistance, with deep tenderness, in the affected loin. As the abscess forms, pain and tenderness increase, there is induration and, later, redness of the skin and œdema in the lumbar region. The tumour first tends to spread backwards, obliterating the normal hollow in the loin, and then as pus collects it may spread forwards, forming a tender tumour palpable from the front. In its relations to the colon it resembles a renal tumour, but does not move with respiration. There is resistance or rigidity of the abdominal wall on the affected side. There is an increasing polymorphonuclear leucocytosis up to 20,000 or even 40,000. The urine is febrile in character, containing a trace of albumin and perhaps a few white blood corpuscles; it does not contain pus, unless the kidney itself is involved, but hæmaturia may occur. In some cases the disease runs an acute course, and there may be rigors at an early stage.

Course.—When the condition is simply associated with chronic nephritis it has no separate significance. When it proceeds to suppuration the abscess may rupture into the peritoneum, colon or pleura, or on to the surface, unless the abscess is opened and drained.

Diagnosis.—Before localising signs appear the condition may be mistaken for typhoid fever, malaria or septic endocarditis. The blood examination is important for the purpose of excluding malarial parasites; leucocytosis is against typhoid fever, and when above 15,000 is in general against infective endocarditis. Absence of agglutination of micro-organisms of the typhoid group is further evidence.

When the tumour exists it has to be distinguished from a renal tumour or pyonephrosis. Renal and adrenal growths may be accompanied by fever, but do not usually give the general symptoms of suppuration; they tend to extend forwards rather than backwards, and induration of the tissues is absent. Pyonephrosis causes symptoms of suppuration and a tender swelling, but the tumour is circumscribed, moves with respiration, and does not cause any bulging in the lumbar region. Pyuria is usually present.

The diagnosis of caries of the spine, hip disease, and even of myositis as distinct from perinephritis may be difficult. Since perinephritis in itself induces lumbar rigidity and some degree of scoliosis, X-ray examination may be required to exclude caries of the spine. Hip-joint disease is excluded by absence of local tenderness and by the freedom of flexion and rotation of the thigh.

Treatment.—In the early stages, before there is evidence of suppuration, and when the chief symptom is lumbar pain, the treatment is that of a patient acutely ill with a general toxæmia. The bowels should be kept well open, and fomentations or poultices applied to the lumbar region. Aspirin

may be given to relieve pain. An operation should be performed and the abscess evacuated as soon as the diagnosis is definitely established.

TUBERCULOSIS OF THE KIDNEY

Small grey tubercles are frequently found scattered through the kidneys in persons who die of acute miliary tuberculosis; the kidney disease, however, scarcely affects the clinical aspect of the case, and this form of renal tuberculosis will not be considered here. Further, in patients who die of pulmonary tuberculosis it is not uncommon to find tuberculous foci in the kidneys post mortem, although there was no indication of their presence during life.

Clinical renal tuberculosis is either the fibro-caseating form of the disease, or it is tuberculous hydronephrosis. In either case, the tuberculous infection is generally primary in the kidney in so far as its clinical expression is concerned.

Ætiology.—It is more common in women than men. The maximum age incidence is in the third and fourth decades; the disease is uncommon in the young and rare in the old. At an early stage the disease is unilateral. In the majority of cases the tubercle bacilli are carried to the kidney by the blood stream from a tuberculous focus, such as a caseating lymph gland. Recent experimental work has shown that bacteria do not ascend in the lumen of the ureter unless it is diseased, when the infection may spread by direct extension in its walls. Infection may also reach the kidney via the lymphatics in a proportion of cases. The path of infection is by way of the ureteric lymphatics, and it is probable that in pelvic tuberculosis, for example, tuberculous prostatitis, may spread to the kidney by this route. There is also reason to think that tubercle bacilli from a diseased kidney may infect the opposite healthy kidney by the same lymphatic path, the bacilli first travelling in the urine and walls of the ureter from the diseased kidney and causing disease of the bladder, and then travelling from the bladder by way of the ureteric lymphatics to the sound kidney. On the other hand, there is a shorter path for infection from one kidney to another by the para-aortic lymphatic system. Since the disease in the other kidney takes the same anatomical form as it originally had in the kidney first affected, it is probable that, if the first is due to a blood-borne infection, so is the second. Vesical tuberculosis is, as a rule, secondary to infection elsewhere in the urogenital system commonly in the kidneys.

Pathology.—The initial lesion is in the cortex, or one of the pyramids, and it consists of one or more tubercles. The morbid process spreads by destruction of kidney tissue; there is caseation in the centre of the lesion, inflammatory reaction, with intense small-cell infiltration, giant-cell formation and more or less fibrosis at the periphery. The lesion also spreads by the deposition of tubercles at a distance; these are scattered through the cortex, singly or in groups. Extension through the capsule is uncommon, but extension to the renal pelvis is frequent. Complete destruction of one or more pyramids may occur, or the disease may spread and involve one or more calices or the entire pelvis. The resulting infiltration and cicatricial contraction may lead to hydro- or pyo-nephrosis. The disease tends to extend

down the ureter, and the bladder is commonly infected at an early stage. Secondary infections may lead to metastatic abscesses in the kidneys and ultimately to destruction of the whole organ.

Symptoms.—Frequency of micturition is often the earliest symptom; it is first noticed by day and later at night. Urgency and painful micturition develop next. The urine may show no other abnormality than a trace of albumin at an early stage; characteristically it is pale and a little turbid from the presence of pus; it is acid in reaction, it may contain a few renal cells, and it is sterile on culture. By appropriate staining tubercle bacilli may be demonstrated in the centrifuged deposit. Hæmaturia may be the first symptom, or the disease may develop insidiously with lumbar pain. On examination, the kidney is sometimes enlarged, and it may be hard and irregular; it is often tender. Tenderness along the course of the ureter or thickening of the ureter, as determined by abdominal or rectal examination, is of great importance. The rest of the urino-genital system requires close examination; this should include cystoscopy and in some cases ureteral catheterisation. X-ray examination of the abdomen may reveal calcified tuberculosis of the kidneys or lymph glands, and it may be required in the differential diagnosis from renal calculus. Finally, a careful review of the patient's history and present condition for evidence of a chronic bacterial toxæmia or of tuberculous infection elsewhere must be made.

Diagnosis.—The presence of tubercle bacilli in the urine, whether determined by microscopic examination of the stained deposit or by guinea-pig inoculation, is not absolute proof of renal tuberculosis, because the bacilli may be excreted by a healthy kidney or they may come from some other part of the urinary tract. Nevertheless, the demonstration of tubercle bacilli in the urine is of the first importance in a doubtful case, and the diagnosis may be established by cystoscopy. The cases which require most careful examination are those with an atypical onset, such as massive hæmaturia, and those in which there is a gross secondary infection when first seen. The possibility of renal tuberculosis must always be borne in mind in hydro- and pyo-nephrosis. The differential diagnosis from simple albuminuria and the several forms of Bright's disease is made on the presence of pyuria and the absence of signs and symptoms of chronic nephritis. Patients with pulmonary tuberculosis are perhaps more prone than others to chronic nephritis on account of the secondary infections which complicate their disease.

Course and Prognosis.—The onset is insidious and the course progressive. Natural recovery is hardly known, though occasionally an unsuspected caseous kidney may be found at autopsy in patients dying of other diseases. The disease runs an uncertain course, having a duration of a few years up to ten or even twelve years from the date of diagnosis. Death results from tuberculous toxæmia, secondary infection, or failure of renal function.

Treatment.—When the disease is unilateral the kidney should be removed but nephrectomy is rarely justified if the other kidney is involved. In any case the patient's health and resistance should be raised to the utmost by rest, fresh air and good food, on the general lines of treatment of tuberculosis of the lungs. Cautious tuberculin treatment may be indicated when the disease cannot be treated surgically.

RENAL CALCULI (NEPHROLITHIASIS)

Renal calculi may be composed of calcium oxalate or carbonate, uric acid, urates, phosphates, cystin, or of a mixture of these.

Ætiology.—All these materials are sparingly soluble in water and their solubility in urine is dependent on (i) its pH. If this stands at 5 uric acid is precipitated, while phosphates and carbonates are deposited at pH about 8; the others at some intermediate point. (ii) On the presence of urea, which renders both uric acid and oxalates more soluble. (iii) The protective action of certain non-albuminous colloids. If these become coagulated their protective influence is lost. Thus two factors are required to form a renal calculus: crystals derived from the urine and some colloidal material to bind them together. Hence, as Benjamin Moore pointed out, the commonest nucleus of a stone is calcium oxalate, since oxaluria excites albuminuria and even hæmaturia, thus providing the necessary colloid. Prolonged recumbency, as after fracture of the femur, provides opportunity for calculus formation apparently from stagnation in the dorsal portion of the calices. Infection of the urinary tract such as pyelitis is not considered so important as formerly, and indeed may be merely secondary to the calculus; but it is a factor in cystinuria, which will not lead to a calculus unless the urine becomes infected. Pure uric-acid stones may occur in quite young children, but the definite deposit of uric-acid crystals in the pyramids and pelvis of the kidney which is almost a normal event does not seem to lead to calculus formation and milk is usually sufficiently diuretic to remove them. The most important single factor in the prophylaxis of stone is adequate diet. Lack of vitamin A is especially prone to excite stone. In a recent investigation 96 per cent. of cases of renal calculi showed evidence of its deficiency. It is necessary for the maintenance of the proper nutrition of epithelial linings everywhere. The former comparative frequency of uric-acid stones in the children of the poor in London was probably related to the scarcity of fresh vegetables in the diet. Such stones were also common in Norfolk and the neighbouring fens. Chalk in the soil or in the drinking water does not predispose to stone. The factors leading to the deposits of various crystals in the urine have already been discussed (see Abnormalities of the Urinary Secretion).

Calculi may occur at any age, but are very rare in the old. They are commoner in males than in females. Those of sedentary habit are more liable to them. Alcohol and lead are said to predispose to renal calculi. A high blood calcium, whether due to excess of parathormone (as in parathyroid tumours) or of vitamin D, can be an important factor in producing calculi of calcium phosphate.

Pathology.—The pure oxalate stone is very hard, mulberry-shaped, stained by altered blood, and varies in size from that of a mere granule to that of a walnut. If it is encrusted with uric acid it becomes brown, and in form a coral-shaped mass, representing a cast of the renal pelvis and calices. Phosphatic stones are generally smooth and white. A cystin stone is hard, oval, light amber or greenish in colour, with a glistening surface. Other forms are rare. If the stone remains in the renal pelvis it may (1) by gradually increasing in size lead to the atrophy of the renal tissue; (2) by eroding the

capsule of the kidney produce a fistula into the perinephric tissues, resulting in a perinephric abscess; (3) by obstructing the outflow of urine cause hydronephrosis or, more frequently, pyonephrosis. If it passes into the ureter it may become impacted, in this way again exciting hydronephrosis or pyonephrosis, or if it obstructs the ureter completely, may produce atrophy of the kidney. If it causes ulceration of the ureter, this may be followed by stenosis. If it passes into the bladder it is very likely to excite ammoniacal decomposition, and thus become encrusted with phosphates.

Symptoms.—A stone may remain latent in the kidney without causing any symptoms. More usually it causes pain, particularly on any jolting movement. This is occasionally referred to the opposite side, a point to be borne in mind when considering operation. A bout of pain may be accompanied by hæmaturia, and there may be albuminuria for some days afterwards. A small oxalate stone may produce more pain than a large uratic stone, because of its hardness and roughness. A large, branched uratic stone occasionally causes profuse hæmaturia without any pain. The results of renal calculi may be classified as (a) mechanical, (b) septic. Under the first heading come colic, hæmaturia, anuria, hydronephrosis; under the second, pyelitis, perinephric abscess, pyonephrosis.

Renal colic is the most severe and distressing manifestation of calculus. It is particularly likely to be started by riding on a horse or in a train or omnibus, which causes the calculus to engage in the entrance to the ureter. Violent paroxysms of pain then occur, radiating along the course of the genito-crural nerve down into the groin and testis, which becomes retracted in the scrotum. The pain is also felt in the loin, and the muscles overlying the kidney become rigid. Vomiting and sweating are common. The patient is unable to keep still, and rolls about or gets on to his hands and knees, calling out with each paroxysm. He becomes pale and his pulse increases in frequency, and the temperature is apt to rise. During or after the attack there is usually some hæmaturia, and crystals may be found in the urine. The attack may last several hours and then end as abruptly as it began. Anuria is a serious symptom and implies that the ureter is completely blocked, and the other kidney is either diseased or its secretion reflexly inhibited. Occasionally both ureters may be blocked by calculi. Symptoms referred to the bladder, prostate or seminal vesicles do not occur until the stone reaches the bladder or the lower end of the ureter.

Diagnosis.—The occurrence of renal colic and hæmaturia suggests stone, but these symptoms may be produced by the passage of a blood clot from renal neoplasm or by acute pyelitis, especially in a movable kidney. Ordinary examination of the abdomen reveals nothing beyond lumbar tenderness in uncomplicated cases. X-ray examination is of great value. Oxalate stones are the easiest to detect by that method, as even when small they throw a dense shadow. This is fortunate, since oxalate stones are the commonest. Pure uratic stones may not be detected unless they are large. Cystin stones throw very little shadow. Calcareous abdominal glands and phleboliths may be mistaken for calculi on X-ray examination. In doubtful cases, pyelography, intravenous or instrumental, should be done. A skiagram of the pelvis should never be omitted, since a stone may have passed down to this region. Attacks of pain and hæmaturia with the presence of calcium oxalate crystals in the urine, but with a negative X-ray examination, are probably

due to crises of oxaluria (see p. 1296). Appendicular colic may simulate renal colic, but the point of maximum tenderness is different.

Prognosis.—As long as there is no serious destruction of kidney substance or septic complication the outlook as to life is good, if treatment be adequate. Attacks of renal colic may occur from time to time, with great suffering, and even after stones have been removed by operation they may form again, though this is exceptional. Occasionally stones may be followed by a true chronic nephritis with its usual consequences.

Treatment.—The methods which should be employed when crystals likely to form stones are found in the urine have been described under urinary deposits. Careful attention must be given to the diet, especially to its vitamin content. Disinfection of the urine should be carried out as described under bacilluria and pyelitis. It is well, however, not to render the urine alkaline when a stone is suspected, since this would lead to a deposit of phosphates upon it. A book of litmus papers should be given to the patient with instructions to place blue and red strips in the morning urine, which is likely to be the most acid. Enough citrate of potash should be given to render the urine amphoteric but not alkaline. Probably 20 grains at night will be sufficient for this purpose. The urine should be kept dilute by taking water freely. Mineral waters, such as Contrexéville and Evian, are helpful, the former particularly for uric acid, the latter for oxalates. Whey is also helpful when uric acid crystals are present. If a renal calculus is present, and this is confirmed by X-rays, removal by operation is indicated. The following points, however, are generally contra-indications for operation: (i) large bilateral stones; (ii) stones which are only the size of a pea or smaller, unless there is severe pain, extensive absorption of renal substance causing toxic symptoms, or obstruction to the outflow of urine. If a small stone is not passed as a result of medical treatment, its removal by operation should be seriously considered; (iii) in some patients small calculi are repeatedly formed and passed. In these cases operation is better postponed because of the likelihood of recurrence. If the diagnosis is uncertain, or operation is refused or postponed or considered inadvisable because of the patient's general condition, the treatment appropriate to the deposit found in the urine should be continued. Violent exercise and jolting movements should be avoided. Small stones can often be got rid of by giving the patient 5 to 10 minims of tincture of belladonna with 10 grains of potassium citrate every 4 hours for a few days, and directing that 5 pints of water should be taken in the 24 hours. For the symptomatic relief of pain, aspirin in 10-grain doses, hot baths and kaolin poultice (antiphlogistine) may be of service. Morphine should be avoided in the treatment of chronic renal pain, on account of the danger of establishing a habit.

For an attack of renal colic, $\frac{1}{4}$ th to $\frac{1}{2}$ rd of a grain of morphine tartrate, together with $\frac{1}{100}$ th of a grain of atropine sulphate, should be given hypodermically. The anti-spasmodic effect of the atropine aids the onward passage of the stone, while the morphine relieves the pain. If morphine be given alone, the pain is apt to recur as soon as its anodyne effect passes off. Ten minims of tincture of belladonna should then be given in an ounce of water every 3 or 4 hours, with abundant fluids, as described above, until the pupils are dilated and the face rather flushed. Inhalations of chloroform may be necessary at the onset, until the drugs have had time to act. Hot applications

to the loins or hot baths may help to relax spasm. Inversion of the patient has been advised, to attempt to disengage the stone from the ureter. After the paroxysm is over, the aid of X-rays should again be invoked to locate the stone if it has not been passed.

HYDRONEPHROSIS

Definition.—A condition in which the pelvis and calices of the kidney are distended by the accumulation of non-infected urine due to ureteral or urethral obstruction.

Ætiology.—**CONGENITAL.**—The condition may be congenital, due to an abnormality of the ureter or urethra; other congenital defects may be present. The ureteral stricture is commonly found at the exit of the ureter from the pelvis of the kidney, or near its entrance into the bladder. Other congenital causes are a faulty connection of the ureter to the pelvis of the kidney, or an aberrant renal artery. Hydronephrosis is sometimes found post mortem in infants and children without evidence of obstruction to the outflow of urine. In these cases the condition is presumed to be due to a neuro-muscular inco-ordination comparable to congenital hypertrophic stenosis of the pylorus.

ACQUIRED.—It is more common in females than in males, and the maximum age incidence in 74 cases collected by Herringham was between the third and sixth decade.

(a) *Bilateral* hydronephrosis results from stricture of the urethra, phimosis, enlarged prostate, obstruction within the bladder, or from a pelvic tumour; the last named is the commonest cause.

(b) *Unilateral* hydronephrosis is due to ureteral obstruction from—

1. Obstruction of the lumen by a stone, growth or blood clot.

2. Stricture of the ureter following ureteritis.

3. Pressure from without due to growths.

4. Torsion of the ureter by displacement of a movable kidney. It is also thought that chronic prostatitis or cervicitis may cause sufficient inflammation to produce some dilatation of the kidney pelvis and upper ureter which lengthens and thus kinks the latter.

Pathology.—Two types of hydronephrosis are recognised, namely, the pelvic type due to upper urinary tract obstruction and the renal type from obstruction to the lower tract. In the former the pelvis of the kidney is dilated and there is less absorption of renal paracachyma in the calices. In the latter the calices are more dilated and there is considerable destruction of kidney substance.

It is generally held that hydronephrosis results from intermittent obstruction. It has been produced experimentally, however, by ligature of the ureter causing complete obstruction. But complete obstruction is more usually followed by atrophy of the kidney.

Symptoms.—Many cases are latent, and give rise to no symptoms. The tumour may be discovered accidentally, or there may be complaint of pain in the flank or back. The onset is insidious.

The symptoms by which a hydronephrosis is indicated are the presence of a renal tumour and complaint of an aching pain in the flank or back, and

sometimes polyuria or hæmaturia. In intermittent hydronephrosis, the tumour suddenly disappears with the passage of a large quantity of watery fluid; after an interval the tumour gradually reappears and finally empties suddenly as before. This sequence may be repeated at intervals. Where true polyuria or hæmaturia occurs it is due to a coincident nephritis or pyelitis. There may be acute exacerbations of the chronic pain, with vomiting and collapse; such attacks may accompany emptying of the hydronephrotic sac.

Course.—When unilateral, hydronephrosis may never cause serious trouble, and intermittent cases may persist for years and finally disappear. In bilateral cases uræmia may supervene. Infection of the kidney is not uncommon, and may lead to acute pyonephrosis. The sac may discharge spontaneously through the ureter, and the fluid never reaccumulate. The sac may rupture into the peritoneum, or rarely through the diaphragm into the lung. Cases have occurred in which the ureter of the sound kidney has been blocked by a calculus.

Diagnosis.—The condition, especially when bilateral and unaccompanied by symptoms, is generally overlooked. In its most characteristic form, where the hydronephrosis is intermittent, the diagnosis is readily made. When the condition is apparent simply as a renal tumour the diagnosis from renal neoplasm (or retro-peritoneal glands in a child) is difficult. When the tumour is large it may be mistaken for an ovarian tumour. The diagnosis can be established by intravenous pyelography supplemented, if necessary, by instrumental pyelography. Aspiration of the sac has been occasionally done for diagnostic purposes; but surgical exploration is a safer measure. Fluid from a hydronephrotic kidney is clear or slightly turbid; it contains albumin, and traces of urea and other urinary constituents; in the deposits are epithelial cells.

Prognosis.—This depends on the cause of the hydronephrosis and the condition of the opposite kidney.

Treatment.—The first indication is to remove the cause. Cases of intermittent hydronephrosis that do not cause serious symptoms should be treated on general lines. An abdominal belt to support a hydronephrotic mobile kidney may be of service.

In unilateral hydronephrosis causing serious symptoms, or of large size, von Lichtenberg's plastic operation or nephrectomy is advisable. Since the state and function of the opposite kidney can be fairly accurately ascertained by pyelography and examination of a sample of urine obtained by ureteral catheterisation, nephrectomy is a less serious risk than it was before these exact methods of diagnosis were available. Sympathectomy has been recommended, but it is not clear how this can produce the desired effect.

In bilateral hydronephrosis the main indication is to remove the cause when possible, and to adopt every measure that may aid in preventing infection of the urinary tract.

PYONEPHROSIS

Definition.—Distension of the renal pelvis with pus, to an extent sufficient to cause a renal tumour.

Ætiology.—The affection is a sequela of pyelitis or hydronephrosis.

There are two main types, namely, tuberculous and pyogenic pyonephrosis. The latter, which is the commoner, is most frequently due to an impacted calculus.

Symptoms.—The patient is wasted, toxic and febrile. Rigors are common. There is a renal tumour, which is tender on palpation, and moves to some extent with respiration. Pyuria is present, unless the ureter is completely obstructed.

Diagnosis.—The differential diagnosis from hydronephrosis is made from the presence of pyuria and of local and general symptoms of bacterial infection. Perinephric abscess gives signs of a more diffuse swelling, usually with œdema and redness of the surrounding skin, and does not move with respiration.

Treatment.—In bilateral cases the treatment is palliative. In unilateral cases nephrectomy is indicated, if tests show that the other kidney is adequate.

TUMOURS OF THE KIDNEY

BENIGN GROWTHS

These are of relatively slight importance.

ADENOMATA are the most common, occurring in the cortex or under the capsule. They may be single or multiple; multiple nodules commonly occur in sclerotic kidneys in old age. They seldom attain any size.

FIBROMATA are not uncommon as nodules, sometimes multiple, in the cortex or medulla. **LIPOMATA** and **ANGIOMATA** are rare.

MALIGNANT TUMOURS

DYSEMBRYOMATA.—These tumours are found most commonly in children under 3 years, and almost always under the age of 11 (Hadfield). They are more often bilateral than carcinoma. They consist of cells remaining at the embryonic level and failing to differentiate in any direction ("Round-celled Sarcoma"). There is a stroma of undifferentiated foetal connective tissue which resembles spindle-celled sarcoma. In some tumours some degree of differentiation may take place. Thus these tumours may contain embryonic striped muscle, primitive cartilage or nervous tissue, and primitive poorly-formed tubules can usually be found. They are yellow and homogeneous on section.

ADENOCARCINOMA OF RENAL TUBULES.—As a result of an examination of the 74 specimens of tumours of the kidney in the St. Bartholomew's Hospital Museum, Hadfield has come to the conclusion that renal tumours previously known as Hypernephromata are in fact Adenocarcinomata. These tumours are single, large, well circumscribed, and often surrounded by a capsule of compressed kidney tissue which is destroyed by pressure rather than by infiltration. These tumours consist of ^{small} of splintering columns of cells. Their blood supply consists of irregularly ^{small} lobed, lake-like sinusoids which lie between the tubules of the growth in contradistinction to adenomata, which are composed of well-formed tubules having well-defined lumina and a simple capillary circulation. Both in adenomata and

adenocarcinomata, and especially in the latter, the cells are infiltrated with a lipoid-fat-glycogen complex ("lipoid infiltration"), which gives these tumours their peculiar yellow colour. Recent and old hæmorrhage is commonly seen. Cystic degeneration often occurs. On section there is fine and coarse lobulation. These tumours may spread along the renal veins into the inferior vena cava, and to the pelvis of the kidney and perinephric tissues.

Symptoms.—1. Hæmaturia is the first symptom in more than 70 per cent. of the cases. It is much less frequent in children. The blood is fluid or clotted, and moulds of the pelvis or ureter may be passed. The hæmaturia is spontaneous, profuse and intermittent; it is little influenced by rest, nor is it provoked by exertion. It may be the only evidence of a neoplasm, and after lasting for a week or 14 days may cease, leaving no further evidence of the growth until at some later date a tumour is felt. The urine frequently contains albumin at intervals.

2. Pain is uncertain. It may be a dragging feeling, or a constant ache. The passage of clots may give rise to renal colic; otherwise the hæmaturia is not accompanied by pain.

3. The presence of a tumour is a most important sign. It is felt on deep palpation bimanually. It is first palpable below the ribs, outside the rectus muscle, as a solid swelling, with rounded borders, that moves with respiration. It may be possible to define its upper border. As the tumour increases, it tends to go forward. It may fill the hollow below the twelfth rib behind, but does not cause a swelling in the back. Large renal tumours cause asymmetry and bulging of the abdominal wall and marked displacement of neighbouring abdominal viscera. On the right side, the ascending colon lies in front, on the left the last part of the transverse colon and the upper part of the descending colon; the tumour is, therefore, resonant on percussion in front. When the tumour is highly vascular, pulsation is felt in it, and a systolic bruit may be heard over it. In later stages, the tumour is liable to become fixed by adhesions.

4. Progressive emaciation is generally late. It may be absent although the tumour is large.

5. Metastases are sometimes the first sign of a renal neoplasm, occurring in the lungs, bones or brain. Secondary deposits in the para-aortic lymph glands may cause obstruction to the inferior vena cava, or this may result from pressure of the tumour itself.

Diagnosis.—Diagnosis is made on the presence of hæmaturia, with a tumour. When hæmaturia occurs alone, and other causes have been excluded by careful clinical, bacteriological and X-ray examination, then a more detailed investigation of the urinary tract must be undertaken immediately. This entails cystoscopy, intravenous pyelography and on occasion retrograde pyelography. When a tumour is the only sign an exploratory laparotomy is advised. The tumour requires to be distinguished from splenomegaly, hepatomegaly and Riedel's lobe. A renal tumour has not the definite edge characteristic of splenomegaly and enlargement of the liver. Enlargement of the liver is often a source of difficulty. A Riedel's lobe is continuous with the liver, does not extend back into the loin, and is dull on percussion. Splenic tumours are recognised by the fact that they tend to occupy an oblique position in the abdominal wall, by the presence of a notch and of a

sharp inner margin, free movement with respiration, and dullness to percussion.

A differential diagnosis from retroperitoneal tumours, including those of the suprarenal, is not always possible, though the suprarenal growths may sometimes be recognised by certain characteristic features. Thus, there is the medullary sarcoma type described by Hutchison, generally occurring in children, characterised by metastases in the skull, ecchymotic swelling of the eyelids, papilloedema and severe anæmia, and the "infant Hercules" type of tumour of the adrenal cortex.

Prognosis.—The disease is almost invariably fatal. Many die within 2 years, and the majority within 4 years, though exceptional cases of survival for 5 to 10 years after operation have been recorded.

Treatment.—Surgical treatment alone holds out a prospect of cure. Symptomatic treatment includes the use of drugs for the relief of pain and the control of hæmaturia.

CYSTS OF THE KIDNEY

SOLITARY CYSTS

These may occur in an otherwise normal organ. They vary in size from very small cysts to tumours of considerable bulk. They result from dilatation of an obstructed tubule, and they may be congenital.

MULTIPLE CYSTS

Multiple cysts of small size are commonly met with in sclerotic kidneys. They result from chronic inflammatory changes that lead to obstruction of the tubules with subsequent dilatation. There are also rare cases of multiple cysts, of large size, whose ætiology and course are little known.

POLYCYSTIC DISEASE OF THE KIDNEYS

Definition.—Polycystic kidneys appear as a massive conglomeration of cysts, varying in size from a pin's head to a marble, separated by dense strands of fibrous tissue, in which little or no renal tissue is evident on naked-eye examination.

Ætiology and Pathology.—The commonest age incidence is between 40 and 50 years; they are relatively common in the decades preceding and following; they may occur in infancy and childhood, and of these a large proportion are in still-born infants. Those occurring in infants are congenital, and other congenital abnormalities may be present. The disease in adults is probably also congenital in origin. In this case it must be progressive, because the renal damage in the later stages is too severe to have been compatible with many years of active life. In this connection it is noted that the disease is often found in more than one member of a family and in successive generations. Its familial incidence, congenital origin, and association with cysts in other organs, especially the liver, all suggest that this disease belongs to the group of congenital-developmental errors.

The organs are enlarged in size, and weigh 20 to 30 ounces each, or even 3 to 4 lb. They have been compared to a bunch of grapes in appearance. The cysts project from the surface and form the mass of the organ. They are lined by a layer of flattened cells, and are filled with fluid. This fluid is clear or turbid, limpid or viscid, colourless or yellowish; it is sometimes blood-stained, giving it a red, purple or green colour. Urea has been found in the fluid, which may also contain fat globules, cellular débris, cholesterol and triple phosphate crystals. On microscopic examination more or less renal parenchyma is found in the septa between the cysts; the tubules are distorted, and exhibit varying degrees of atrophy, degeneration and dilatation, while the glomeruli show changes characteristic of chronic interstitial nephritis. The blood vessels of the kidney undergo sclerotic changes; there is increased fibrous connective tissue and small cell infiltration. In some cases cysts are also found in the liver, ovaries, broad ligament, uterus, pancreas and spleen; but they are rare in any other organ than the liver.

Symptoms.—The affection is nearly always bilateral. When the tumours develop to large size in the foetus, difficulty in labour may result. In the adult there may be no symptoms, or any of the symptoms of chronic nephritis may develop and may terminate in uræmia, cerebral hæmorrhage or cardiac failure. General arterial disease, with raised blood pressure and cardiac hypertrophy, is commonly present; on the other hand, the condition may reach an advanced stage and fatal termination without appreciable cardiac hypertrophy. In a third group the bilateral renal tumours are the most striking features, associated with general malaise, dull aching pain in the loins, and recurrent hæmaturia. The tumours are not tender, and present the ordinary signs of renal tumours (*q.v.*). The urine is of low specific gravity, and commonly contains a trace of albumin; there may be polyuria.

Course.—This usually follows that of “chronic interstitial nephritis.”

Diagnosis.—A condition of “chronic interstitial nephritis” with large palpable kidneys should suggest polycystic disease. Renal neoplasms other than sarcomata are nearly always unilateral. The absence of fever and pyuria excludes bilateral pyonephrosis.

Treatment.—The treatment is that of chronic nephritis. Operation is contra-indicated, since both kidneys are nearly always equally affected.

OTHER FORMS OF CYSTIC DISEASE

Echinococcus cysts may occur in the kidney, and the discharge of the daughter cysts has produced attacks of renal colic. *Cystic degeneration of renal neoplasms* is described elsewhere.

MOVABLE KIDNEY

The kidney is normally held in place by the perirenal fat, the renal vessels and the peritoneum stretched over it. But this does not prevent a certain amount of respiratory excursion, as may be seen either by X-ray examination or in the operating theatre. The range of movement varies between 1 and 2 inches, and is more marked on the right than the left side. The term movable kidney should therefore only be applied to cases where there

is an excessive respiratory descent, so that the upper as well as the lower pole can be felt, or where the kidney can be moved about by external manipulation. As the kidney slips downwards, the lower pole gradually passes towards the middle line, while the organ rotates slightly, causing the hilum to look somewhat upwards.

Ætiology.—Movable kidney is about seven times more common on the right than on the left side. The ascending colon and the hepatic flexure lie on the inner aspect of the right kidney, thus tending to drag it down when the bowel is loaded or dropped. On the left side, on the other hand, the strong costo-colic fold suspends the splenic flexure much more securely, while the descending colon lies to the outer side of the left kidney.

The condition is much commoner in women than in men. In men the kidney pouches are deep, narrow and rapidly diminish in breadth from above downwards, while in women they are much shallower and broader, and diminish only slightly in breadth from above downwards. This natural difference is accentuated in the spare long-waisted women with narrow loins, who are recognised as specially liable to floating kidney. The greater liability of women to chronic constipation further helps to induce dropping on the right side.

Pathology.—Many reasons have been given for the occurrence of movable kidney; but few will stand investigation. Wasting with loss of perirenal fat, or loss of tone in the muscles of the abdominal wall, have been held responsible, but movable kidney is so common apart from such conditions that their importance is doubtful. Glénard emphasised the frequency with which movable kidney is associated with a general visceroptosis; indeed it is rare to find a movable kidney without coloptosis. Naturally, if there is general visceroptosis, the kidney is its most obvious sign. It is a firm organ which can be readily grasped, while the other dropped viscera would elude palpation. As Landau says, "Pleased with his discovery, the physician may impute all subsequent symptoms to the movable kidney." Most of these are really due to visceroptosis.

A serious sequel is the occasional occurrence of hydronephrosis produced by torsion of the ureter during the forward rotation of the organ or by its becoming kinked over the renal vessels. If hydronephrosis occurs, a subsequent infection may convert it into a pyonephrosis.

Symptoms.—There may be no symptoms at all and, if the movable kidney is only discovered in the course of routine examination, it is better not to tell the patient of its existence. It may be well, however, to inform a reliable relation, if such can be found, in order to protect oneself against a less discreet medical attendant subsequently revealing the fact to the patient. The commonest symptom is a constant dragging pain owing to traction on the renal plexus. This most frequently first declares itself between 25 and 35 years of age. A zone of hyperæsthesia corresponding to the distribution of the tenth thoracic segment may also be present. More serious symptoms directly due to movable kidney are Dietl's crises; but these are not common. The attacks are characterised by intense pain radiating down the ureter and through the back, shivering, nausea, vomiting, fever and collapse. The urine is scanty, and may contain blood. Sometimes the pelvis of the kidney may become distended, giving rise to an obvious increase in the size of the organ. This may pass off later, with abundant

discharge of urine, showing that the crises are due to kinking and consequent partial obstruction of the ureter. If repeated, they may lead to hydronephrosis.

The other symptoms which have been attributed to floating kidney are really due to the associated visceroptosis (*q.v.*). But there is no reason to attribute far-reaching nervous consequences to movable kidney, yet, for some enthusiasts, hysteria in women, hypochondriasis in men, and even insanity, are common outcomes. There is little doubt that far too much stress has been laid upon this condition as a cause of manifold complaints.

To detect a movable kidney on the right side, the left hand should be placed under the loin while the patient is recumbent, though some authorities prefer a semi-recumbent posture. The patient should then be told to take a deep breath while the right hand is placed just under the edge of the liver in the nipple line. The kidney may then be felt to slip between the fingers. Usually, this does not cause the patient a definite pain, but a dull, sickening sensation. In the more advanced degree of the condition, the organ may be felt far from its normal position, even to the left of the middle line or nearly down to Poupart's ligament. In examining on the left side, the observer should stand on the patient's left, placing his right hand behind the loin and palpating in front with his left.

A movable kidney usually feels larger than the normal excised organ. This is because of the surrounding investments through which it is felt.

Diagnosis.—Usually this is obvious, as the shape and mobility of the organ are so characteristic. Occasionally, a Riedel's lobe has been taken for movable kidney; but the continuity of the former with the liver should prevent this mistake being made. In the same way, a distended gall-bladder is continuous with the liver, and cannot be separated from it. Moreover, it is not nearly so movable, and curves characteristically towards the umbilicus. Carcinoma of the pylorus has offered difficulties in some cases; examination of the stools for occult blood, a test-meal and X-ray examination would clear up the diagnosis. Scybala near the flexures of the colon may be mistaken for floating kidney; but their indefinite shape and inelasticity generally help to distinguish them. Their disappearance after a series of enemata would settle the question. In one case a mesenteric cyst appeared closely to resemble a floating kidney.

Prognosis.—Apart from the development of hydronephrosis, movable kidney does not tend to shorten life in any way. It is doubtful whether a kidney once prolapsed can ever maintain the normal position unaided.

Treatment.—Some cases call for no local treatment, though the associated visceroptosis and neurasthenia will require attention. If pain is felt, the adoption, for a short time, of the knee-elbow position will help to replace the kidney and relieve the tension on the renal plexus. If pain is at all frequent, some form of abdominal support, such as a specially designed corset, should be worn. Hurst has urged that the support should be designed to increase the general intra-abdominal pressure, and not to replace any one viscus. We are convinced that this is sound and that, in many cases, a "kidney belt" is worse than useless, while the addition of ingeniously placed pads only increases the discomfort. Whatever the form of the support, it need only be worn while the patient is in the erect posture, and it is best fitted while she is recumbent, preferably with the pelvis raised on a pillow so as to

aid the replacement of the kidney. Often, when a support of this kind has been worn for a year or two, it is possible to give it up without recurrence of symptoms. Breathing exercises to develop the expansion of the lower thorax, with exercises to improve the tone of the abdominal wall have in many cases proved more efficacious than a passive support. Operation should not be advised except for recurrent Dietl's crises or when there is evidence of hydronephrosis, when nephropexy may be done. But even this may not be successful, and ultimately nephrectomy may be required for the hydronephrosis.

Treatment of Dietl's crises.—The patient must be put to bed and hot fomentations or antiphlogistine applied to the affected side. A hypodermic injection of a quarter to one-third of a grain of morphine may be required if the pain is severe. Usually this is sufficient but, should the attack last more than a few hours, an attempt must be made, under an anæsthetic, to rectify the position of the kidney by manipulation. Naturally, conditions are unfavourable for nephropexy during or immediately after a crisis, because of the congested state of the organ.

W. LANGDON-BROWN.
GEOFFREY EVANS.

SECTION XVII

DISEASES OF THE JOINTS AND INFLAMMATORY DISEASES OF THE FIBROUS TISSUES AND MUSCLES

ARTHRITIS

The diagnosis of "arthritis" should be reserved for cases in which there are pathological changes at the surface of a joint. In the past there has been a tendency to employ this term too loosely.

The clinical conception of arthritis is much simplified by modern classification, which divides it into two clear-cut *clinical* types, each of which presents distinctive features. These are the rheumatoid arthritic type and the osteo-arthritic type. Consequently such terms as "arthritis deformans" should no longer be used.

The features of the rheumatoid and osteo-arthritic types of arthritis will be found under their respective headings below, and since the criteria of these types are clinical, this terminology can be correctly employed in those cases in which the ætiology remains obscure.

The most basic difference between these two types is that rheumatoid arthritis is a generalized disease in which the most obvious local effects fall upon the locomotor system; while osteo-arthritis is a degenerative type of condition, which, without affecting the patient's general health, for various reasons becomes localised in certain joints.

There are, in addition, certain cases which are referred to as "Mixed arthritis," in which the degenerative lesions of osteo-arthritis become superimposed upon those of an inflammatory arthritis of the rheumatoid type.

1. RHEUMATOID ARTHRITIS

Synonyms.-- Atrophic Arthritis; Infective Polyarthritis.

Rheumatoid arthritis has generally been described as being of two types, namely, the "classical" or idiopathic type, of which the causation is unknown; and the infective type, in which a discoverable infective agent is causative. It was the opinion of the committee appointed by the Royal College of Physicians (1934) that the term "rheumatoid arthritis" is best reserved for the first type, and "infective arthritis" for the second. It is, however, generally impossible to distinguish between the two types on purely clinical grounds, and the distinction should therefore depend on the pathological findings. Either type may, however, be correctly referred to as the "rheumatoid type" of arthritis.

Rheumatoid arthritis is a generalised progressive disease affecting principally the joints, which are swollen and painful. If unchecked great destruction and deformity results.

Ætiology.—The malady is said to occur at least five times as frequently among females. The type of patient most commonly affected by the classical form of the disease is a young woman between 20-40 years of age, and of slender delicate build, with a somewhat narrow back and acute costal angle. The affection seldom commences after the menopause. Predisposing factors exist in many cases and include the climateric, chronic gout, malnutrition, emotional shock, and minor focal infection which is not in itself causative of the condition.

Pathology.—The pathological processes are inflammatory in nature. The soft tissues and the white fibrous structures around the joints are the first to be affected. The inflammation then spreads to the capsule and synovium, and granulation tissue forms in the angle made by the articular cartilage with the synovium. The latter then gradually extends inwards as a ring of "pannus," covering and eventually replacing the articular cartilage. As this happens on both articular surfaces the tendency is for them to adhere, especially if the joint is immobilised, and so fibrous ankylosis occurs, which in some cases progresses to a bony ankylosis.

—There are atrophic changes affecting the skin, subcutaneous tissues, muscles, ligaments, joints and bones. This latter condition of generalised osteoporosis shows as the first *X-ray* evidence of the disease. The peri-articular swelling can also be seen in outline, but actual joint changes do not occur until considerably later. Some degree of patchy recalcification may be observed when the progress of the disease is checked. Osteophytes are never found in rheumatoid arthritis, but in late cases very considerable disorganisation of the joints take place, and in these areas the bone sometimes gives the appearance of having been dissolved away.

The chief pathological change in the blood is an increase in the sedimentation rate of the red blood cells. This is an important index of activity, and the response of a patient to treatment over a considerable period can be estimated with some accuracy by means of this together with clinical observation. A secondary anæmia is usually present in the pre-arthritis phase. The glucose tolerance of the patient is generally found to be reduced in the active stages of the disease.

Symptoms.—There is in most cases a prodromal period, during which the patient loses a considerable amount of weight; and fatigue, both mental and physical, is a marked feature in nearly all cases. There may be other symptoms, such as paræsthesia, Raynaud-like phenomena, irregular menstruation, tachycardia, sweating, and a secondary anæmia.

The onset of the arthritic phase is often announced by a swelling of the mid-phalangeal joints of the second and third fingers of both hands. It is usually insidious, but is acute in about 10 per cent. of cases. In the case of the former, it is not uncommon for the disease to be marked by long periods of low and intermittent fever. The cause of this is not known. The thyroid gland is also sometimes enlarged, and fibrositic pains may be complained of.

Wasting of the small muscles of the hands is generally the next event, while the uncompensated pull of the interossei muscles, which appear to be affected a little later in the disease, tends, in combination with trauma, to drag the fingers into the typical position of ulnar flexion, in which they often become ultimately fixed. The affection then spreads centripetally

towards the trunk, involving in turn the wrists (which often become the seat of ankylosis), ankles, elbows, knees, shoulders, hips and jaw. The bilateral and symmetrical way in which all the joints are affected is a striking feature of the disease. This point, however, is not so well marked in cases of infective arthritis as in the idiopathic type of rheumatoid arthritis. In some cases the spine itself in due course becomes affected. (See p. 1362).

Whenever a joint becomes involved it will be noticed that the muscles which control it, particularly the extensors, waste rapidly, giving rise to the varying flexion deformities seen in the later stages. These may be perpetuated by a fibrous ankylosis of the affected joints, and contraction of the joint capsule. Bony ankylosis may follow this stage, and when it does so it generally occurs in the wrists and the bones of the carpus in the first place.

In certain cases enlargement of the lymphatic glands occurs, and even the spleen may become palpable. Sufferers with rheumatoid arthritis generally experience considerable pain which interferes with their sleep, and this adds progressively to the severity of the condition.

Symptoms often clear up during pregnancy occurring during the course of the disease, but in most cases they return with renewed vigour after parturition. It should not be forgotten that periodical intermissions are a well-observed occasional feature of the disease, which sometimes results in undeserved credit to any treatment which is being undertaken at the time.

Prognosis.—Under properly planned and supervised treatment, about 20 per cent. of patients should prove completely amenable to therapy, a further 50 per cent. should show great or moderate improvement, and an additional 20 per cent. are improved to some extent; leaving 5–10 per cent. of cases which appear to be entirely resistant to treatment of all kinds. With modern methods of splintage, gross deformity should very seldom occur, even when ankylosis takes place. It should be remembered, however, that in many cases treatment of some sort for months or even years is required, and that relapse may occur after apparent cure. Enlargement of the lymphatic glands has often seemed to be of bad prognostic significance, especially when the white cell count is profoundly altered in addition, and subcutaneous nodules appear. If the disease is not checked, the end-result is complete and painful crippledom. This often takes place within a very short period, particularly when the patient is young. Cases which occur later in life tend on the whole to be less virulent in their course.

Treatment.—As yet there is no specific form of therapy. It is by a careful and intelligent selection and combination of methods suited to the individual patient that success will be achieved.

Diet.—This should be rich in vitamins, especially B and C, and should be of the high caloric type, except in the rare cases in which the patient is overweight. When the sufferer is much underweight and does not return to normal by dietetic means alone, a small dose of insulin (5–10 units) may be administered 15 to 20 minutes before two meals in the day, which should be rich in carbohydrates.

Physical Therapy.—In the acute phase, most methods of external treatment will prove too exhausting and are therefore undesirable. An exception is progressively graded ultra-violet rays, which stimulate the skin, and to some extent the patient's powers of resistance. Later, massage and heat

will help to relax the muscle spasm, which is often a cause of pain and ultimate deformity.

Drug Therapy.—This has only a limited scope in arthritis of all types, and should never be the sole method of treatment employed. On general principles such patients need iron for anæmia, laxatives for constipation, and analgesics—especially aspirin, phenacetin, and, when necessary, in addition Dover's powder (gr. 10–15), or codeine phosphate (gr. $\frac{1}{4}$ – $\frac{1}{2}$)—for pain and sleeplessness. A valuable remedy is guaiacol carbonate, which is an intestinal antiseptic and analgesic. It may be given in doses of 5 to 10 grs. three times daily after food. A useful combination is guaiacol carbonate, grs. 8, aspirin, grs. 4, in cachet form, three times daily after food. Guaiacol carbonate is free from any danger of toxic symptoms. Arsenic is of considerable tonic value, especially if combined with nux vomica and taken before meals. Cod-liver oil also has great value and, if preferred, may be combined with malt extract. It should be given in full doses of one ounce two or three times daily if such can be tolerated. Thyroid is useful in cases occurring about the menopause. Iodine in the form of liquor iodi simplex once daily is useful in some cases.

Injections of gold salts have recently been found helpful in many cases suffering from the idiopathic type of the disease, especially when the onset is fairly recent and the blood sedimentation is high. When the malady is known definitely to be the result of an infection their employment is inadvisable until this has been eradicated.

This method of treatment is contra-indicated with renal or hepatic damage, diabetes mellitus, eczema, severe anæmia, colitis, pregnancy, hæmophilia, or a history of having suffered from any severe alteration in the white-cell count in the past, or purpura. With regard to other cases, the dangers of reaction, complications and mortality should be taken into consideration. There should be a complete blood count, blood sedimentation test, and an examination of the urine for albumin. The chief complications are purpura, rashes, boils, exfoliative dermatitis, gastro-enteritis and colitis, nephritis, jaundice, aplastic anæmia and stomatitis. There is a mortality of about 1 per cent. attendant upon this form of therapy. The patient herself should decide after being informed of the danger of reaction, complications and mortality. She should not be allowed to expose herself to strong sunshine or ultra-violet light, for fear of pigmentation.

It is well not to administer gold near the period of the menses, as skin eruptions are said to be more liable to occur then. There are several preparations of gold salts on the market. It is wise always to employ those which are administered intramuscularly. Whether they are suspended in an oily or in an aqueous solution appears to be immaterial. The initial dose should be 0.01 gm., and subsequent doses may be 0.05 and 0.1. This latter dose should not be exceeded, nor should the injections generally be given more frequently than once weekly. The total amount in a complete course should in most cases be limited to 1–1.5 gm. An interval of at least six weeks should then elapse before a further course is commenced. The dosage should be adjusted according to the patient's condition and response to previous injections. Patients of very light weight probably require smaller initial doses and more careful subsequent grading. A complete blood count, blood sedimentation rate test, and urine examination for albumin should be

repeated at regular intervals during the treatment. Great care should be exercised in watching for the first signs of reaction, or any other complication. In the first instance, no further injections should be given until 48 hours after the reaction has entirely subsided. In the second, the injections should be stopped immediately, and not resumed, if at all, until the patient has been free for two to three weeks. If complications appear treatment is palliative, while if the skin is involved also, calamine lotion, with 1 per cent. phenol, should be applied, and a mixture containing bromide and phenobarbitone (gr. $\frac{1}{2}$) should be administered thrice daily until the complication has disappeared. Patients with jaundice should be put to bed and treated as if suffering with catarrhal jaundice. For the other complications 10 c.c. of a freshly prepared 20 per cent. solution of sodium thiosulphate should be given intravenously every day, and 10 c.c. of 10 per cent. calcium gluconate may be administered intramuscularly at the same time.

Vaccine Treatment.—In infective arthritis a course of vaccine injections is a rational form of therapy. In certain cases it is very successful in reducing the activity of the disease. But it should, however, not be expected to take the place of the simple orthopædic and other measures necessary to prevent contracture of the joints. When the patient is suffering from idiopathic rheumatoid arthritis the results of vaccine therapy are less certain. But it is often worth a trial, either previous to the employment of gold therapy or in the interval between the courses. In cases in which gold is contra-indicated or is not well tolerated, it may be the method of choice, except in those who are febrile and much exhausted, when the powers of reaction are very low and harm may result. The right dose is the lowest which is found to provoke a favourable reaction, and not the highest which can be tolerated, as is believed by some.

Protein shock may be given in the form of intravenous T.A.B. vaccine injections. This is a non-specific procedure designed to raise the patient's temperature temporarily in the hope of benefiting him subsequently. Such treatment should never be undertaken when the patient is in an active phase of the disease. Once improvement has started, however, it may be justifiable to endeavour to speed its tempo by this means. Three to five injections should be administered, the dose varying according to the age and weight of the patient. At least 24 hours of normal temperature should be allowed between the injections, which should not in any case be given more frequently than twice weekly.

Additional methods of treatment useful in certain cases include blood transfusions in those in which improvement by other means is long delayed; colonic lavage when there is reason to believe that the lower bowel is implicated; and sulphur injections, which have a similar object to protein shock.

Finally, certain considerations regarding *focal sepsis*: It is a safe rule in such a disease such as rheumatoid arthritis, in which the exact ætiology is often obscure, that "whatever is found wrong—put it right!" This should extend to the discovery of foci of infection. It is, however, unwise to embark upon operative procedures while the patient is in a condition of debility, or while the disease is running an acute febrile course, with marked joint pain and swelling. In such patients an endeavour should previously be made to build up the general health. If after 4–8 weeks no improvement has occurred and the focus is still believed to be of importance, cautious

measures for its removal may be initiated. The patient should in such cases be warned that it is unlikely that the removal of such a focus will cure the arthritis, but that his general health, and the powers of active resistance will be stimulated thereby. The foci of infection which are of particular importance and should always be investigated are situated in the tonsils, the sinuses, and teeth—in that order. Less important foci include the colon, the appendix, gall-bladder, cervix, tubes, prostate and bladder. If more than one focus of infection is found the most apparently active should be treated first. If this cannot be determined the matter should be dealt with in the order mentioned above. In cases in which the sinuses and the tonsils both require surgical attention it is important to allow a period of several weeks to elapse between the two operations.

2. SPECIFIC INFECTIVE ARTHRITIS (OF RHEUMATOID TYPE)

GONOCOCCAL ARTHRITIS

From 1 to 5 per cent. of those infected with gonorrhœa develop gonococcal arthritis. The latter is also found in babies, whose infection occurs at birth. In view of the fact that infection due to this cause frequently ends in great crippling and bony ankylosis of the joints, it is important to make the diagnosis at the earliest possible stage. If this is done the prognosis is now good.

The clinical appearance and course of gonococcal arthritis is similar to that of the idiopathic type of rheumatoid arthritis when the onset of the latter is acute. A differential diagnosis may be made from the following points: (1) A recent history of gonococcal infection or urethritis. Unless specifically questioned, patients often omit to mention this. (2) The onset of joint symptoms within three weeks of such an infection. (3) There is a predominance of 3 : 1 in males, unlike true rheumatoid arthritis. As in the latter disease, however, the malady is usually a polyarthritis from the onset. (4) The knees, wrists and ankles are generally the seat of the most virulent attack; while a painful teno-synovitis around the wrists and ankles is a common forerunner of actual arthritis. (5) Conjunctivitis and irido-cyclitis is not uncommonly associated with gonococcal arthritis.

If pathological aid is available the complement fixation test of the blood is found to be positive in about 80 per cent. of cases after the first month. Gonococci may in many cases also be grown by special methods from samples of the joint fluid, which affords an immediate confirmation of the diagnosis.

The main points in treatment are (1) that of the primary focus and prostate; (2) the sulphanilamide group of drugs; and (3) artificial fever therapy (hyperpyrexia). The last, whenever it is available, is considered by American observers to be "specific" for gonorrhœal polyarthritis.

In the chronic stages cases should be treated on the same lines as rheumatoid arthritis, except that gold salts are contra-indicated. (See also p. 23).

PNEUMOCOCCAL ARTHRITIS

A polyarthritis clinically of the rheumatoid type is a rare sequel of lobar pneumonia. It affects children more commonly than adults. An arthritis

affecting one or more of the larger joints is somewhat commoner. These conditions will generally occur subsequent to the stage of pneumonic resolution. Primary pneumococcal arthritis of either type is uncommon.

Pathology.—The joint fluid is in most cases purulent, and pneumococci can be found in it, except in cases which have received sulphapyridine in large doses.

Prognosis.—If the patient survives the pulmonary infection his resistance to the organism should be good, and joint function is in most cases preserved provided erosion of the cartilage has not taken place.

Treatment.—Joints affected in this way should be aspirated early and irrigated with isotonic saline solution, although drainage is not advisable. They should be immobilised in light plaster splints, which should be removed daily to permit of gentle movement. Sulphapyridine should be given in full doses.

ACUTE SUPPURATIVE ARTHRITIS

This is often polyarticular in its distribution, and may be mistaken at first for acute rheumatic fever or rheumatoid arthritis. It is commoner in children than in adults.

Ætiology.—The condition may be a blood-borne infection (metastatic), or may arise as an extension from neighbouring areas of osteomyelitis, or other infection. The former is the more common and may be secondary to a focus of infection in the middle ear, throat, sinuses or prostate. It may also follow the acute specific fevers, particularly scarlet fever and septic tonsillitis. It has also been reported as following meningitis, septic endocarditis, infected varicosities and burns, pyelitis and furunculosis. An arthritis following typhoid fever is not generally suppurative, but may become so.

The organisms which are chiefly responsible are the hæmolytic streptococcus, the staphylococcus, the pneumococcus and the gonococcus.

Symptoms.—These include an acute onset of chills and sweats, pyrexia, local pain and tenderness in the joints, with redness, swelling and limitation of movement. There is in most cases a high degree of polymorphonuclear leucocytosis.

Course.—The joint fluid rapidly becomes purulent and extensive damage to the joints will occur if treatment is not instituted rapidly. Badly damaged joints generally ankylose ultimately. The mortality amongst such cases is in the neighbourhood of 20 per cent.

Treatment.—This should be directed towards the primary source of the infection as well as the affected joints. If the organism is known to be a streptococcus, pneumococcus, gonococcus or meningococcus, drugs of the sulphonamide or sulphapyridine group will be of value. But they are ineffective in cases of staphylococcal arthritis, in which condition sulphathiazole may be of benefit.

Immediate aspiration of the affected joints is essential, both for diagnosis and therapeutically, and lavage with normal saline should be done. In some cases it is necessary to open the joint for this purpose. Blood transfusion is valuable in these cases; and orthopædic care is required if ankylosis appears probable. Hyperpyrexia treatment is useless in the majority of cases.

TUBERCULOUS ARTHRITIS

Tuberculous arthritis usually occurs in young patients, and is an infection from a primary tuberculous focus elsewhere in the body, and frequently signs of tuberculosis in other parts of the body are present. The possibility of an arthritis in a young subject being tuberculous should always be borne in mind, and an X-ray examination is of great value in differentiating this type from other varieties. The subject is dealt with fully in surgical books, to which the reader is referred.

DYSENTERIC ARTHRITIS

A polyarthritis of the rheumatoid type follows bacillary dysentery in about 3 per cent. of cases, at an interval varying from three weeks to several months after the cessation of acute symptoms. It may also occur in the course of a chronic ulcerative colitis. In some cases the process will only affect one joint, but in either event the process commences as an inflammation of the periarticular tissues and progressively invades the joint surfaces. Suppuration is very rare.

Treatment will be directed to the dysenteric condition, and should be palliative so far as the joints are concerned.

UNDULANT FEVER OR BRUCELLIASIS

A mild polyarthritis due to the organism of this disease is probably more frequent than is usually believed. It is generally associated with myalgia, and sometimes with intermittent hydrarthrosis. The onset may be acute or chronic, and the clinical picture may closely resemble rheumatoid arthritis or rheumatic fever. Cases have also been confused with pulmonary tuberculosis and "neurasthenia."

The general symptoms are indefinite and multiple, and include malaise, long-continued low-grade pyrexia, which "undulates," loss of weight, sweating and depression. The blood may show a secondary anæmia and a leucopenia with relative lymphocytosis. The agglutination tests will probably also be positive if the disease is of some weeks duration.

DENGUE

This disease gives rise to a very acute form of peri-arthritis. Intense pain and sometimes swelling occur in the tendons and muscles around the joints. These usually disappear when the fever subsides, but in the stage of convalescence may recur and last for weeks or months. The condition should be differentiated from rheumatic fever, from which it differs in being epidemic and in not responding to salicylates.

MENINGOCOCCAL ARTHRITIS

This is not uncommon in the second week of cerebro-spinal fever, and may be polyarticular or monarticular. It sometimes becomes purulent. It resembles gonococcal arthritis closely, except that it is generally less severe. There is also a sporadic form—chronic meningococcal septicæmia—

which occurs in the absence of meningitis. The diagnosis in these cases rests upon the presence of intermittent fever, a rash, which may be purpuric, and a positive blood culture. The fluid from the joints affected may contain the organism. The patients often seem surprisingly well, and complain of little except joint pains. Both these types respond well and rapidly to sulphapyridine therapy.

3. OSTEO-ARTHRITIS

Synonyms.—Hypertrophic Arthritis; Arthrosis; Arthritis Deformans; Morbus Coxæ Senilis (of hip).

Osteo-arthritis is a degenerative condition which affects the articular cartilages and weight-bearing surfaces of the larger joints.

Ætiology.—The known factors include trauma, certain disorders of metabolism, and nervous diseases, *e.g.* Charcot's joints. Senility is also a cause, as may occasionally be prolonged toxæmia. There is, however, no evidence that focal sepsis is primarily concerned in the ætiology of this type of arthritis.

The malady tends to occur principally in men over middle life who have led a strenuous existence; while in women it mainly affects the knees and is secondary to the proliferative synovitis which is often a distressing feature of the menopausal period.

Pathology.—The changes which occur in the joints affect primarily the articular cartilage, which in the early stages will show grooving and "fibrillation," starting at the points where the pressure of the opposing surfaces is greatest. Later, the cartilage may actually wear through at these points, and the two bony surfaces will come into contact. When this happens, the constant rubbing of bone on bone will gradually polish and "eburnate" these areas. At the same time a gradually progressive enlargement of the articular surfaces will occur, which culminates in the production of "lipping" and of bony outgrowths from the joint margins called osteophytes. These excrescences may be easily palpable at the joint margins, and are the typical lesions of osteo-arthritis. No constant changes are found in the synovium.

Symptoms.—The onset of the disease is insidious. The first symptom to be complained of is usually stiffness, often accompanied by some pain after exertion. The site is generally one or more of the larger joints; or it may be any joint which is subjected to particular stresses as the result of the patient's occupation or sport. In the course of time considerable wasting of the muscles controlling the affected joints supervenes. These therefore tend to become unstable and so liable to further trauma. The coarse grating which can be elicited in joints affected with the disease is due to an accompanying teno-synovitis, and is no measure of the actual damage to joints. When extrinsic joint changes have occurred, the patient usually experiences considerable pain, particularly on bearing weight. The movements of the joint also become much limited on account of spasm of the surrounding muscles, which may in itself be a cause of pain. There is generally not much effusion present. Occasionally new bone formation may limit the movements of the joint, although this is not very common. Small rounded bony swellings on the terminal phalanges of the fingers and thumbs, termed

Heberden's nodes, not infrequently develop during the course of the disease. These may be the cause of considerable pain in their early stages.

The examination of a hip joint affected with early osteo-arthritis will reveal some limitation and pain on rotation and often also of abduction of the joint, long before the movements of flexion and extension are appreciably interfered with. In addition, it may be found that such a patient when standing will not support his weight equally on both hips, in order to avoid pain. Some wasting of the gluteus muscle on the affected side will also be evident fairly early; while in advanced cases actual shortening of the affected limb will occur, either the result of absorption of the femoral head or from its dislocation upwards. Another point to remember is the possibility that even severe pain complained of in the knee may in reality be referred from a diseased hip; and in such cases if a full examination of the patient is omitted treatment may be directed to the wrong joint. "Sciatica" is often found to be the result of osteo-arthritis of the hip, or of the lumbar spine, and is sometimes the initial symptom.

Osteo-arthritis of the lumbar spine is frequently present without giving rise to symptoms. Such cases are often discovered radiologically in the course of an examination for another purpose. This sometimes gives rise to difficulty in compensation cases when existing symptoms may be attributed to this cause *post hoc*. The sacro-iliac joints are in the same way frequently reported as being the seat of osteo-arthritis. In many cases, however, there is an absence of symptoms and so no treatment is required, unless low backache or sciatica supervenes.

The osteo-arthritic joint does not ankylose, but may become locked as the result of excessive osteophyte formation. In other cases it becomes unstable, owing to continued use in the presence of insufficient muscular support, due to muscular wasting; in these cases the joint surfaces may ultimately become very disorganised.

When a weight-bearing joint is affected, the patient suffers great pain on standing, and a certain amount of absorption of articular bone may occur, resulting in some shortening of the limb.

In some cases the articular cartilage may become fragmented, or osteophytes may break off into the joint cavity. In both these circumstances they form loose bodies which give rise to all the symptoms usual in that condition, in addition to those of the arthritis.

The results of radiological examination and blood sedimentation rate and glucose tolerance are described under Diagnosis.

The general health is not affected, unless the result of unaccustomed inactivity necessitated by the affection.

Diagnosis.—Osteo-arthritis should never be confused with rheumatoid arthritis, the two conditions being entirely different. The former is a degenerative condition affecting one or two joints, usually the larger ones. There is generally a history of trauma in the past, or of continued trauma of a minor nature, such as some occupational or sporting stress or strain; or a postural defect; or in some cases a sudden increase of weight, with resultant strain on the ankles, knees, or hips, as may occur at the menopause. Apart from the menopausal group of cases, the patient is more often a male, and his general health is not directly affected by the disease. Again, the pain is generally relieved by rest.

In rheumatoid arthritis, on the other hand, the joint changes are inflammatory in nature, and trauma is not a marked feature. The onset is in the smaller joints, many of which are generally affected, symmetrically and bilaterally. Finally, the patient is usually a woman, and there are indications of general ill-health and also loss of weight, which often preceded the joint manifestations.

The *radiographic* appearance of a joint affected with osteo-arthritis is generally typical. The bone density is unaffected and the joint space is narrowed; this depends upon the amount of erosion which has occurred in the cartilage. Osteophytes will be seen at the joint margins, and there is frequently also a deposition of calcium in the attached ends of certain tendons, such as those of the patella and ligaments, *e.g.* the cruciates, which result in an appearance of "spiking." Such, however, should not be confused with osteophyte formation, as it will sometimes be found independently of the existence of osteo-arthritis. In osteo-arthritis of the hip considerable deformity, both of the head of the femur and of the acetabulum, may be seen, and, in addition, small degenerative cystic areas adjacent to the joint are not infrequently noticed.

The blood sedimentation rate and the glucose tolerance are normal.

Differential Diagnosis.—Conditions which are liable to be confused with osteo-arthritis are Paget's disease, osteochondrosis desiccans, and occasionally neoplastic growths of the articular ends of bones.

Prognosis.—If not treated the course of osteo-arthritis is progressive and generally ends in disablement. Much, however, can be done in the early stage to prevent this, by relieving the affected joint of all possible strain, and by support and correct treatment. The outlook is perhaps best in that form known as "menopausal arthritis," provided full and adequate measures are taken before the malady becomes established. In such cases a return to the normal may be anticipated in the course of time.

Treatment.—As in osteo-arthritis there is an absence of general symptoms, there is no indication for general treatment other than seeing that the patients receive an adequate supply of vitamins and that their bowels act regularly, while definite periods of rest are advisable. If the patient is obese, a diet low in fats and carbohydrates should be insisted upon if success from other measures is to be achieved. The caloric value should not exceed 900–1600 calories, and fluids should not be taken at the same time as food. The patients should be reassured that they will not become extensively and hopelessly crippled, as may occur in rheumatoid arthritis.

It is the experience of most observers that little benefit is to be anticipated as the result of removing foci of infection. If, however, these are found they should be dealt with on their merits. If varicose veins are present, as is often the case when the malady is situated in the knees, treatment directed towards these will frequently result in improvement in the joints.

The drugs chiefly valuable in this condition are those of the analgesic group, such as aspirin, guaiacol carbonate, phenacetin and occasionally codeine. They should not be prescribed as a routine and should only be used when pain is severe. The iodides are also employed on general principles, and appear to be of value in some cases, as are sulphur (by mouth) and guaiacum. Gold salts should not be used in this type of arthritis.

In cases which arise about the time of the menopause small doses of thyroid are often of value, but should be combined with the other measures outlined, particularly those for reduction in weight, muscular re-education and joint support by means of elastoplast.

Physical therapy of some sort is essential in treatment of osteo-arthritis. The desiderata are: heat, to stimulate the failing circulation locally and to relieve muscle spasm and consequently pain; massage, to maintain and stimulate the nutrition and drainage of the skin and underlying tissues; and movement, in order to maintain the mobility of the joint, to prevent or repair muscle wasting, and discourage the formation of adhesions.

Movement should, if possible, be active, *i.e.* special exercises, or if this is not feasible at the outset, electrical stimulation by means of the surging faradic current, or hydrotherapy, if available, may be substituted. Movement of the affected joint should, however, as far as possible be disassociated from weight-bearing through the affected joint, in order to allow of repair in the cartilage.

A further method of stimulating the circulation reflexly is by means of histamine ionisation. Sufficient current should be used to produce a temporary urticarial reaction in the skin under the pad. When dealing with the extremities, paraffin wax applied at a high temperature is also useful as a means of applying heat.

The question of posture, or "body mechanics," is an important and neglected aspect of many cases of osteo-arthritis. For instance, pronated feet may cause strain and, later, arthritis in both knees and lumbar spine; as may a pendulous abdomen. Proper postural exercises should be taught, which the patient should continue until the correct posture is maintained reflexly.

When after a period of rest the patient begins to put weight on to the affected joint again, the latter should always be adequately supported. For this purpose a crêpe bandage, or better still elastoplast, is of great value. In some cases special appliances, such as the Howard-Marsh splint for the knees, or a back support when the spine is affected, may be needed for a period in order to protect the joint. For the feet proper arch supports may be necessary, while in cases of severe arthritis of the hip or knee, when weight-bearing continues to give great pain, some form of walking caliper, whereby the weight of the body is "by-passed" from the ischium down to the heel of the shoe by a light metal rod, is indicated.

In very advanced cases operative procedures will sometimes prove necessary. Chief amongst these are synovectomy; in those cases in which soft tissue proliferation is not responsive to other treatment. When in the hip-joint a small degree of painful movement is all that remains possible of achievement, arthrodesis, either by open operation or by means of the Smith-Petersen pin, will often be the best method of treatment. Multiple bone puncture has recently been advocated (empirically), but no controlled series of the effects of this has yet appeared.

Recently X-ray treatment has been considerably employed as an anæsthetic procedure in painful osteo-arthritis. The effects are very variable but on the whole appear to be more successful when it is the superficially placed joints which are to be treated.

Finally, the question of climate should be considered. If the economic

status of the patient will allow him to live in a warm climate, this will in most cases prove to be very beneficial.

4. HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY (see p. 1375)

5. ARTHRITIS DUE TO GOUT (see pp. 432, 434)

6. SPONDYLITIS

Spondylitis is arthritis of the spine. Most cases fall into one of three types: (1) the ankylosing type—spondylitis ankylopoietica; (2) rheumatoid arthritis, in which the spine is involved as a secondary spondylitis; and (3) osteo-arthritic type of spondylitis.

1. ANKYLOSING TYPE OF SPONDYLITIS

Synonyms.—Ankylosing Spondylitis; Spondylitis Ankylopoietica; Atrophic type of Spondylitis; Spondylose Rhizomélisque; Von Bechterew's Disease; Marie-Strumpell Disease. It is now realised that all these terms apply to the rheumatoid type of spondylitis or sub-varieties of this.

Ætiology.—As the condition is a form of rheumatoid arthritis with a special localisation to the spine, the predisposing and exciting factors are the same as in that disease (*q.v.*). The age of onset is also similar, occurring chiefly in the young. The malady differs, however, in chiefly affecting males. It is considerably less common than the classical form of rheumatoid arthritis.

Pathology.—The only true joints in the spinal column are those which permit of movement between the intervertebral articular facets and the costo-vertebral joints. This type of arthritis commences as a synovitis of these joints, together with some osteoporosis of the vertebral bodies. In the vast majority of cases radiological evidence of an infective process will be found in the sacro-iliac joints, concurrently with these changes. The nature of this is unknown. The ligaments surrounding the affected joints subsequently calcify, and those portions of the spinal column become rigid. When this process is complete the lateral borders of the intervertebral discs also calcify, as do the anterior and lateral longitudinal ligaments, the whole process resulting in a bamboo-like appearance which is typical of the condition.

Symptoms.—The early symptoms of the rheumatoid type of spondylitis are usually of a diffuse and insignificant nature, and are therefore generally overlooked until they are localised in the spinal region, which may be comparatively late in the disease. The principal complaint will often be of a diffuse fibrositis which chiefly affects the upper part of the body. This syndrome, if it persists, should always arouse a suspicion of spondylitis in the case of a young male. In others pain may be referred directly from the affected spinal segment, and may simulate that of renal calculus, pleurisy or even of tabes. Sometimes neuritis or numbness and loss of power in the limbs, or severe spasm of the muscles of the back may be complained of. All these symptoms are usually intensified on forced movement of the spine,

and in addition pain is often elicited by firmly tapping the spinous processes involved.

There is also increasing stiffness in the back, and ultimately this may become completely rigid (" poker back "), generally in a position of kyphosis and slight forward flexion of the spine. In untreated cases this position becomes very exaggerated, the chin sinks into the chest wall, and the patient is unable to stand erect or move. Those who are not bedridden move with a characteristic slow, bent, shuffling gait. As the costo-vertebral joints ankylose, so does the respiratory expansion of the chest diminish.

For the results of radiological examination, see later.

Complications.—Those who die with this disease generally do so from an intercurrent respiratory infection, the result of the diminished or absent expansion permitted by the ankylosed costo-vertebral joints. The disease in itself usually does not shorten life.

Diagnosis.—All patients, especially young and athletic males, who complain of vague pains affecting the limbs and upper part of the body which do not respond to treatment should be suspect. The presence of detectable rigidity of the spine is unusual at this stage. If the blood sedimentation rate test is high this increases the suspicion and should lead to the patient being X-rayed. The radiological appearances at this stage are loss of definition (" wooliness ") of the sacro-iliac joints, osteoporosis of the neighbouring bones and lumbar vertebral bodies. Later, the sacro-iliac joints become obliterated, and the intervertebral joints, if they can be shown, are hazy and later on ankylosed. The edges of the intervertebral discs and the spinal ligaments are calcified. The vertebræ primarily affected are usually those of the lumbar and lower cervical portions of the spine. Osteophytes are not found. At this later stage the diagnosis becomes obvious as the whole spine is rigid, and the hip and shoulder joints are also sometimes ankylosed. Hysterical contracture of the spine should be differentiated.

Course and Prognosis.—In cases in which the onset is acute, ankylosis of the spine may occur within a few months. The younger the patient the more likely is this to happen. An associated swelling of other joints in the body develops at some period of the disease in about 25 per cent. of all cases, while the hips and shoulder joints are liable to be permanently involved in about the same proportion of cases. Iritis will occur in a small proportion, and is of bad prognosis.

Treatment.—It should be remembered that spondylitis of this type is a systemic disease and thus needs general treatment, as well as more specialised attention to the spine. Treatment will be more successful if it can be started at an early stage of the disease. The general treatment is similar to that advised for rheumatoid arthritis: rest, high caloric and vitamin diet. Vitamin C is particularly important in this malady. Any definite foci of infection should be cautiously removed and anæmia counteracted. Ultra-violet light is a useful general tonic, but gold salts do not appear to be useful in this condition.

The special treatment needed in the active stage includes rest in bed and mobilisation of the chest by means of breathing exercises. Fracture boards should be placed under the mattress, to prevent sagging, and the patient should be as flat as possible, the pillows being removed several times daily to hyperextend the spine. Breathing exercises done in this position

should aim at keeping the chest wall mobile by restricting abdominal breathing. Physical treatment is chiefly of value in the form of radiant heat or infra-red rays to the back, in order to relax spasm and relieve pain. Gentle massage is useful for the same purpose, and later on active movements should be performed under supervision. If there is already some deformity of the spine a plaster cast of the back should be made, and the patient should lie in this night and day to relax completely all spasm. The shell should be altered frequently so as to take advantage of the gradual postural improvement, and when the patient first assumes the upright position he should be fitted with a light spinal brace to relieve the back from all strain.

Recently X-ray treatment has been advocated in the treatment of this disease, but the results so far are variable.

Even with the greatest care it is sometimes impossible to avoid ankylosis of the spine supervening. But if it does occur it is almost always possible to ensure that it does so in the optimum position and thus the patient will ultimately be able to lead his life in an erect posture.

2. RHEUMATOID ARTHRITIC TYPE OF SPONDYLITIS (see p. 1352)

3. OSTEO-ARTHRITIC TYPE OF SPONDYLITIS

Synonyms.—Osteo-arthritis of Spine; Hypertrophic Spondylitis; Degenerative Spondylitis.

Some degree of osteo-arthritis of the spine is said to be demonstrable by means of the X-rays of nearly all those over 50 years of age; but it is rare for these changes to cause symptoms. Its incidence is rather higher in men than women, and it seldom occurs before middle life.

Pathology.—The changes are not inflammatory in nature and are identical with those of osteo-arthritis met with elsewhere in the body. Bony ankylosis does not occur in this type of arthritis, but osteophyte formation is always seen, together with narrowing of the intervertebral spaces. The areas most commonly involved are the cervical and lumbar regions.

Symptoms.—When symptoms are present they may include the following: root pains, of which sciatica is the most frequent example; increasing stiffness of the back, which, however, never becomes completely rigid. Headache and pains in the neck, shoulders, and arms are common, sometimes in conjunction with areas of paræsthesia or anæsthesia in the skin. Pains are generally aggravated by movement of the spine, as the nerve roots are liable to pressure in or around their exit from the spinal foramina.

Diagnosis.—This should be confirmed radiologically. The lesion typical of osteo-arthritis is the osteophyte; while marginal exostosis and shrinking of the vertebral margins, with narrowing of the intervertebral spaces, are also seen. The sacro-iliac joints are generally normal. In all cases the possibility of malignant growths in the spine should be borne in mind, as the symptomatology is the same.

Treatment.—This is similar to that of osteo-arthritis elsewhere in the body, and includes the correction of body posture and flat feet. The frequent application of heat in some form, followed by rest and muscular relaxation, is important. Massage and faradism at a later stage will tone up muscular support of the back. Sometimes it will be necessary to supplement the

support of the back mechanically by a plaster shell or a brace for a time. Deep X-ray treatment will sometimes relieve intractable pain which proves resistant to analgesic drugs, such as aspirin, pyramidon, or codein.

7. STILLS' DISEASE

This disease is believed by some observers to represent true rheumatoid arthritis in childhood. But although the articular changes are of an inflammatory nature their effects are more often confined to the periarticular tissues. Again, even in cases of long standing, it is not unusual to find little or no radiological evidence of destruction at the actual joint surfaces.

Ætiology.—The sexes are affected in about equal proportion, while the age incidence resembles that of rheumatic fever, namely, that the malady is seldom met with before the age of three years and the average age at onset is between six and seven years. The cause remains unknown, although the factors held to be responsible for rheumatoid arthritis are usually invoked to account also for Stills' disease. These are focal infection, such as in the teeth, tonsils, sinuses or bowel; metabolic or endocrine disorder; or, in some cases, unsuspected or attenuated infection with the bacillus tuberculosis. A hæmolytic streptococcus is commonly present in the nasopharynx and other foci. In the majority of cases it is very difficult to assign any one cause. It is certain, however, that once the disease has commenced a cold damp environment will exacerbate it considerably.

Symptoms.—If a careful inquiry of the history of the patient's illness be made it will very often be found that a considerable period of prodromal ill-health preceded the onset of the joint symptoms.

The onset of the joint condition is not infrequently rapid, with pyrexia, and pain and swelling of several joints. This often leads to an initial diagnosis of rheumatic fever, but it is soon found that sodium salicylate has no beneficial effect and that the joint swellings, far from being transient, increase in number and intensity. When the onset is gradual there is little pyrexia but a slowly progressive degree of swelling and limitation in movement of the knees, wrists, elbows, fingers and ankles—usually in that order. Later, the cervical spine and also the hips may become affected and the patient will be completely crippled. The foregoing joints are usually attacked symmetrically, and their appearance is characteristic in that the periarticular swelling renders the joint fusiform in shape. The skin over this swelling is rather stretched and often slightly bluish. The muscles adjacent to the affected joints waste, which further exaggerates the fusiform appearance. In the case of the wrists, ankles and elbows the bony contours are often completely obscured. The affected joints are generally tender on pressure, but usually are not painful except on movement. This leads to further voluntary limitation of movement and so intensifies the muscle wasting already present.

In many cases of this disease the joint swelling and muscular wasting is accompanied by a lymphatic reaction which will show as enlargement of the superficial lymphatic glands, especially those around the elbows and in the axillæ. In about half the cases seen there is also enlargement of the spleen. These changes were present in the cases originally described by Still

in 1897. The enlarged glands are not tender, and are generally discrete and "rubbery." Subcutaneous nodules are sometimes found in addition.

There is generally a secondary anæmia, and quite often patches of light-brown pigmentation on the skin. When the disease is established, the extremities are always cold and clammy, and there is an increase in the temperature before other joints are affected. In long-standing cases normal growth is considerably interfered with.

In the late stages the type of deformity seen in adult rheumatoid arthritis develops. There is flexion of the fingers and ulnar deviation of the hands, and also flexion of the knees and elbows.

The X-ray picture is chiefly remarkable for the advanced degree of the osteoporosis which always occurs. There is often little or no actual joint changes, and osteophytes are never found in this type of disease.

The end result, so far as the joints are concerned, is a fibrous ankylosis, or a fibrosis of the joint capsule, which is sufficiently complete to resist all attempts at movement of the joint.

It is stated that at post-mortem examination diffuse pericardial adhesions and adhesive mediastinitis are often discovered, although unsuspected during life, and evidence of valvular disease of the heart has occasionally been reported.

Prognosis.—Until recently there was considerable doubt as to what was the ultimate fate of these patients. Some authorities held that they recovered while others explained the rarity of the affection seen in Adult Out-patient Departments by assuming that the patients either died or became rapidly bedridden after leaving the children's departments. A recent "follow-up" at Great Ormond Street Hospital showed that the mortality in those under five years of age is about 25 per cent. (due to intercurrent infections); that complete recovery occurred in a small proportion; while in the majority, the disease remains apparently arrested, often for several years at a time, only to resume its ravages at increasing intervals until the patients are entirely crippled and bedridden.

Treatment.—When the presence of an infective focus is established, this should be dealt with at an early stage of the disease. In all cases the child's resistance should be built up by all available means. A nourishing diet, an open-air life and a dry sunny climate are indicated. In addition, cod-liver oil and malt, syrup of iodide of iron, and courses of an arsenic-containing tonic are important.

Vaccine treatment with autogenous very weak vaccine is worthy of trial if a hæmolytic streptococcus is found.

Salicylates have little or no beneficial effect. Non-specific protein therapy is often recommended, but is possibly too drastic and temporary a measure to employ except in the later stages. Recently good results have been reported from small doses of gold salts administered intramuscularly in short courses. But in some cases these salts provoke unfavourable reactions, and so should be used very cautiously. No case should receive a larger dose than 0.1 gm. and a total course of 0.75 gm. should rarely be exceeded. The injections should be given at weekly intervals. At least six weeks should elapse before any subsequent injections are administered, and the onset of toxic nephritis, stomatitis, diarrhoea and dermatitis should be especially looked for.

All swollen joints should be bandaged and lightly splinted, or put into thin plaster-of-paris casts, to avoid the contraction deformities which will otherwise inevitably occur. The child should always sleep in these, and will soon become accustomed to them. They should be removed daily, however, for a short period, during which the joint must be given passive movement, to prevent fixation. Dry heat from a radiant heat or infra-red ray lamp is comfortable and renders the performance of these daily active movements easier. These movements are also essential to remedy the muscular atrophy present. Massage is generally unnecessary in these cases.

In the very late stages, and the patient is bedridden owing to extensive contraction deformity, minor surgical procedures, such as tenotomy, are sometimes justifiable to remedy the deformity.

PSEUDO-ARTHRITIS (JOINT EFFUSIONS)

Effusion of fluid into the joints may be associated with various conditions, and is often of a temporary or intermittent nature. If not followed by damage to the joint surface, it should not be strictly referred to as an arthritis.

Apart from the various forms of arthritis already described, the following may give rise to joint effusion :

(i) *The specific fevers*, especially scarlet fever, meningococcal fever, puerperal fever, influenza, syphilis, subacute bacterial endocarditis, typhoid or paratyphoid fever, measles and malaria. In the United States a form of pseudo-arthritis associated with lympho-granuloma venereum is not uncommon.

(ii) *Abnormal blood conditions*, such as purpura simplex or purpura rheumatica (Schönlein's disease), scurvy and hæmophilia will give rise to swelling of the joints due to an effusion of blood. The knees are most frequently affected.

(iii) *Growths affecting the bones*, in near proximity to a joint. An X-ray examination will elucidate the ætiology in such cases.

(iv) *Injection of animal sera*.

(v) *Trauma* will cause synovitis of the affected joint. It should be noted that the strain imposed on certain joints due to faulty body posture will often result in a chronic form of hydrarthrosis.

(vi) *Intermittent Hydrarthrosis*. A periodic recurrence of joint effusion of unknown ætiology which persists for several days and usually affects the knees. Attacks tend to recur at regular intervals ; they show no local evidence of inflammation ; and they are refractory to most forms of treatment. Some authorities consider this condition is the result of an allergic sensitisation, and advocate treatment directed along these lines.

NON-ARTICULAR RHEUMATISM ; FIBROSITIS

Fibrositis may be defined as a condition in which acute or chronic inflammatory changes involve the fibrous tissues of the body, such as the subcutaneous tissues, the superficial and deep fascia, the muscle sheaths and tendons, the fibrous portions of the joint capsules and ligaments, the bursæ,

and the fibrous sheaths of the nerves. The affection gives rise to pain and impairment of movement. It may be subdivided broadly into three types according to the nature of the structures primarily attacked :

1. Panniculitis : Inflammation of the subcutaneous tissue and fat.
2. Inflammation of the muscle sheath and the fibrous tissue between the muscle fibres, the aponeuroses, the tendons and the superficial and deep fascia.
3. Peri-arthritis : Inflammation affecting principally the fibrous portions of the joint capsules, ligaments and bursæ (bursitis).
4. Peri-neuritis : Inflammation affecting primarily the nerve sheath (perineurium) and the fibrous tissue between the nerve fibres.

Ætiology.—A large group of cases can be traced to the presence of focal infection, and both the teeth and the tonsils are generally suspect. This is perhaps more particularly so in cases in which the upper part of the body, including the arms, are chiefly affected. In this connection it is to be remembered that negative X-ray evidence of apical infection of the teeth should not be accepted as conclusive evidence, since changes which can be demonstrated by this means are necessarily of a chronic and advanced nature. Further foci of infection should be sought in the sinuses. Occasionally the bowel, particularly the colon, may be thought of as a focus of infection, but when considering the question of vaccine treatment the old aphorism that it is “a poor bowel which does not grow something” may be remembered with advantage. Another focus of infection, which is sometimes overlooked, is the prostate, and in the absence of other infection it is worth while to perform prostatic massage and culture the “bead” so obtained.

A further group of cases would appear not to be of a primarily infective nature but to be allied to gout or to a special sensitivity to certain types of food.

Finally, a certain proportion of cases of fibrositis can be traced to chronic strain, often secondary to faulty posture. A common example of this will be found in those cases in which the fascia lata of the thighs is tender and painful in conjunction with a flattened plantar arch. Such patients are usually cured when the distribution of the body weight is readjusted by raising the inner edges of the shoes. Some cases of low backache seem also to be attributable to the same cause. Other causes of this chronic strain will arise out of the occupation, or sometimes the sports of the patient, while any unaccustomed muscular exercise should be inquired into, especially if in conjunction it has been associated with exposure to cold and wet, *e.g.* the frequent occurrence of sciatica in doctors who drive a car in wet clothes for a long period.

Morbid Anatomy.—The morbid anatomy of this affection was investigated originally by Stockman, who found that the results of injury to fibrous tissue, whether bacterial or traumatic, had the effect of producing in the acute stage an exudate and inflammatory œdema. This is followed by organisation of the exudate, and the growth of new fibroblasts and new blood vessels with thickened walls. There is no migration of polymorphonuclear leucocytes and no pus formation, a few lymphocytes alone being attracted to the inflamed area. As the condition becomes chronic there is a production of dense connective tissue in nodules or strands, which differ from normal fibrous tissue in having more fibroblasts, in having arteries

with thickened walls, and in leaving the sheaths of the nerves passing through it in a state of interstitial inflammation. The pain of fibrositis is apparently due to the swelling of the tissue through the inflamed exudate, and subsequently to the involvement of nerve fibres in the new fibrosis, apart from the interstitial inflammation of the nerve twigs themselves.

Symptoms.—It is to be noted that the pain complained of in fibrositis is not always at the real seat of the lesion but may be referred to other areas and therefore careful location of the actual seat of the inflammation is essential. Again, the symptoms will vary according to the area of the body affected. As examples, the predominant effect of involvement of the muscles of the neck will often be headache; of those of the limbs, numbness and tingling; and of the fibrous tissue surrounding joints, stiffness and pain on moving these, which is often wrongly attributed to true arthritis.

Panniculitis is met with most typically in the early stages of "menopausal arthritis" in which tender pads of thickened tissue are found over the internal aspects of the knees, the back of the neck, the extensor surfaces of the arms, the outer aspects of the thighs and elsewhere. It occurs most frequently in stout people, and Dercum's disease or adiposis dolorosa is a progression of this process. In some cases this syndrome appears to be associated with moderate hypothyroidism.

In the case of inflammation of the muscle sheaths and the intramuscular fibrous tissue, the local effect is to keep the muscles in a state of spasm during the acute period. Subsequently the spasm will relax, but localised patches of nodular induration may be palpable in the muscles. Occasionally, also, thin fibrous cords may be felt running through the subcutaneous tissues. These "nodules" will generally, but not invariably, prove tender on palpation. Lumbago is perhaps the most common manifestation. Its onset may often be very acute. It should be distinguished from arthritis or caries of the spine, sacro-iliac disease, perinephric abscess and renal disease, all of which may simulate it. Pleurodynia is a rheumatic inflammation of the intercostal muscles which gives rise to severe pain when the affected muscles are brought into action, as on coughing or deep breathing. Usually local tenderness can be elicited on palpation; but careful examination is needed to exclude such sources of pain as pleurisy or intercostal neuralgia. The muscles of the abdomen are sometimes the site of a local lesion, and this will occasionally simulate intra-abdominal disease. They are also affected in epidemic myalgia (Bornholm disease). The extensor muscles of the thighs, when they are the seat of rheumatic inflammation, give the clinical appearance of sciatica, and this possibility should always be considered before making a diagnosis.

One of the commonest causes of pain, generally diagnosed as brachial neuritis, is the presence of a degenerative lesion in the tendon of the supraspinatus muscle. This lesion will sometimes calcify and may then be seen in an X-ray, if the shoulder be externally rotated. This type of lesion is often the cause of inflammation of the subacromial bursa through which this tendon passes. "Tennis elbow" is the term applied to the painful fibrositis affecting the origin of the extensor tendons of the forearm from the external condyle of the humerus.

Bursitis may occur in any of the large bursæ. The most commonly affected is the subacromial bursa, referred to above. The chief clinical

manifestations of "deltoid bursitis" are great pain on actively abducting the affected arm to an angle of 90° with the body. Above this point abduction can generally be completed without pain; the pain returning, however, at the same point when the arm is again lowered. Passive movement through this range is not painful. Pain of this type will encourage the sufferer to immobilise the affected arm, and this will allow the inflammation to spread to the joint capsule, which will contract, and so ultimately limit, or even entirely prevent, movement taking place in the joint subsequently. The patient is often unaware of the full degree of limitation of the movement which has occurred in such cases, as a considerable degree of movement is possible by virtue of the mobility of the scapula. This condition is the severest type of peri-arthritis, and the apparent ankylosis of the joint must be differentiated from a true arthritis by means of radiology, as peri-arthritis of this type may be cured by the employment of diathermy, gentle manipulation, and remedial exercises. The next most commonly affected bursæ are those over the olecranon process, around the knee joint, over the ischium and over the great trochanter. Inflammation of any of these should be differentiated from a true arthritis of the neighbouring joint. Another form of peri-arthritis depends on inflammation of the tendon sheaths of muscles surrounding joints. This may occur as part of a chronic rheumatic process unassociated with trauma or gout. Its association with gonorrhœal and dysenteric infections has been mentioned. The flexor tendons of the wrists and knees are the most commonly attacked. Pain, swelling or crepitus results and sometimes synovial effusion. The palmar fascia is sometimes the site of a chronic fibrositic process, and the resulting thickening and contracture is known as Dupuytren's contracture. This is seldom painful, but can give rise to considerable disablement of a somewhat intractable nature. The condition, which is commoner in males, is often found to be familial. A somewhat similar condition, which, however, does not cause so much contracture, is known as "painful heel." In some cases a small spur of bone is found radiographically at the insertion of the plantar fascia into the os calcis. But in the majority of cases, no cause for the pain can be found.

In the case of peri-neuritis, the sciatic nerve is the most commonly affected, next in order are the nerves of the brachial plexus, and then the intercostal nerves. Some forms of Bell's "palsy" are thought to be of similar origin. The symptoms in a fully developed case do not differ from those of a true neuritis, but the distinction can generally be made from a history of an initial fibrositis of neighbouring structures which later spreads to the nerve sheath. Since the introduction of radium therapy for carcinoma of the lung, cases of axillary fibrosis, followed by severe and sometimes permanent brachial neuritis, have been seen.

The subjects of fibrositis are usually found to have some degree of defective skin circulation, as evidenced by abnormal sensitivity to cold weather or to local draughts, spontaneous bruising, or the fact that they perspire in the hottest weather only with difficulty. Attacks of fibrositis will occur in many people without apparent detriment to their general health, and in such persons the suspicion should arise that the cause may not be primarily an infective one.

The group of cases which appear to be allied to gout or to a special sensitivity to certain types of food may be suspected by the excellent general

health, even during attacks, the periodic or seasonal nature of the attacks, a history of familial gout or of being "unable to digest" certain foods or drinks, and finally by the fact that the fibrositis tends to affect the lower limbs and other lower parts of the body. Such patients, in addition, often exhibit the symptom-complex described by the French as "hépatisme." This is shown principally by morning headache, furred tongue, and a tendency to incomplete bowel emptying, with light-coloured and offensive stools; often, too, there is slight tenderness on palpation in the neighbourhood of the liver.

Prognosis.—Provided sufficient care be taken and the value of external as well as internal remedies is remembered, the outlook is good. An exception, however, is in the case of very old patients, for the senile form of fibrositis is sometimes intractable to all the usual remedies.

Treatment.—When symptoms do not call for urgent treatment, the first indication is to investigate the ætiology. In the great majority of cases in which the malady is believed to be allied to gout or to a special sensitivity to certain types of food, appropriate treatment should be adopted.

At the outset, a mercurial purgative, such as calomel (gr. $\frac{1}{2}$ –2), followed next morning by a saline, should be prescribed.

In the acute stage rest in bed is desirable. The internal administration of analgesic drugs is indicated. Aspirin and calcium acetylsalicylate (5–15 grs.) are in the majority of cases the most efficient for this purpose. If necessary, potassium iodide (2–5 grs.) may be added, as may phenacetin (5–10 grs.) or caffeine (5 grs.) at four-hourly intervals. A useful addition to aspirin and phenacetin is codeine phosphate ($\frac{1}{4}$ th gr.) or Dover's powder (5–15 grs.). Amidopyrine (3–10 grs.), a centrally acting drug, is sometimes effective when the salicylates fail. It should be borne in mind, however, that if this drug is used there is a danger of agranulocytosis in susceptible individuals and therefore frequent blood counts are required. An ointment designed to act either as a rubefacient or as a counter-irritant should also be prescribed. A hot linseed poultice containing opium will also often give considerable relief if applied every few hours; as may a hot cloth wrung out in a solution of ordinary mustard in water. Massage is undesirable in the acute stage.

In certain cases benefit will result from a short course of colonic lavage twice a week for 2 to 3 weeks.

When it is desired to immobilise the muscles of the back during the acute stage of lumbago, the most effective method is by means of a perforated belladonna plaster, which should be made to cross the mid-line behind and come round to the front. Ordinary wide strapping is a good substitute.

When the condition is less acute and the patient is able to get up, the application of both heat and massage to the affected regions is indicated. The former may be applied in a dry or a moist form, the one often succeeding when the other has failed. Dry heat may be given by means of a portable lamp, an electric heating pad, exposure to a gas-fire (which gives out infra-red rays), the application of a hot iron through brown paper applied to the skin, a hot-water bottle, or a bag of salt or sand which has been heated thoroughly in the oven. When the condition is deep-seated, diathermy will be the best form in which to apply it.

Moist heat may be applied in the form of kaolin, bread or linseed poultices, kaolin poultice (antiphlogistine), mud packs, or applications of hot paraffin wax of a special melting-point (which is sold for this purpose), or, if the patient is in a condition to have such, a Turkish bath. In the course of the last, the patient should be instructed to drink fluids copiously during and after it, as otherwise the temporary concentration of the blood is likely to provoke a further acute attack. Perhaps the simplest method of moist heat is an ordinary bath, to which 4 lb. of Epsom salts (or common salt) have been added. This should be taken as hot as possible, and, contrary to the general belief, the patient should not "soak in it" but should get out after only 5 to 10 minutes' immersion and be briskly rubbed down, after which some analgesic ointment should be rubbed rapidly into the affected areas, and he should be wrapped in a blanket and put to bed for several hours. After this, deep massage should be ordered for the affected areas, although painful at first.

Hydrotherapy and Counter-irritation.—When the patient is near a Spa or an Institution equipped for hydrotherapy, "Vichy" douche massage, followed by "contrast douching" (alternate hot and cold water directed on to the painful areas under pressure from a hose-pipe), is probably the best follow-up treatment; it stimulates the skin to resume its normal function.

In certain cases, particularly when the complaint is that of lumbago, counter-irritation by means of a small cautery may be of great value. A small blister should result from each application, and the whole area may then be covered over with a gauze dressing. Another method is to produce blisters by means of "blistering fluid," or cantharidin plasters, but these are not of such value as the actual cautery. Dry-cupping is a somewhat obsolete method of treatment, but is occasionally effective; as is full exposure to a mercury vapour lamp at a distance of 18 to 24 inches from the affected area.

In the chronic stage massage is essential if an attack is to be terminated in the minimum time and also if recurrence is to be avoided. To be effective, the indurated areas (nodules) should be carefully sought for in the muscles, and at these points the massage should be very deep. It will be found that following deep kneading with the finger-tips or thumbs, after an initial period during which they may be increasingly painful, they will gradually become insensible to palpation, and ultimately disappear. This process will be considerably facilitated if it be preceded on each occasion by 20 to 30 minutes' application of heat in one of the forms mentioned above. In certain cases a "nodule" will prove to be too painful for deep massage treatment, which will then induce protective spasm in the surrounding muscles, and so render further "kneading" impossible. In such cases a dose of aspirin or some other analgesic may usefully be administered before beginning the treatment. When one or more discrete nodules can be felt and when the pain is found to be chiefly localised in these sites, the effect of injecting a few cubic centimetres of a local anæsthetic, such as procaine hydrochloride ($\frac{1}{2}$ per cent. in saline) or "A.B.A." compound, is sometimes dramatic. When there is more diffuse pain and tenderness, this method of treatment is not of much use, and unless the injection is made with great accuracy into the nodule the trouble may even be exacerbated.

Diet and After-care.—If there is obesity, this should be treated (see

p. 438). In cases in which a gouty origin is suspected, this should be corrected (see pp. 435, 436).

After an attack of fibrositis, it is important that the patient should be taught to contract the affected muscles daily by means of appropriate exercises. He should also make a point of obtaining some regular exercise in the open air, even at the cost of rising somewhat earlier in order to walk part of the way to the office. The obese subject must not be allowed to regain his lost weight once the attack recedes. The question of the localisation of attacks, as the result of the occupation or hobbies of those predisposed to suffer, should not be omitted.

MYOSITIS OR INFLAMMATION OF THE VOLUNTARY MUSCLES

Three forms occur—(1) the suppurative type; (2) the non-suppurative type; and (3) myositis ossificans progressiva.

1. SUPPURATIVE MYOSITIS.—In this condition there is a primary inflammation of the affected muscles associated with the local signs of inflammation and the general symptoms of a septic infection. Abscesses form in the affected muscles, which require incision, and in the pus obtained pyogenic organisms, such as staphylococci, or less commonly streptococci, are usually found.

2. NON-SUPPURATIVE MYOSITIS.—It must be remembered that the voluntary muscles are affected in the course of other diseases. Thus, degeneration of the striped muscle, known as Zenker's degeneration, may occur in any acute infection of long duration, and it was first observed in typhoid fever. In scurvy, intra-muscular hæmorrhages are very common, and these are followed by a chronic inflammation, which usually clears up; but in a few of such cases we have seen suppuration occur. Trichinosis is accompanied by a myositis, set up by the encapsulated larvæ of the trichina spiralis deposited in the voluntary muscles.

Dermato-myositis is an acute or subacute inflammation of the muscles of unknown origin, which is associated with dermatitis and œdema. The onset is usually gradual, and ultimately all the muscles of the body may be involved. Pain is an early symptom, and fever of a mild intermittent type occurs. Œdema develops over the affected muscles, and is accompanied by a dermatitis of erythematous or urticarial type. Sweating is common, and enlargement of the spleen usually develops. Owing to involvement of the respiratory muscles broncho-pneumonia is a late complication. The disease is usually progressive, and generally fatal, though some recoveries have been recorded. The treatment adopted has been for the relief of symptoms, and no specific treatment is known at present.

A type of the disease in which hæmorrhages occur in and between the muscles is known as "polymyositis hæmorrhagica."

3. MYOSITIS OSSIFICANS PROGRESSIVA.—This is a progressive inflammatory affection of the locomotor system of unknown origin, characterised by the deposition of bony substance in the fasciæ, muscles, aponeuroses, tendons, ligaments and bones, with resulting ankylosis of most of the articulations. The disease is rare. It usually commences in early life, and is commoner in males. Three stages occur in the muscle changes. In the first stage, swelling

and infiltration of the affected muscle with embryonic connective tissue occurs. In the second stage, the embryonic connective tissue becomes organised and forms ordinary connective tissue, which retracts to a hard fibrous mass. In the third stage, calcification of the fibrous mass occurs, and this becomes replaced by bone.

The muscles of the back and neck are usually the first involved, and the vertebral ligaments become ossified, so that irregular bony swelling occurs and deformity and fixation of the spine result. The upper and lower limb are later involved, the muscles contracting and causing fixation of the joints. The muscles of mastication become finally involved and prevent movement of the lower jaw. Ultimately the patient becomes helpless and bedridden, and usually dies from some intercurrent affection, such as pneumonia, or pyæmia resulting from bedsores. The disease is always progressive, but is usually of long duration, and there may be a cessation in its progress for several years. No specific treatment of value is known.

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SECTION XVIII

DISEASES OF THE SKELETON

Diseases of the skeleton will be considered under three headings, according to whether bone, endochondral ossification, or bone marrow is primarily affected.

DISEASES OF BONE

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Synonyms.—Hippocratic Fingers ; Marie's Disease ; Acropachy.

Definition.—A symmetrical enlargement of the bones of the hands and feet, and of the distal ends of the long bones, accompanied by clubbing of the fingers and toes, occurring in association with certain chronic diseases, especially of the lungs.

Ætiology.—The primary diseases in the course of which hypertrophic osteo-arthritis may develop are :

1. Diseases of the lungs, such as (a) chronic cavitating tuberculosis and fibroid phthisis ; (b) empyema and bronchiectasis ; (c) malignant disease of the lung, pleura or mediastinum ; and (d) fibrosis of the lung, non-tuberculous in nature.

2. Congenital heart disease, and infective endocarditis.

3. Chronic diseases such as dysentery, pyelonephritis, alcoholism, Raynaud's disease, and jaundice, as in hypertrophic cirrhosis of the liver.

4. Rarely a neuritis may lead to clubbing of the fingers and osteo-arthritis. Pressure on the brachial plexus by a subclavian aneurysm has given rise to clubbing of the fingers on the affected side.

It appears that in the majority of cases a chronic infection leads to the development of osteo-arthritis. This is the case in the lung diseases above mentioned. In congenital heart disease, the circulatory defect itself will lead to marked clubbing of the fingers ; in this condition, however, the long bones are not appreciably affected, and the changes are limited to the soft parts of the terminal phalanges, the hypertrophy of which leads to clubbing.

The disease is eight times more common in males than females. All ages may be affected. The most striking examples are seen from 30 to 50 ; but in congenital heart disease and in chronic lung disease in children, such as bronchiectasis and fibroid phthisis, signs of the disease often appear in early life.

Pathology.—The bones most frequently affected are the metacarpal bones and the first two rows of phalanges. The radius and ulna may be

affected, and more rarely the lower end of the humerus and the scapula. In the lower extremities the corresponding bones are affected. X-ray examination shows a thin layer of newly formed bone spread over the shaft. The periosteum is raised unevenly, so that the outline appears serrated and the deposits beneath it are unevenly calcified giving a lace-work effect. The bony changes are the result of a chronic inflammation, and the thickening of the periosteum and new formation of bone beneath it may be accompanied by atrophy and rarefaction of the pre-existing bone. There are no bony changes in the terminal phalanges, the soft tissues and nails alone being affected.

Symptoms.—The onset is usually gradual, and little local pain is experienced, though stiffness and clumsiness of movements occur. Sometimes marked clubbing of the fingers develops in a few weeks; but usually several months or more elapse before the condition is characteristic. There is a remarkable symmetry in the pathological changes. The ends of the fingers and toes may be cyanosed. The nails are large, broad and curved, both longitudinally and transversely—the so-called parrot-beak. They show longitudinal striation and are brittle and easily split. The root of the nail is raised above its bed, and if pressure is applied at the root a distinct space between them can be made out. Sometimes the joints in the neighbourhood of the affected bones show swelling, from effusion and thickening of the synovial membrane. Osteo-arthritis changes in the joints are only present in the severe osteo-arthritis type.

Three types of cases are seen, but these may be only grades in the development of the extreme form of the disease.

1. *Cases showing only clubbing of the fingers, in addition to the signs of the general primary disease.*—This symptom may disappear if the primary disease is cured, as, for example, empyema.

2. *Cases showing clubbing of the fingers and painful thickening of the bones of the hands and feet, forearms and legs, in addition to symptoms of the primary disease.*

3. *The "osteoarthritis hypertrophica" type.*—The hands and feet become greatly enlarged, owing to the bony changes and thickening of the soft parts. The forearms and legs are thickened. The pelvis, sternum, ribs and clavicles may be thickened, and the vertebræ may show changes resulting in kyphosis. Osteo-arthritis occurs in the parts involved, so that movement of the joints is painful and difficult. In this type of case the very remarkable changes in the bones and joints overshadow the symptoms of the primary disease.

Diagnosis.—The disease is recognised by the presence of the characteristic changes in the extremities, and by the presence of signs of one of the primary diseases already mentioned.

Infective arthritis is distinguished by the absence of clubbing of the fingers, and by the characteristic changes shown by X-ray examination.

Acromegaly is to be distinguished by the spade-like hand, the spatulate fingers, enlarged knuckles, and the characteristic facial appearance. The kyphosis is more often cervico-dorsal, whereas in hypertrophic pulmonary osteo-arthropathy it is more often dorso-lumbar.

Osteitis deformans shows irregular enlargement of the bones but there is a good deal of bowing, the hands are normal, and the X-ray appearances are pathognomonic.

Prognosis.—The prognosis appears to depend on the primary disease. If that can be arrested or cured, there is hope of arrest or improvement in the hypertrophic osteo-arthritis.

Treatment.—This should be directed towards the cure or improvement of the primary disease. Other treatment is symptomatic and similar to that adopted in the treatment of infective arthritis.

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OSTEITIS DEFORMANS

Synonym.—Paget's Disease of Bone.

Definition.—A chronic and somewhat rare disorder causing enlargement and deformity of many bones. It is not a generalised disease of the skeleton. The bones are affected in the following order of frequency: pelvis, spine, femur, tibia, skull, fibula, clavicle, humerus, radius, and rib. In a few cases the disease is confined to one bone or to part of one bone: tibia, femur, clavicle, a vertebra, the ilium, or half the pelvis.

Ætiology.—This is unknown. The disease is sometimes familial. It rarely begins before the age of 40, and the commonest age of onset is 55. The sexes are affected in the proportion of three men to two women. Osteitis deformans is not inflammatory in origin. It seems likely that it is a disorder of mineral metabolism. Syphilis is not an ætiological factor. No alteration in the parathyroid glands nor in any other endocrine gland has been demonstrated. Both histological and chemical investigations have proved beyond doubt that generalised osteitis fibrosa (hyperparathyroidism) is unrelated to osteitis deformans.

Pathology.—There is a great alteration in the architecture of the bones affected. They become enlarged, irregularly thickened, and sometimes bowed. The skull is very thick, the sutures and foramina being narrowed in consequence. The cortex of the long bones ceases to be pure ivory bone but looks coarse and spongy with red streaks and dots. Histologically there is continuous excessive resorption of bone associated with an increased new bone formation that more than compensates for the bone lost. The excessive erosion disturbs the skeletal architecture, the compact bone being replaced by irregular angular trabeculae, which also form the cancellous bone. There is still an attempt at structural adaptation to stresses, but this is very imperfectly achieved because the material is not used to the best mechanical advantage.

Biochemistry.—The serum calcium and plasma phosphorus are normal. The plasma phosphatase is constantly high, as in many other diseases of bone. In more than 80 per cent. of cases the calcium output in the urine is increased and sometimes reaches four or five times the normal figure. There seems to be a complete absence of correlation between the length of history, the density of bone shadows in radiographs, and the calcium output. A case showing increased density of bone trabeculae throughout pelvis, lumbar spine, and femora is just as likely to reveal a high output of calcium in the urine as a low output.

Symptoms.—The disease may remain symptomless for ten years or more. It is very slow in progress and rarely influences the general health, giving rise in most cases to few symptoms other than those which are due

to changes in the shape of the bones. In 80 per cent. of cases there is pain, and the patient usually recognises its origin in the bones. It varies widely in severity from a dull ache to a severe shooting or stabbing like a knife. The back and lower limbs are the parts usually affected but headache is fairly common. When the skull is involved the patient may have to take a larger size in hats. The enlargement in the circumference of the head leads to the forehead being prominent and the face small in proportion. In the later stages the head is held forward and the back is so bent that the arms appear too long and an ape-like attitude results. There may be considerable reduction in total height. The lower limbs especially are bowed, the knees being widely separated and held slightly flexed. The bones are enlarged, and bowing usually takes place in such a manner as to accentuate the normal curve of the bone. The enlargement is particularly noticeable in the case of the tibia. The changes in the vertebrae may cause encroachment on the spinal canal, resulting in compression paraplegia. Bony compression of the optic nerve may lead to optic atrophy, and of the oculomotor nerves to diplopia. Otosclerotic deafness is common in advanced cases. Spontaneous fracture is rare but when it takes place there is no delay in union. Osteogenic sarcoma may occur, but is much less common than Paget thought and is not seen until the changes in the bones have been present for ten years or more. Osteo-arthritis of the hip, knee, ankle, or spine is an occasional complication. Arterial degeneration, sometimes with hypertension, is found in most cases over the age of 50. It is possible that the excess of phosphoric esterase in the blood accelerates and intensifies the deposition of calcium salts in degenerate vessels. Retinal arteriosclerosis is a frequent finding, and it may be associated both with retinal hæmorrhages and extensive choroidal changes.

Radiological appearances.—The altered bone appears in radiographs in two forms, which may be called the spongy and the amorphous, the former being the more common. The two types are often found in the same patient. The spongy form consists of coarse irregular striæ arranged either as parallel trabeculae or running in the direction of normal lamellæ of cancellous bone. The amorphous form is a generalised deposit producing an opaque finely granular appearance. The diameter of the bone is increased, sometimes to a marked degree, and in the medullary cavity the trabeculae are accentuated and too widely separated, giving a streaky appearance. The corticalis is partly or entirely replaced by bone similar to that seen in the medullary cavity, and in an extreme case the impression is that the whole bone consists of cancellous tissue highly magnified. Irregular cyst-like areas are sometimes observed. Widening and bowing of bones are important points in the radiological diagnosis. The vault of the skull is thickened, and the differentiation between the inner and outer tables is lost. Small islands of dense bone are evident alongside pale, cyst-like areas. A large clean cut area called *osteoporosis circumscripta* may sometimes be noted. In those cases in which part of one bone is affected there is a definite line of demarcation where the abnormal ends and the normal begins. Thus there may be definite changes in the upper two thirds of the tibia, while the lower third is normal. The average rate of progress of such a lesion is about 1 cm. in two years. Radiographs reveal the shadows of arterial calcification in more than 40 per cent. of cases. Such calcified arteries are best seen in the lower limbs. There

is no evidence of a higher incidence of renal or vesical calculus in osteitis deformans than in the normal.

Diagnosis.—When advanced the condition is unmistakable. In the early stages muscular rheumatism or osteo-arthritis may be wrongly diagnosed. Pulmonary osteo-arthropathy is distinguished by the clubbed fingers. In radiographs the amorphous type of osteitis deformans is sometimes mistaken for secondary carcinomatosis of the osteoplastic type. The difference is distinct and important, namely, that in carcinomatosis the bones are neither enlarged nor bowed. Syphilis of bones is now very rare, but when only one or two bones are involved in a supposed case of Paget's disease the Wassermann reaction should be performed.

Prognosis.—Because the disease is uncommon there is a tendency to regard its effects as dreadful. To announce the diagnosis as though it were a profound mystery may alarm both patient and relatives unnecessarily. Paget's disease is slowly progressive but does not usually shorten life. Thus, one patient though much deformed, continued to drive a crane in a dock-yard 15 years after the onset of the disease. Another was quite happy to have somebody hold him on a rock while he fished a stream, long after he was unable to walk unaided. Death usually results from the effects of arteriosclerosis or intercurrent infection, and only rarely from compression paraplegia or sarcoma of bone.

Treatment.—No known treatment alters the course of osteitis deformans in the slightest degree. Since the bones at one stage are sufficiently decalcified to bend, methods have been used which aim at increasing the calcium intake. The patient is given a high calcium diet, that is a diet containing three pints of milk or milk products daily, together with butter, cheese, and eggs. If milk is not tolerated in these quantities calcium caseinate or calcium lactate (10 grammes a day) may be prescribed. Vitamin D may be conveniently given in the form of tab. calciferol. (3000 units) one or two daily. The claim that prolonged exposure to general ultra-violet irradiation has resulted in increased density of the shadows of bones in radiographs has not been confirmed. Such treatment can be carried out, starting with short exposures to the mercury vapour or carbon arc lamp. Paget treated his patients with potassium iodide, but was not enthusiastic over the results. When there is pain in the bones Lugol's solution (of iodine in potassium iodide) may be given in milk, beginning with a dose of three minims three times a day, and increasing to ten times this amount. If iodine fails to relieve the pain, aspirin, amidopyrine, or allonal should be tried. Exploration of the neck for a parathyroid tumour is never justified. Osteotomy is rarely necessary, but it is interesting that when portions of bone have been removed for histological section relief of pain has sometimes occurred. Occasionally, and especially in those cases with secondary osteo-arthritis of the hip joint or knee joint, an ambulatory splint supporting the weight of the body on the tuber ischii is of value. A cork sole is often necessary, and when kyphosis causes pain a spinal jacket is useful.

LEONTIASIS OSSEA

Synonyms.—General Hyperostosis of the Skull; Cranio-sclerosis; Megaloccephaly.

Definition.—The term *leontiasis ossea* is now used in two senses, specifically for a progressive sclerosing hyperostosis of the skull, and symptomatically when osteitis deformans and the various types of osteitis fibrosa happen to involve the bones of the calvaria and face.

Ætiology.—This is unknown. The fact that the disease commonly arises in the region of the nasal sinuses has led to an erroneous view that it is infective in origin.

Pathology.—When Virchow suggested the use of the term "*leontiasis ossea*" in cases of hyperostosis of the skull he had in mind fibroma molluscum in which masses of new connective tissue develop in the skin. He believed that the overgrowth of bone in hyperostosis corresponded exactly to elephantiasis of the soft parts, and he decided to call these cases *leontiasis ossea*, not because the bone disease produced a leonine appearance, but because he considered it to be analogous to the disease of the soft parts which did. The disease is very rare. It occurs in either sex, arising usually in early adult life. In most instances it begins in the nasal fossæ and sinuses, though in some cases the origin is near the orbit or in the base of the skull. Dense ivory bone appears and spreads slowly under the periosteum, being held up sometimes in the region of the suture lines but ultimately breaking through and spreading in many directions across the skull. The serum calcium and plasma phosphorus are normal.

Symptoms.—The early clinical features include nasal obstruction, blocking of the lachrymal ducts, and alteration in the shape of the face and jaws. Ultimately large masses of bone, increasing in various directions, give rise to terrible disfigurement. The cavities of the mouth, nose, and orbit may be greatly lessened. The eyeballs may protrude even beyond the lids, and blindness may occur from optic atrophy. There may be loss of the sense of smell, and interference with the mobility of the lower jaw. Except in the later stages pain is unusual.

Diagnosis.—Paget's disease usually begins at 55, and the pelvis, spine, and lower limbs are nearly always affected. Generalised osteitis fibrosa leads to decalcification of the whole skeleton, with a high blood calcium and low blood phosphorus. Focal osteitis fibrosa often shows multiple lesions scattered throughout the skeleton.

Treatment.—No treatment has any permanent effect though it may be possible to remove some of the more disfiguring masses of bone.

HYPERPARATHYROIDISM (GENERALISED OSTEITIS FIBROSA CYSTICA (see p. 498)

FOCAL OSTEITIS FIBROSA

Synonyms.—Osteitis Fibrosa Circumscripta (Schmidt); Local Fibrocystic Disease; Benign Giant-celled Tumour; Osteoclastoma; Osteogenetic Myeloma; Myeloid Sarcoma.

Definition.—A focal or multifocal disease of bone unassociated with constitutional symptoms or with any known endocrine disturbance.

Ætiology.—This is unknown. The disease occurs chiefly in adolescence,

and is much more common than is generalised osteitis fibrosa (hyperparathyroidism).

Pathology.—The lesions are benign, firm, grey or brown tumours. Histologically they show osteogenetic fibrous tissue and giant cells which, of course, are osteoclasts. This explains the numerous synonyms which are used. The tumours sometimes expand the corticalis and may give rise to cysts lined by osteoclasts. Even when the lesions are multiple the rest of the skeleton consists of normal bone. The figures for serum calcium and plasma phosphorus are invariably normal, a finding in striking contrast to that of the generalised disease. The calcium balance is usually normal, and, taken in conjunction with the normal blood chemistry, this finding is strong evidence against hyperparathyroidism.

Symptoms.—The malady affects one or more bones, is usually not disabling, is of slow progress, and shows a tendency to become arrested. Pain is unusual and the disease is often symptomless until spontaneous fracture occurs. Severe cases of the multifocal type may show considerable deformity, especially of the pelvis, femora and skull.

Radiological appearances.—In radiographs the principal changes are found in the ends of the long bones. Usually more than one-third of the shaft is affected by a fusiform enlargement composed of a pale cyst-like area divided by a few coarse trabecular strands. The cortex is thin and may be expanded. The periosteum and adjacent bone are normal. Radiographs taken with controls show that the whole skeleton apart from the lesions is normally calcified. The floor of the skull and the lower jaw may be affected.

Diagnosis.—The normal blood chemistry serves to distinguish the focal from the generalised disease. In adult cases it is sometimes difficult to differentiate between focal osteitis fibrosa and osteitis deformans, and it may then be necessary to follow the progress of the condition over a period of time before a definite conclusion is reached.

Treatment.—Fractures are treated in the usual way. If spontaneous fracture occurs in a long bone through one of the lesions, union is usually strong, and radiographs subsequently show that the pale cyst-like area of osteitis fibrosa becomes filled with bone. Exploration of the neck for a parathyroid tumour is quite unjustified.

THYROTOXIC OSTEOPOROSIS (see Hyperthyroidism, p. 488)

OSTEOMALACIA

Synonym.—Mollities Ossium ; Adult Rickets.

Definition.—A generalised disease of the skeleton due to vitamin D deficiency. Two types are found. The first is due to a diet deficient in vitamin D and calcium salts and may be referred to as dietetic osteomalacia. The second is a conditioned dietary deficiency disease, arising from deficient absorption of vitamin D and calcium salts ; it is seen in idiopathic steatorrhœa.

Ætiology.—Osteomalacia is rare in England. It is endemic over wide areas in Northern India, Japan and Northern China, and occurs sporadically in the Rhine Valley, Danube Valley, Vienna, and certain parts of Italy, Switzerland, Flanders and the Balkans. Heredity plays no part. The

disease pre-eminently affects women, and is likely to recur earlier and with greater severity with each successive pregnancy. But it is a mistake to suppose that pregnancy is essential in the aetiology. The malady is sometimes seen at puberty and is quite well known to occur, though rarely, in boys and men. In the majority of cases the symptoms begin between the twentieth and thirtieth year.

Pathology.—Rickets and osteomalacia are essentially identical. What difference exists is merely that of age incidence. Osteomalacia is adult rickets. Morbid anatomists agree that in rickets and osteomalacia the essential abnormality is a deficient calcification of osteoid tissue. This deficiency is generalised throughout the skeleton. The broad osteoid seams in both diseases are due to deficiency of the calcifying mechanism, which should convert osteoid tissue into true bone. In osteomalacia the bones throughout the skeleton are so soft that they readily bend and cut with a knife like rotten wood. Spontaneous fractures are common. The blood chemistry is comparable in experimental rickets of rats, in children with rickets, and in women with osteomalacia. The plasma phosphorus and sometimes also the serum calcium are diminished. The occurrence of foetal rickets has been proved in babies born of osteomalacic mothers.

Symptoms.—Pain is a prominent symptom. It occurs especially in the back and thighs, is aching in character and is worse in the winter months. The pelvis, thorax, or long bones exhibit deformity in a haphazard way; one woman suffers in the pelvis, another in the ribs, and a third in both. Besides the changes in the pelvis, marked deformities occur in the chest and spine. Severe kypho-scoliosis may reduce the height by several inches and cause the head and neck to sink downwards and forwards on to the chest. Deformities of the sternum and ribs give rise to marked prominences and depressions in the chest wall. Coxa vara and irregularly curved long bones are less common. The bones are soft and flexible, rather than fragile, so that bending is much more common than is spontaneous fracture, though both are well recognised. The patient develops a characteristic waddling gait, and muscular weakness may add to her incapacity. In many cases the pelvic deformities interfere with marital relations or with labour, Cæsarean section frequently being necessary. Tetany is common. The teeth are normal. The course of the disease may be fairly rapid, lasting several months, but untreated cases extend over many years. The patient then becomes bedridden, spontaneous fractures, anæmia, cachexia, and bedsores adding to her discomfort and to the difficulties of nursing.

Radiological appearances.—The degree of lack of calcification in radiographs will vary with the severity of the disease, and it is therefore important to take radiographs with controls. In the slight cases the bones of the patient will be slightly more translucent than those of the control. The cortex will be less dense than normal but the bone pattern, especially the trabeculation, will be accentuated by contrast. In the severe examples there will be little or no difference between the density of the bone and surrounding soft tissues, and the cortex will appear as a mere pencilled outline. The bone pattern will have disappeared, the long bones will bend, and occasionally show fracture. All deformities apart from fracture are the result of weight stress or muscular action. The pelvis is tri-lobate, owing to the thrusts of the heads of the femora and sacrum. Lordosis is marked and kyphosis may

be present. In severe cases the chest and ribs are usually deformed. The vertebræ are biconcave, having the appearance of fish vertebræ. In severe cases the vault of the skull may show numerous areas of uneven translucence, varying in size and shape but all fairly clean cut. The spontaneous fractures are usually subperiosteal, and radiographs sometimes show pseudo-fractures. These appear as areas of complete translucence, running across the bone, the edges being quite clean cut, and separated from each other by one or two millimetres.

Diagnosis.—The occurrence of pregnancy and the examination consequent upon this lead commonly to the recognition of the pelvic deformity and of the disease which has given rise to it. Differential diagnosis from other generalised diseases of the skeleton usually produces no difficulty. In hyperparathyroidism there is a high serum calcium, a low plasma phosphorus, and an increased calcium excretion in the urine. In senile osteoporosis the patient suffers from kyphosis and a tendency to fractures particularly of the neck of the femur, and the blood chemistry is normal. In thyrotoxic osteoporosis, the usual signs of hyperthyroidism are present, and the blood chemistry is normal. In myelomatosis the Bence Jones protein is found in the urine in 75 per cent. of cases, the serum globulin is usually increased, and the albumin:globulin ratio diminished. The serum calcium is usually normal, but sometimes raised. The plasma phosphorus is normal, but it rises in cases showing renal insufficiency. In radiographs the condition may closely resemble osteomalacia.

Treatment.—Pure vitamin D is called calciferol because of its power to induce calcification in tissues, especially in osteoid tissue. It is 300,000 times as potent as cod-liver oil, weight for weight. The good effects not only of calciferol but also of cod-liver oil and ultra-violet irradiation have been noted both clinically and chemically, since they are capable of raising the serum calcium to normal. In cases where tetany is present calcium salts should be administered in addition. The diet of a woman suffering from osteomalacia should contain 3 pints of milk a day, with plenty of milk puddings, eggs, butter, cheese, green vegetables, and even nuts and raisins. The dose of cod-liver oil should be large, up to 2 or 4 oz. daily. This treatment relieves the pain in 3 to 4 weeks. Some cases are refractory, and it is then necessary to add 0.5 mg. of calciferol to the cod-liver oil daily. Tetany is rapidly removed by treatment with cod-liver oil and calcium lactate. A powder containing at least 10 grammes of the latter should be used daily, and is best administered fasting with a glass of milk. The patient should be exposed to sunlight when this is possible; otherwise treatment by ultra-violet irradiation may be used, starting with a short exposure to a carbon arc lamp and increasing gradually up to 30 minutes. There is no evidence that phosphorus is of any value in the treatment of osteomalacia. Where the disease exists in great endemic areas, questions of diet, and social and religious customs are proving very difficult. In large areas of China and India the diet is often deficient in quantity, and inadequate in calcium and vitamin D. In the high mountain valleys of these countries and in areas of India where purdah is practised, darkness adds to the danger by causing further deprivation of vitamin D. With regard to China, Maxwell states: "We want flocks and herds, milk and meat, with security of life and property." The suggestion has been made that it might be practicable

in India and China to dispense calciferol freely at a low price just as quinine is dispensed in malarial districts. The relation of ovarian function to calcium metabolism has not yet been settled. Osteomalacia gets worse during lactation, no doubt because of the great drain of calcium from the body. Improvement has been observed after ovariectomy. This operation may act merely by preventing pregnancy, and it is presumably just as reasonable to ligate the Fallopian tubes. When pelvic deformity demands it Cæsarean section is necessary.

OSTEOMALACIA IN IDIOPATHIC STEATORRHOEA

When osteomalacia occurs in the course of idiopathic steatorrhœa (Gee's disease), the following features may be present: fatty stools, dilatation of the colon, tetany, anæmia, skin lesions, and infantilism (see p. 476). The disease occurs in both sexes and the history nearly always goes back to early childhood. The symptoms develop in spite of an adequate diet. We must, therefore, suppose that there is some disturbance of gastro-intestinal function resulting in deficient production, absorption or utilisation of one or more essential factors. The serum calcium is low and the plasma phosphorus is low or normal. The total fat in the stools may reach 40 per cent, or more, and the bulk of this is unsplit fat. The clinical and radiological features are exactly the same as in dietetic osteomalacia. An opaque enema will reveal dilatation of the colon. In treatment the fat in the diet must be cut down to a minimum, and the calcium salts and vitamins kept high. Vitamin D must be given in a solid and not in an oily medium. The prognosis of this type of osteomalacia is good especially in young people. Splinting or even osteotomy may be necessary to correct deformities such as genu valgum. The pelvic deformity may necessitate Cæsarean section.

OSTEOGENESIS IMPERFECTA

Synonyms.—Fragilitas Ossium Congenita; Osteoporosis Congenita; Congenital Osteopsathyrosis; Osteopsathyrosis Idiopathica.

Definition.—A generalised disease of the skeleton, congenital, and in some 25 per cent. of cases hereditary, in which the bones are so fragile that repeated fractures occur. Multiple fractures may occur in utero (pre-natal type of Vrolik, 1849), or fractures may not occur until after birth (post-natal type of Lobstein, 1833). Both sexes are affected equally.

Ætiology.—This is unknown.

Pathology.—In both types the basic defect appears to be defective osteoblastic activity. The cortex of the bones may be scarcely thicker than paper, and the trabeculæ of spongy bone are extremely thin. In the pre-natal type many fractures are seen; in some cases practically every bone in the body has been fractured. The older fractures exhibit good callus formation. In extreme cases, especially in the pre-natal type, the cranial ossification is so disorganised that the vault of the skull consists of a mosaic of small Wormian bones. Congenital hypoplasia occurs in other mesenchymal tissues, notably the ligaments and the sclerotics. There is no evidence whatever of vitamin deficiency. No abnormality in the serum calcium,

plasma phosphorus, or calcium output has been demonstrated. The plasma phosphatase tends to show a raised value but this is not constant.

Symptoms.—The general health of the patient is good but fractures occur from the most trivial violence or even normal muscle action. In the course of time, 20, 30, or even 100 spontaneous fractures may occur. They are often subperiosteal and cause little pain. The patient tends to be short in stature and slender in build. As a result of anomalous cranial ossification, the shape of the head is often striking. A bitemporal protuberance so marked as to turn the ears outwards is frequently observed, but protuberances in the occipital and frontal regions are also seen. Every bone in the body may be deformed. The limbs are often bowed and of unequal length. Kypho-scoliosis, distortion of the ribs and sternum, and asymmetry of the pelvis all occur. Three other defects are commonly found in association with the fragile bones, namely, leaden blue sclerotics, a tendency to dislocation of joints, and after the age of 20 years otosclerotic deafness. Amongst the adult population affected with blue sclerotics approximately 60 per cent. have an associated liability to fracture, approximately 60 per cent. an associated otosclerosis, and 44 per cent. suffer from all three defects. Osteogenesis imperfecta sometimes occurs in an hereditary form without blue sclerotics.

Diagnosis.—Severe cases and all those with blue sclerotics are unmistakable. In the new born great shortening of the limbs may suggest achondroplasia, but the skull is quite different. Cases of spontaneous fracture in the adult occurring in hyperparathyroidism, hyperthyroidism, myelomatosis, osteoclastic carcinomatosis, and neuropathic atrophy of bones really cause no difficulty.

Prognosis.—Severe cases of the pre-natal type are either stillborn or live only for a short time. In post-natal cases the condition proves more severe the earlier the first fracture appears. Multiple fractures in the first few years of life may lead to such deformities that the patient can never walk and may die before puberty. In those who survive, the liability to fractures tends to become less before puberty. In general the longer the patient lives the greater will be the improvement, and in many of the adult cases the disability is slight only.

Treatment.—The utmost care must be taken to avoid the occurrence of fractures. Treatment consists in gentle handling and careful splinting. Union usually occurs without delay and is firm. Dislocations are reduced without difficulty. Vitamin D, calcium salts, and a high calcium diet have no effect on the course of the illness.

OXYCEPHALY

Synonyms.—Tower Skull; Steeple Head; Sugar Loaf Head; Acrocephaly; Craniostenosis.

Definition.—A congenital deformity of the skull due to premature synostosis of the cranial sutures. The skull is short from front to back and its vertical diameter is increased. Allied forms of craniostenosis are scaphocephaly, the boat-shaped head, and plagiocephaly, the obliquely flattened head.

Ætiology.—This is unknown. The disease is more common in males than females. It is sometimes hereditary and familial. It is usually present at birth but it may develop subsequently up to the age of six.

Symptoms.—In its slightest form it attracts attention, while in its grosser forms there is no passer-by but is shocked by the disfigurement and repelled by its hideousness. The forehead is much increased in height, sloping gradually upwards to the vertex with feebly marked superciliary arches. The vertex of the skull appears pointed instead of flattened or rounded, and a thin bony prominence is sometimes felt in the region of the bregma. The hairy scalp may be raised above the normal level and present the appearance of being perched on the top of a cone. Viewed laterally, the ears appear placed on a lower level than normal. Proptosis is present in most cases, and it may be so considerable that the eyeballs become dislocated in front of the lids. Failure of closure of the eyes, especially during sleep, may lead to lachrymation and conjunctivitis. Divergent squint is common and nystagmus is present in some cases. Symptoms arise from insufficient room within the skull for the developing brain. There is increased intracranial pressure with headache and sometimes vertigo. The condition is compatible with normal intelligence, but not infrequently optic atrophy supervenes. This is secondary to papilloedema in some 85 per cent. of cases. In the remainder it is brought about by narrowing of the optic foramen and is of the primary type. The sense of smell is often completely lost, but taste is affected very rarely. Hearing is unaffected. The following associated congenital malformations have been described in a few cases: webbing of the fingers and toes; malformation of ears, elbow and shoulder joints, and fingers.

Radiological appearances.—Radiographs show an increased vertical diameter of the skull with its highest point either at the bregma or somewhere between it and the lambda. The anterior fontanelle closes late, and its site is marked by a slight protuberance over which the bone is thinned. The sutures of the vault are partly or entirely absent, but the basal suture between the sphenoid and the occipital bone may be widely open. The air sinuses are rudimentary, and the middle fossa bulges forward. The most characteristic feature is the presence of numerous deep convolutional markings.

Prognosis.—The optic atrophy, whether primary or secondary, may advance to complete blindness. There is nothing to show that oxycephaly shortens life.

Treatment.—Anodynes should be used in the relief of headache. If the symptoms of increased intracranial pressure become marked, and the changes in the optic discs progress, decompression may be necessary.

DISEASES OF ENDOCHONDRAL OSSIFICATION

ACHONDROPLASIA

Synonyms.—Chondrodystrophia foetalis (Kaufmann); Micromelia foetalis.

Definition.—A disease of foetal life in which defective endochondral

ossification makes the bones preformed in cartilage short, but stout and strong.

Ætiology.—This is unknown. Both sexes are affected equally. It is hereditary and has been recorded in six generations. Several members of the same family may be affected. The condition is unrelated to rickets, cretinism, syphilis, or tuberculosis.

Pathology.—The essential abnormality is found in the cartilaginous epiphyses. The cartilage does not prepare itself for ossification, which is in consequence so slow that the long bones are too short. Since, however, the periosteum goes on laying down bone normally, the bones are stout and strong. The membrane bones of the skull are unaffected, so that the calvaria is of normal size. Premature synostosis of the cartilaginous bones at the base of the skull leads to shortening, and consequent depression of the bridge of the nose. The clavicles are not affected. The pelvis is distorted and contracted, the sacrum being tilted forwards. Extreme lordosis may be present. The costo-chondral junctions are enlarged to form a rosary. The scapula is so small that the glenoid fossa scarcely holds the head of the humerus.

Symptoms.—The patient is dwarfed but of normal intelligence. The usual height of the adult is about four feet. The vault of the head is large and the frontal and parietal eminences prominent. The face is small and the nose has a depressed and flattened bridge. The nostrils are large, the lips thick, and the lower jaw and chin well developed. The teeth are normal. The trunk is of normal size but the extremities are much shortened, and with the arms at the sides the fingers reach no farther than the great trochanter of the femur. The humerus and femur are relatively more shortened than the other bones of the extremities, so that the proximal segments of the limbs show the most marked shortening. The arms are muscular and are held a little abducted from the trunk. The hands are short, thick, and trident-shaped, the fingers being almost equal in length. The lower limbs are thick and often show deep transverse furrows as if there were redundancy of the soft parts. This appearance is due to the packing of well-developed muscles into the restricted long axis of the limb. This muscular development enables the achondroplastic to perform feats which are surprising in one so small. He rises from the lying-down position by a characteristic springing movement from the legs without any assistance from the arms. The curving and enlargement of the ends of certain bones gives rise to bow legs and beading of the ribs. The lumbar curve is increased owing to tilting forward of the sacrum and excessive development of the buttocks. In consequence the gait has a peculiar duck-like waddling character. The genital organs are normal. The fact that the female may become pregnant makes the pelvic deformity of great importance. The conjugate diameter is greatly narrowed, and it is almost impossible for an achondroplastic woman to give birth to a living child except by Cæsarean section. That the disease has existed for something like five thousand years is known from models found in mummies of two achondroplastic gods of ancient Egypt, namely Ptah-Sokar and Bes. In the Middle Ages the attractive antics of achondroplastics made them much sought after as court jesters or dwarfs. To-day not infrequently they play the parts of clowns at fairs, circuses and music-halls, and sometimes break chains on the stage.

Diagnosis.—In the new born the great shortening of the limbs may suggest osteogenesis imperfecta, but the skull is quite different. In childhood the malady is readily distinguished from rickets and congenital syphilis by careful attention to the physical signs. Achondroplasia differs from cretinism in that the patient is of average intelligence, and has normal skin, hair and voice. The pituitary dwarf presents no difficulty because the limbs and trunk are in perfect proportion.

Prognosis.—The majority of infants suffering from achondroplasia are either still-born or die shortly after birth. If the child does survive, the expectation of life is normal. The female achondroplastic faces greater risks in parturition than a normal woman.

Treatment.—No treatment is of any avail. Orthopædic treatment for bow legs is unnecessary. The pelvic deformity may necessitate Cæsarean section.

DYSCHONDROPLASIA

Three clinical conditions are included under this heading. In all of them islands of ectopic cartilage are found giving rise to multiple ecchondromata or enchondromata. The three conditions are grouped together because of one feature they have in common, namely arrest or perversion of the normal process of endochondral ossification in certain bones. This change differs from that seen in achondroplasia only because it is neither symmetrical nor universal. Different manifestations of dyschondroplasia may occur in various members of the same family.

(i) *Hereditary multiple ossifying ecchondromata (hereditary deforming chondrodysplasia, diaphysal aclasis, or multiple cartilaginous exostoses).* This is a fairly common disease in which multiple bony tumours are found in association with certain other skeletal deformities. It is hereditary and may affect several individuals of the same family. It is more common in males than in females in the proportion of 3 to 1. It is usually discovered in childhood. Palpable bony tumours up to 2 cm. or more across are found more or less symmetrically placed near the knee, shoulder, hip, ankle and wrist. The scapula, ribs and pelvic bones may sometimes be affected. The stature is shortened and the limbs may be unequal in length. In the majority of cases the ulna and fibula are disproportionately short in relation to the radius and tibia. Bowing of the radius, ulnar deviation of the hand, irregular length of the fingers, and valgus deformity of the foot all may occur. Sarcoma supervenes in 5 per cent. of cases. Local exacerbation of symptoms in a patient over 30 years of age may be the first indication of its onset. Rarely pressure of an exostosis upon the spinal cord may cause paraplegia, or upon a nerve trunk pain or local paralysis. Aneurysm has been recorded from pressure upon an artery. The radiological appearances are characteristic. The metaphysis of the bone affected is broadened and distorted, and ossifying ecchondromata with broad bases and pointed tips project from it. The cartilaginous cap of the tumour is not seen unless it is calcified. The earlier the ecchondroma occurs the nearer to the centre of the shaft will it be. Where ecchondromata protrude between adjacent bones such as the tibia and fibula, local fusion may occur. The ulna is

likely to be short and to end in a point, articulating with the radius on its mesial aspect but not partaking in the carpal articulation. Usually no treatment is required but should it be necessary to remove any particular swelling this is easily carried out.

(ii) *Multiple chondromata (Enchondromatosis)*. This is a rare disease affecting the bones of the hands and feet. Cartilaginous swellings in the fingers and toes begin in childhood and increase in size up to the age of 30 years. The swellings are firm, elastic, rounded and slightly translucent. The skin over the larger ones may be tightly stretched and shiny and show prominent veins. The hands and feet may become hideously deformed. Sometimes a rib near the costal cartilage, the sternum, the pelvis, and the scapula are affected. In certain cases the ulna and fibula are disproportionately short as in diaphysial aclasis. Spontaneous fractures may occur, and sarcoma may supervene after years. Radiologically chondromata are seen as rounded, eccentric translucent areas expanding the corticalis, interrupting its outline, and projecting into the soft tissues. Sometimes the swellings are trabeculated and they may contain dense, punctate, calcified areas. Where operation is undertaken to excise some of the chondromata care must be exercised to avoid spontaneous fracture of the phalanges or metacarpals.

(iii) *Unilateral chondrodysplasia (Ollier's disease)*. This is a very rare type of chondrodysplasia occurring in children and sometimes familial. It usually has a completely unilateral distribution, but some cases have only one bone or one limb affected, and others are bilateral. Some abnormality is often first noticed between the first and second years of life, when as a rule one limb is found to be shorter than its fellow. The difference in length becomes progressively greater as growth proceeds. Deformity may occur either because weight bearing causes bending of the bone, or because of the different rate of growth where only one of the paired bones is affected. Most patients seem to reach adult life, when their symptoms are mainly those of their deformities and sometimes of a secondary arthritis. In a small proportion of cases sarcoma supervenes. The diagnosis largely depends upon examination of radiographs. The ends of the long bones show translucent longitudinal striæ interrupted by small pale mottled areas and dark punctate spots. In the areas affected there is extensive alteration in the pattern of the corticalis and spongiosa, but the centre of the shaft remains normal. As the child grows older the typical striped appearance disappears and is replaced by dense punctate speckling due to areas of calcification. The disease has occasionally been mistaken for osteitis fibrosa, but the radiological appearances are pathognomonic. Treatment is concerned with the prevention and relief of deformities, and proceeds along the usual orthopaedic lines. Osteotomy is sometimes necessary. Fractures are of fairly common occurrence, and like the osteotomies appear to unite well.

DISEASES OF THE BONE MARROW

MULTIPLE MYELOMA

Synonyms. — Myelomatosis ; Kahler's Disease ; Plasmacytoma ; Hæmatogenous Myeloma.

Definition.—A fatal disease characterised by the development of multiple tumours in the skeleton, which arise from cells of the bone marrow. It is very rare. The bones are affected in the following order of frequency : spine, ribs, sternum, skull, scapula, pelvis, clavicle, humerus and femur.

Ætiology.—Multiple myeloma is of unknown origin. It is a malignant neoplasm of the hæmatogenous marrow occurring in multiple foci. The disease is related to leukemia, but differs from it in the sharper localisation of the neoplasia, the absence of enlargement of the spleen or lymph-glands, the much smaller tendency for the abnormal cells to enter the blood stream and the frequent appearance of Bence Jones protein in the urine. Intermediate forms occur with features of both diseases. It is associated with interesting alterations of protein metabolism. The disease is sometimes familial. It begins most commonly at the age of 55, and only 10 per cent. of cases occur before 40. The sexes are affected in the proportion of three men to two women.

Pathology.—Multiple deep red or reddish-grey sharply defined tumours are found distributed throughout the red bone marrow. They are usually a few millimetres in diameter and very numerous. Occasionally a tumour may reach a diameter as great as 5 cm. They are composed of blood-forming cells, either myelocytes, myeloblasts, erythroblasts, or cells resembling plasma cells. They erode bone, sometimes expand the cortex, and cause deformities and spontaneous fractures. Rarely a diffuse hyperplasia of the marrow is associated with foci of tumour formation. Tumours may also be found outside the skeleton in the tonsils, liver, spleen, kidneys, or sex-glands, and these lesions may even precede those in the bones. The marrow tumours give rise in the urine to the Bence Jones protein which appears as a cloud when the urine is heated to 55° C., redissolves at 85° but reappears on cooling. It is found in 75 per cent. of cases, from a trace to a large amount. In some cases it appears early in the disease, in others late. Its occurrence may be continuous or periodic. Sometimes a substance allied to amyloid material is deposited in the muscles and in nodules connected with the periosteum, bursæ, tendon sheaths and joints. It is possible that both the Bence Jones protein and the amyloid substance are produced from the breakdown of myelomata. The serum globulin is usually increased even as much as 8 per cent. (normal 2 per cent.). The albumin-globulin ratio may drop from the normal 2·2 to a figure as low as 0·5. The formol-gel reaction is positive (see p. 253), and there is a very rapid rate of sedimentation of the blood. Metastatic calcification while by no means constant has been frequently observed in the kidney, lung, stomach, myocardium and uterine mucosa. The serum calcium is usually normal, but, taking into account the bone destruction which occurs as the result of erosion by the marrow tumours and also the metastatic calcification, it is not surprising

that high serum calcium values have sometimes been recorded. Figures from 13 to 16 mg. per 100 c.c. have been found. Those cases with a normal serum calcium have a normal calcium output, while those with a high serum calcium have an output up to double the normal. Where renal insufficiency complicates multiple myeloma the plasma phosphorus is found to be high and may rise as the kidney condition becomes worse. The parathyroids are not enlarged in multiple myeloma.

Symptoms.—The initial symptom is pain, often bilateral, in the thoracic, abdominal and lumbar regions, and sometimes in the neighbourhood of the joints. Progressive kyphosis or angular curvature of the spine with loss of total height follows. The spine, sternum and ribs may be tender on percussion. It is unusual for any of the myelomata to be palpable. In 60 per cent. of all cases spontaneous fracture occurs in the ribs, sternum, or later in the long bones. In no other type of bone tumour does pathological fracture occur so frequently. In some cases amyloid masses may be palpable as firm, rounded, slightly tender, subcutaneous nodules more than a centimetre in diameter. They are felt especially in the scalp, along the spine, near the joints, and in the musculature, particularly that of the pelvic and shoulder girdles. There is usually a hypochromic anæmia, which becomes aggravated in the terminal stages. In a few instances cells of the type which constitutes the tumour enter the blood stream in larger or smaller numbers, and it is probable that they can be found in the majority of cases if a sufficiently careful search is made. In rare instances the anæmia is of the leucocrythroblastic type (p. 783). Nephritis without hypertension is fairly common. The temperature is usually normal but recurring fever has been observed. The patient ultimately becomes bedridden and cachectic. It seems justifiable on clinical grounds to consider separately what may be called the vertebral form of the disease. Here the growth is confined for some time to the vertebral and extradural tissues. Moreover, death may occur before the growths become widespread, and sometimes without the Bence Jones protein having appeared in the urine. In this variety the patient rapidly develops signs of a transverse spinal lesion with blockage of the spinal canal. The thoracic cord is usually the site of compression and there is focal spinal tenderness. Radiographs show destruction of the corresponding vertebral body.

Radiological Appearances.—In radiographs the marrow tumours are found mainly in the spine, ribs, sternum and skull. They are seen as clean-cut elliptical or circular areas of complete translucence, set closely together and varying from 1 mm. to 5 cm. in diameter. The larger tumours may expand the cortex of the bone affected. There is a good deal of generalised osteoporosis throughout the affected bones. The spine shows collapse of the bodies of one or more vertebræ. The skull is not thickened. Pathological fractures, especially in the ribs, are very common.

Diagnosis.—Once the lesions have appeared in many bones the diagnosis is easily made. The age of the patient, multiple involvement of the bones of the thoracic cage, spontaneous fracture of a rib, Bence Jones protein in the urine, progressive anæmia, cachexia and characteristic radiographs make an unmistakable clinical picture. Biopsy of a portion of bone or examination of a bone-marrow smear from a sternal puncture may reveal the characteristic myeloma cells. Secondary carcinomatosis of bones may

cause difficulty, especially in cases in which the primary growth is symptomless. It is essential to differentiate the disease from generalised osteitis fibrosa (hyperparathyroidism). There is some resemblance in the clinical picture as it affects the skeleton, but the presence of the Bence Jones protein and the blood chemistry are characteristic. In multiple myeloma the serum calcium is usually normal. If it is high it is associated with a high plasma phosphorus, whereas the characteristic effect produced by parathyroid hyperfunction is a high serum calcium with a low plasma phosphorus. In osteomalacia the patient is usually a woman in the child-bearing period of life, and a good deal of bending occurs in the bones. The blood chemistry is characteristic. Sometimes in the early stages of multiple myeloma widespread pain in the thoracic, abdominal and lumbar regions may lead to a mistaken diagnosis of fibro-myositis. In tuberculous caries of the spine neither the ribs nor the sternum are involved. The fact that the Bence Jones protein is found in the urine in an occasional case of leukemia need cause no mistake. In those cases in which nephritis complicates multiple myeloma the albuminuria may cause difficulty. The Bence Jones protein may be detected in the presence of albumin by making the urine slightly acid with acetic acid, boiling it and filtering while hot, using a funnel with a hot-water jacket. If Bence Jones protein is present the filtrate will become cloudy as it cools.

Prognosis.—The prognosis is hopeless. Death often occurs within six months of the onset of symptoms, but occasionally a patient survives for two years or more. Broncho-pneumonia, cachexia, or compression paraplegia with ascending pyelo-nephritis are the usual terminal events.

Treatment.—The patient should be treated by rest in bed, anodynes and suitable splinting when necessary. Occasionally deep X-irradiation can be used with good effect. It alleviates pain and reduces the size of the tumours, but it does not retard the progress of the disease. It is clearly unjustifiable to explore the neck in search of a parathyroid tumour. When the symptoms and signs point to compression of the spinal cord surgical intervention may be worth while. Laminectomy reveals a grey or reddish-grey extradural mass either pushing the cord backward or encircling it. Removal of the mass decompresses the cord and is followed by improvement. Deep X-irradiation and the wearing of a spinal brace are advised after laminectomy.

DONALD HUNTER.

LESLIE J. WITTS.

GAUCHIER'S DISEASE

In 1922 Pick discovered a gross osseous form of Gaucher's disease. It is exceedingly rare. The symptoms are pain in the bones, pathological fractures and sometimes angular curvature of the spine. In radiographs the bones show patchy osteoporosis. A characteristic feature is that the ends of the femora are widened evenly. Both skull and pelvis may be involved. Sometimes scattered through the bones there are focal pale rounded areas which expand the cortex. These areas are deposits of kersin, a galactolipin. The usual characteristics of Gaucher's disease are, of course, present (see pp. 834, 835).

HAND-SCHÜLLER-CHRISTIAN'S DISEASE

The lesions of Hand-Schüller-Christian's syndrome (lipoid *granulomatosis* or *xanthomatosis* of bones) are not confined to the calvaria, the orbit or the sella turcica (see p. 836). Erosions of the maxilla and mandible have been described, resulting in loosening or falling out of the teeth. Erosion of the petrous bones may lead to a syndrome simulating otitis media, and bilateral deafness has been observed. Large areas of rarefaction have been described in the long bones of the extremities, and in the spine, pelvis, ribs and clavicles. Pain may occur in the bones affected, especially the head, pelvis and thigh. Spontaneous fracture is not uncommon. When the pelvis is involved there may be deformity, including shortening of one lower limb. In some cases the skull escapes entirely, diabetes insipidus and exophthalmos being absent. Radiologically the deposits of cholesterol-ester are seen as irregular clean-cut translucent areas sometimes with a few coarse trabeculæ. In order to distinguish the condition from multifocal osteitis fibrosa it may be necessary to excise a portion of bone for histological section. The lesions tend to yield temporarily to treatment by X-irradiation.

DONALD HUNTER.

SECTION XIX

DISEASES OF THE SKIN

I. ANATOMY AND PHYSIOLOGY

IN order that the diseases which affect the skin may be understood it is necessary to give a brief account of the anatomy, physiology and general pathology of the skin.

ANATOMY.—The skin is a fibrous structure varying considerably in thickness in different parts of the body and covered externally by several layers of epithelial cells. On section its main bulk is seen to be made up of white fibrous tissue bundles running chiefly parallel to the surface and bound together by thin fibres of elastic tissue. The surface of this fibrous mass, which is called the *dermis*, is not level but is surmounted by a number of finger-like projections, called *papillæ*, which fit into corresponding depressions or pits on the under surface of the epithelial covering which is called the *epidermis*.

In the fibrous stroma of the dermis blood vessels, lymphatics and nerves ramify. The *arteries* form a plexus of large vessels at the junction of the dermis with the subcutaneous fatty layer and from this deep plexus arteries pass upwards, frequently near hair follicles or sweat ducts to which numerous twigs are sent, to another superficial or sub-papillary plexus situated just below the bases of the *papillæ*. From this smaller vessels pass upwards to end in the *papillæ*. The *veins* follow a similar course in the opposite direction.

Lymph circulates freely in the spaces between the cells of the epidermis and the fibres of the dermis, but definite lymphatic vessels are also found in the *papillæ* and in the dermis, accompanying the blood vessels.

The *nerves* of the skin are both medullated and non-medullated. They also follow the course of the blood vessels and are distributed to the hair-follicles, sweat and sebaceous glands, blood vessels, *arrectores pili* muscles and to the connective tissue bundles in their passage through the dermis. Losing their medullary sheath in the sub-papillary layer some fibres pass up and are distributed to the *papillæ* and to the basal and mucous layers of the epidermis, while other medullated fibres end in curious whorls in the *papillæ*, which are called the touch corpuscles of Meissner; a few end in small ovoid bodies, known as Pacinian bodies, in the subcutaneous tissue.

The *epidermis* consists of several layers of cells, varying considerably in thickness in various parts of the body. The layer nearest the dermis consists of regular cubical cells to which it is intimately attached, and it is from this layer that the rest of the epidermis is developed; it is spoken of as the *stratum germinativum* or *basal layer*. The layers above this consist

of cells in various phases of transformation into horn cells, which are seen in their final form in the outermost layers. Above the basal layer there are several layers of large polyhedral cells with large nuclei and a spongy cell substance; they are bound to one another by fine fibrils from which they have obtained the name "prickle" cells; this is the *mucous* or *Malpighian layer*. Above this are one or two layers of lozenge-shaped cells, lying parallel to the surface of the skin, whose protoplasm contains large deeply staining granules, giving to it the name *granular layer*. Then comes a thin transparent layer, the *stratum lucidum*, and above this the *horny layer*. Here the cells have lost their nuclei and protoplasm, and consist only of a cell capsule which has been converted into a highly resisting substance called keratin; the cells are intimately bound together and can only be separated with great difficulty. Thus a strong protective layer is produced which can only be destroyed by strong acids or alkalis or by violence.

In the cells of the basal layer are produced granules of *pigment* which act as a protection against light rays. The pigment is an iron-free substance named *melanin*, and its method of production is still a matter of controversy. In the dark races the deeper cells of the mucous layer also contain melanin granules, and these can also be found in wandering cells in the dermis but are not formed in these cells.

Dipping down from the epidermis into the dermis are certain epithelial structures, the hair follicles with their sebaceous glands, and the sweat glands.

The *hair follicles* are pockets of epithelium which contain in their walls all the layers of the epidermis in a modified form. They penetrate the whole thickness of the dermis and often pass into the subcutaneous tissue for some distance. The *hairs* grow from enlarged papillæ at the bottom of the pits and also consist of modified epidermis, so modified that the cellular structure is only visible on the outer layers formed of superimposed scale-like cells, the cuticle of the hair; the remainder of the hair structure consists of an outer fibrous part, the cortex, and a more succulent centre, the medulla. Hairs are present all over the skin except on the palms and soles, and vary very much in size. Their ordinary characteristics need no description. The hair follicle is inserted obliquely in the skin, and on the aspect where it forms an obtuse angle with the surface, a small band of unstriated muscle, the *arrector pili*, is found, attached below to the hair follicle near the papilla and above to the fibrous tissue underlying the surface epidermis. This muscle on contraction erects the hair.

From the same side of the hair-follicle, and lying between it and the muscle, so that it is compressed when the muscle contracts, is found a sacculated gland growing out of the follicle; this is the *sebaceous gland*. It secretes an oily substance which lubricates the hair and the skin surrounding the follicle. The secretion is produced by fatty degeneration of the cells of the gland itself, and is expressed by contraction of the *arrector pili* muscle. These glands vary much in size and in some cases far exceed that of the hair follicle; in this case they often open directly on the surface of the skin in common with the hair follicle. They are most developed on the face, back, chest and scrotum.

The other epithelial appendages are the *sweat glands*, which are found everywhere in the skin. They are tubular structures which pass down to the lowest part of the dermis or into the subcutaneous tissue and end in a coil,

the *sweat* or *coil gland*, the straight portion passing to the surface being the *sweat duct*. Both the duct and gland consist of a single layer of cubical cells which becomes continuous with the basal layer of the epidermis. The duct has no special epithelial lining through the epidermis. Involuntary muscular fibres, which expel the secretion of the gland and are under the control of special pilo-motor centres, are present among the coils of the glands. The secretion of the gland is a true secretion and is not produced by degeneration of its cells, as in the case of the sebaceous glands. Certain large sweat glands, called *apocrine glands*, occur in the axilla, nipples and pubic region, which do, however, show breaking up of the cell protoplasm during activity.

The only other skin structures that require mention are the *nails*. These are simply modifications of the horny layer of the skin. The nail grows from that portion of the nail bed which is partly hidden by the nail fold and partly seen as the lunula of the nail, which forms a pale half-moon shaped area above that structure; this area is called the *matrix*.

PHYSIOLOGY AND PATHOLOGY.—The functions of the skin are four in number—(1) It forms a protective covering over the whole body; (2) it is an organ of secretion; (3) it is the seat of tactile sensation; and (4) it plays an important part in regulating the temperature of the body. The skin also allows of absorption, though this can scarcely be considered one of its primary functions. From the point of view of dermatology the two first functions are the most important.

The *protective function* is a double one: firstly the skin as a whole lying on a loose connective tissue pad, protects the deeper structures from damage by acting as a buffer; secondly, the resistant characters of the horny layer protect from irritants, not only the deeper structures, but also the layers of the skin lying beneath it, for the moist cells of the body unprotected by these dry keratinised cells would perish if exposed even to the ordinary atmosphere. Damage to the horny layer is responsible for a very large group of inflammations of the skin.

The horny layer, however, does not act quite alone: it is made more impermeable to simple external irritants by the presence of a thin layer of oil on its surface which is provided by the secretion of the sebaceous and sweat glands. As will be shown later, absence or deficiency of this oily secretion renders the skin much more susceptible to external irritants. On the other hand, excessive sweat secretion from the large amount of water it contains may make the horny layer sodden, and therefore more liable to damage. Similarly an excessive sebaceous secretion tends to make the horny layer thicker and is an excellent medium for the growth of organisms.

It must further be remembered that the horny layer does not form a complete sheet, but that innumerable invaginations which form the hair follicles and sweat ducts are present. These considerably weaken the protective power of the horny layer, and it will be found that at these spots inflammatory reactions, due to damage of this layer, are most likely to occur. It is also practically certain that absorption takes place at these follicular openings.

The *secretions of the skin* are the sweat and the sebum, the latter of which is the secretion of the sebaceous glands. The former is a watery fluid which contains traces of sodium chloride and other mineral salts, extractives, and a very small quantity of urea and fats. It varies very much in quantity, but

normally about equals the quantity of urine voided and, therefore, is responsible for the removal of nearly 50 per cent. of the total water excreted by the body. The main function of this excretion of water is to maintain the temperature balance of the body and, therefore, in hot weather the amount of sweat is increased in order to cope with more rapid evaporation and so keep the body surface cool, the quantity of urine being correspondingly diminished unless larger quantities of water are imbibed. In cold weather the reverse is the case.

A small amount of carbon-dioxide is also excreted by the skin, and the latter may therefore be said to be an accessory *organ of respiration*.

The sweat has special interest to the dermatologist not only from its lubricating effect on the horny layer, but also from the fact that certain drugs are sometimes excreted by it, and it is possible that some of the eruptions caused by the ingestion of these drugs may be produced by their irritating action during the process of excretion by the sweat.

The sebum is an oily secretion whose function appears to be entirely that of lubricating the hairs and surface of the skin: it not only helps to protect the skin from chemical irritants, but also from the actinic rays of the sun.

The other two functions of the skin, namely, the *tactile sense* and the *regulation of temperature*, will have been dealt with elsewhere, and as they affect the dermatologist but little, they will not be further considered here.

II. GENERAL DESCRIPTION OF SKIN DISEASES

The bulk of skin diseases fall into two great classes, inflammations and new-growths. There are in addition certain conditions which cannot be included under either of these headings, and will require special mention, namely, the disorders of secretion, of sensation, of the circulation, and of pigmentation; atrophies of the skin and certain congenital abnormalities. It will also be necessary to describe the diseases of the hair separately. As so many of the inflammations depend upon disorders of secretion, sensation and circulation, it is proposed to deal with these first. Before proceeding, however, to deal with pathological conditions of the skin, it will be useful to define the terms used in describing clinical manifestations. It must be realised, however, that these terms are used very loosely, and are only a convenient form of nomenclature.

A *macule* is a spot which is not raised above the skin; it may be vascular or pigmentary. The term is usually applied to small lesions up to the size of a pea, a larger lesion being called a "plaque" or "tâche." Large sheets of redness are generally called an "erythema."

A *papule* is a solid elevation usually not exceeding the size of a pea. If the surface is flat and smooth it is called a "plane" papule; if pointed an "acuminate" papule.

A *tubercle* or *nodule* is an elevation usually between a pea and a hazel nut in size. The term nodule is also used for small solid swellings in the substance of the skin and subcutaneous tissue which do not necessarily project above the surface.

A *tumour* is a swelling exceeding a hazel nut in size. It need not

necessarily be solid, but this term is not usually applied to thin-walled superficial fluid swellings.

A *wheel* is a circumscribed elevation of the skin of a transitory character in which œdema is so marked as to force the blood out of the superficial capillaries and so produce a dead white elevation.

A *vesicle* is an elevation not larger than a pea containing clear fluid.

A *bullæ* is a similar lesion of larger size ; in other words, a *blister* or a *bleb*.

A *pustule* is a similar lesion to a vesicle, but contains pus instead of clear fluid.

A *scale* is a lamella of the horny layer of the skin.

A *crust* is a mass produced by the drying of exudates on the skin.

An *excoriation* is an abrasion of the superficial layers of the epidermis.

A *fissure* is a crack in the skin.

An *ulcer* is a circumscribed loss of tissue involving the whole thickness of the epidermis.

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III. CONDITIONS PREDISPOSING TO SKIN DISEASES

A.—DISORDERS OF SECRETION

Under this heading are included deficiency or absence of sweat and sebaceous secretion, and also excessive secretion.

ANIDROSIS OR DIMINUTION OF SWEAT SECRETION

This occurs in many diseases, but is seen in its most marked form in xeroderma and ichthyosis. It also is seen in hypo-thyroidism and in its more marked form myxedema, in the degenerating skin of old people, and in poisoning by certain drugs, of which arsenic is one of the most frequent examples.

The milder cases of hypo-thyroidism show dryness of the skin, dryness, brittleness and thinning of the hair. They improve rapidly under the judicious administration of thyroid extract.

XERODERMA AND ICHTHYOSIS

These two names are applied to the mild and severe types of the same disease. The condition is one of abnormal dryness of the skin owing to the almost complete absence of sweat secretion accompanied by an overgrowth of the horny layer of the epidermis (hyperkeratosis).

Ætiology and Pathology.—The disease is inherited and often occurs in several members of the same family. It attacks both sexes equally. The disease is usually noticed about the second year of life, but some children are born with a condition closely resembling it (*ichthyosis congenita*): these children are frequently premature and generally stillborn. There is no very

definite evidence as to whether the changes in the epidermis follow the absence of secretions, or vice versa, or whether both are dependent on a common cause; possibly the overgrowth of the horny layer is an attempt on the part of Nature to compensate for the protection usually supplied by the only secretions. Histological examination shows very marked increase in the thickness of the horny layer, which is irregular and grows directly from the mucous layer, the granular layer being absent. The sweat glands are apparently normal histologically, although they do not function normally.

Symptoms.—In the milder cases (*xeroderma*) the skin is dry and rough, and there may be a certain amount of branny scaling on the surface. On the extensor aspect of the limbs the hair follicles are prominent and contain small horny spines. The palms and soles are more lined than normal, while the flexures of the body show little change. The hair is dry and lustreless, and occasionally stunted and brittle, while in a few cases only down grows on the scalp.

In the more marked cases (*ichthyosis*) the body is covered with large fish-like scales which are firmly adherent. They may be thin, transparent and colourless, or thick and dark in colour (the so-called alligator skin). In these cases the trunk and extensor aspect of the limbs are most involved, the face and scalp often showing little change, though the changes mentioned above may be present. This dry skin is particularly liable to become inflamed on account of alteration in its protective mechanism.

There is another form in which the disease develops in localised sheets, lines or bands (*ichthyosis hystrix*), but this condition is closely related to the linear nævi and will be dealt with under that heading.

Prognosis.—The disease persists throughout life, and although it can be relieved by appropriate treatment it never really gets well.

Diagnosis.—The dryness of the skin, the origin of the disease in early life, and its persistence, and the presence of fine or coarse scaling with the absence of inflammation render the diagnosis easy.

Treatment.—This consists in an attempt to replace the natural oil of the skin. Frequent warm baths, followed by the application of some oily preparation, are usually sufficient. One of the most useful preparations is glycerin. amyli, adip. lanæ hydros. āā ptes. æq., to which may be added 2 per cent. or 3 per cent. of salicylic acid if desired. Too vigorous use of soap is to be discouraged.

Some authorities recommend thyroid internally, on the grounds that the condition is due to deficient thyroid activity, but the results obtained have been scarcely sufficient to confirm this view.

HYPERIDROSIS

Definition.—This is a condition of over-activity of the sweat glands. It may be general or local.

Ætiology and Pathology.—Sweating in febrile illnesses is not included under this heading. The generalised forms are usually seen in adults, while the localised varieties are not infrequently seen in younger people. They are both probably due to disturbances of the nervous system, though it is difficult to say that they always occur in neurotic individuals. There is

no doubt, however, that hyperidrosis is very liable to produce a neurotic condition.

Symptoms.—*Generalised hyperidrosis.*—In this condition the patient sweats excessively, often on the least exertion or excitement. The sweating may be so severe that the patient has to change his clothes several times a day—even in cool weather.

Localised hyperidrosis.—There are certain regions of the body particularly liable to excessive sweating, namely, the palms and soles, the axillæ, and the genital region and perineum. The sweating is often very excessive, and may last for a very long time; there is, however, a tendency for the condition to diminish with age, it being most marked in the latter half of the second and the third decades of life.

The sweat allows certain saprophytic organisms to grow freely, with the result that decomposition takes place, and an extremely offensive odour develops. This is chiefly noticed in the feet, and is spoken of as *bromidrosis*. Occasionally the sweat is coloured (*chromidrosis*), due to bacterial activity.

The skin constantly soaked in sweat is subject to attacks by irritants, bacterial and otherwise, and various forms of dermatitis are frequent complications of hyperidrosis, especially the forms spoken of as *miliaria rubra* and *dysidrosis* (see p. 1414).

Prognosis.—Localised cases will generally respond to treatment; but the more severe generalised cases are apt to be very persistent.

Treatment.—For the *generalised cases* frequent warm baths are required, to keep the skin clean. Dabbing on a solution of tannic acid (1 per cent.) in 50 per cent. alcohol, or a dusting powder of talc containing 3 per cent. salicylic acid, is often useful. The general health should be looked to, and all dietetic errors and habits liable to cause sweating rectified. Some cases benefit by the internal administration of bromides and belladonna.

The *localised cases*, when extreme, are best dealt with by X-rays. Ten or twelve doses, each of 120 r ($\frac{1}{3}$ skin unit), given in groups of four doses at weekly intervals, with intervals of one or two months between the groups, usually give a satisfactory result. Bromidrosis of the feet is best dealt with by frequent washing and change of socks, by bathing in 1 in 4000 potassium permanganate solution, or by washing with lysoform, or other formalin soap. The feet should then be freely dusted with the powder mentioned above.

SEBORRHOEA

Definition.—By the term seborrhœa is meant an over-activity of the sebaceous glands, resulting in an abnormally greasy skin.

Ætiology.—This condition occurs from the time of puberty onwards, gradually diminishing as age increases. It tends to affect certain races and families, but is also influenced by the habits of individuals. Gastric disturbance, constipation, anæmia, uterine trouble and the like all tend to exaggerate the condition.

Pathology.—The condition appears to be due to some disturbance of metabolism not yet fully determined. Some authorities consider that infection by certain organisms play a part, but the evidence is inconclusive.

Symptoms.—The regions affected are the face—especially the nose, naso-labial folds—the scalp, chest and back. In the milder cases the skin is greasy; in the more severe cases it is thickened—giving rise to a muddy appearance—and the follicles are patulous. This condition Darier has labelled “la kérose.” In other cases the sebaceous follicles are plugged with semi-solid sebaceous material.

Complications.—Seborrhœa is the underlying cause of many skin affections. Acne vulgaris is merely a more marked stage of the follicular plugging noted above. Infection by certain organisms producing seborrhœic dermatitis is very common, while the skin is especially liable to ordinary eczema and impetigo contagiosa. Acne rosacea is particularly liable to occur in seborrhœic individuals.

Treatment.—The general health must receive attention. Diet should be regulated to get rid of dyspepsia and constipation; especially should excess of sugars and starches be avoided. Iron and arsenic are indicated in anæmia, and uterine troubles should be appropriately treated.

Frequent washing with soap and water is necessary. Sulphur has a marked effect in diminishing the secretion and especially in preventing organisms from growing in it; it may be used as a powder—sulphur. precip. 5 parts, pulv. talc to 100 parts; or as a lotion, potass. sulphurat. min. 60; sp. vini. rect. fl. oz. 2, aquam ad fl. oz. 8.

B.—DISORDERS OF SENSATION

The disorders of sensation comprise hyperæsthesia, anæsthesia and paræsthesia.

Hyperæsthesia is generally symptomatic of some organic or functional disease of the nervous system, and has little or no importance in dermatology.

Anæsthesia also is usually symptomatic; but one form occasionally comes under the notice of the dermatologist first, namely, that associated with syringomyelia. The individuals affected show anæsthesia with trophic changes in the skin of the fingers, often with whitlows and other signs of skin sepsis. Further investigation shows the lesions to be only part of a more general disease of the nervous system. This type is spoken of as Morvan's disease, and is dealt with elsewhere (p. 1731). Localised areas of anæsthesia, with redness, are frequently an early sign of leprosy (see p. 122).

Paræsthesia forms the most important group from the dermatological point of view, as it includes itching or pruritus.

PRURITUS

Under this heading are included those cases of itching of the skin in which there is no other obvious dermatosis. It may be general or local.

baths and lotions: for the latter *lotio alkalina* (sodium bicarbonate and borax, 1 per cent. of each in distilled water) answers well.

It is very important to see that the patient changes his undervest at night. Many patients do not do so, and this undoubtedly predisposes to pediculosis. Even, however, in cases where no pediculosis appears to be present, cases are often cured by attention to this detail.

LOCALISED PRURITUS

Certain parts of the body are liable to pruritus; these are the anus, vulva, and scrotum. Other local areas, however, may be attacked, such as the front of the ankle, lower part of the leg, thighs, back of neck and scalp.

Ætiology and Pathology.—Most of these localised cases probably start from some transitory cause, which gets better; but a vicious circle has been started, the scratching bringing on itching, and this causes scratching again. In the case of the anus, piles are a frequent cause. Some cases are, as Castellani has shown, due to fungus infection. Vaginal discharge frequently starts a vulval pruritus, as do sugar and other irritating substances in the urine. Sweating and friction of clothes, and possibly some parasitic condition, such as *dhobie itch*, may start a scrotal pruritus.

For the other cases it is generally difficult to find a cause, and it is usually necessary to treat symptoms.

Symptoms.—The localised itching is often followed by marked changes in the skin from rubbing and scratching. The usual change noted is that called *lichenification*, in which the skin becomes thick and rigid, the lines of the skin deeper, and the area assumes a dull purplish colour, and on clearing up often leaves deep brown pigmentation. This is well seen in the patches in the flexures and on the limbs, but is modified in the moist parts, where it usually takes on a white sodden and swollen appearance, surrounded by a bright red inflammatory zone. The surface is often covered by numerous excoriations or blood-stained crusts. Occasionally these excoriations may become septic, and ulceration may occur. The symptoms are often so severe as to affect the patient's health by sleeplessness and worry.

Treatment.—The treatment recommended for generalised pruritus is often ineffective in the localised cases, such as that directed towards obtaining sleep.

The first thing to do is to remove any local cause; *e.g.* vaginal discharges may require treatment. The bowels should be made to act freely by paraffin, saline aperients or enemata; aloes is better avoided. Irritating food, especially coffee, alcohol, curries, etc., should be interdicted. Piles may require surgical treatment, and any rectal discharge, fistula or worms should be dealt with. All the parts should be carefully washed, mild alkaline lotions or weak antiseptics being useful, and afterwards dried thoroughly and a talc or zinc oxide powder applied. Further relief may be obtained by the application of 1 per cent. phenol and camphor cream, or 5 per cent. oleinum cocainæ (B.P.C. 1923). If these milder measures fail, the parts may be painted with silver nitrate, grs. 10, ap. æther. nit. fl. oz. 1, twice or three times a week, and a bland cream or mild alkaline lotion applied. In cases due to fungus infection, Castellani's fuchsin paint (see p. 1438) usually proves efficacious.

The most radical results, however, are obtained by X-rays. Three or four doses of 120 r ($\frac{1}{2}$ skin unit) given at weekly intervals to the affected area will nearly always remove the itching completely, and the secondary changes in the skin will disappear. For localised body pruritus, excellent results are obtained by painting the affected parts with crude coal tar, which is allowed to dry, and a talc powder applied. This method should not be used if sepsis is present. Further, much relief is often given by exposures to the ultra-violet rays of the mercury-vapour lamp.

C.—DISORDERS OF CIRCULATION

Only certain circulatory disorders have any bearing on skin diseases, if we do not include those disturbances associated with inflammation. Those which are referred to below usually come to the dermatologist on account of secondary changes produced in the skin.

ACROCYANOSIS

Synonym.—Chilblain Circulation.

This is a condition most frequently met with in young women, though by no means confined to them, characterised by persistent blueness of the extremities, including the hands and feet, the nose and the ears. It includes the condition known as erythrocyanosis crurum puellarium frigida, which is fully described on p. 1079.

CHRONIC VASCULAR STASIS OF THE LOWER LIMBS

This condition occurs in almost all individuals approaching middle life, and progresses with age. In individuals suffering from varicose veins it commences earlier. As a rule no special symptoms are produced, but if the skin of the lower part of the leg is damaged—and it is particularly prone to injury—it does not heal well, and there is a great tendency for a dermatitis to be set up. In some cases, however, the venous congestion causes itching, and if the skin in this region is scratched a moist dermatitis is liable to arise, which becomes septic, and healing does not readily occur. In these ways we have the well-known “eczema of the leg” produced. These cases frequently go on to ulceration, and the familiar chronic *varicose ulcer* of the leg is the result.

ROSACEA (ACNE ROSACEA)

Definition.—This is a chronic vascular congestion of the nose and central part of the face, resulting from dyspepsia and other internal conditions, and followed by secondary inflammatory changes in the skin.

Ætiology and Pathology.—The disease is common in both sexes, but rather more so in women. It begins usually after 30, but is occasionally

seen before that age. It is generally associated with dyspepsia, usually of the flatulent type, though in many cases there is no very obvious gastric disorder. Cases have been recorded in which complete achylia gastrica was present, or in which there was considerable diminution in the hydrochloric acid in the gastric juice. Uterine disturbance and the menopause are responsible for some cases. Alcohol and strong tea drinking are potent causes.

The mechanism of this vascular dilatation is not quite clear. It is generally assumed that some toxic substance is absorbed, and acts on the vasomotor system; but it is more probably a neurosis. The follicular lesions are the result of the congestion and of the increased sebaceous secretion which the hyperæmia causes, as well as of increased activity of the skin cocci.

Symptoms.—The early symptoms are either those of transitory flushing of the face, or the nose gradually becomes red. Examination shows the presence of dilated vessels on the alæ of the nose. Later the congestion becomes more marked and not only affects the nose but the adjoining parts of the cheeks, the chin and the centre of the forehead. The redness may be persistent or remittent; it is worse after meals. There is usually an increase in sebaceous secretion so that the skin becomes abnormally greasy. Scattered red papules now appear at the follicular openings, and often a bead of pus is seen in them, but no sebaceous plug or comedo. This is the typical "acne" rosacea. If the skin is very dry, this papular rash may be absent; but the whole affected area may become dry and scaly, especially if exposed to the weather, showing that the congestion renders the skin more susceptible to mild external irritants. In other cases, these inflammatory conditions are absent; but the vessels become very dilated, and much disfigurement results. In the most severe cases there occurs an overgrowth of skin and subcutaneous tissue which converts the nose into a lobulated tumour—*rhinophyma*. A number of cases show a persistent type of conjunctivitis, sometimes associated with a keratitis and corneal ulceration. The severity of the eye symptoms does not, however, appear to correspond with that of the skin lesions.

The patients complain of few symptoms except dyspepsia and flushing of the face; but the unsightliness of the condition brings them for relief.

Diagnosis.—The "acne" variety must be distinguished from acne vulgaris, by the limitation of the lesions on the centre of the face, by the underlying congestion and vascular dilatation, and by the absence of the comedo. The age is also a help, as acne vulgaris is commonest between 15 to 30. It must be remembered that sometimes the two conditions occur together. In dry "eczemas" of the face, the possibility of an underlying rosacea should not be overlooked.

Treatment.—The cause must first be dealt with. A fractional test meal will give useful information as to digestive function. Easily digested food, with a minimum of carbohydrates and green vegetables, should be ordered, little or no fluid should be taken with meals, and alcohol and strong infusions forbidden. Sod. bicarb. grs. 15 to 20 with a bitter three times a day after food is of great help. Dilute hydrochloric acid, min. 20 thrice daily, may be given in achlorhydric cases. The non-dyspeptic cases often do

well on bromides and belladonna. Ichthammol grs. 3 to 5 (in capsules), or menthol, gr. 1, t.d.s., is often useful. The bowels should be regulated.

Local treatment should be sedative in the main. In the acneiform cases, calamine lotion with liquor calcis sulphuratæ (1 in 8) applied two or three times a day should suffice. In the dryer forms, ung. aquosum (B.P.) should be applied night and morning. When the veins are much dilated and unpleasantly prominent they may be destroyed by electrolysis or a fine pointed cautery. In the cases with much hypertrophy it may be advisable to remove some of the overgrown tissue with a knife. This can be done without leaving much scarring.

Of the other disorders of circulation which occasionally come under the notice of the dermatologist may be mentioned *Raynaud's disease* and *erythromelalgia*; but these are dealt with in other sections of this work (see pp. 1075, 1078).

A. M. H. GRAY.

IV. INFLAMMATIONS OF THE SKIN

Having dealt with some of those disorders which predispose to inflammatory changes, it is now possible to consider the Inflammatory Diseases of the Skin. These may be divided roughly into two great classes: (1) The superficial inflammatory dermatoses, due mainly to irritants applied externally; and (2) the deep inflammatory dermatoses, due mainly to toxic substances circulating in the blood. This division is not quite so definite as one might suppose, but it is a good basis on which to work. There are, however, a certain number of inflammatory dermatoses which cannot easily be placed in either group; these will have to be considered separately.

A.—THE SUPERFICIAL INFLAMMATORY DERMATOSES

These are produced as a rule by the application of external irritants to the skin, but there are a certain number of cases in which the external irritant cannot be traced, and in which the general symptoms suggest an internal toxin. External irritants may also cause deep-seated inflammatory dermatoses, but only when they are introduced through the epidermis; thus, the puncture of the hairs of the nettle may produce an urticaria, the infection of a crack an erysipelas, a syphilitic chancre or a patch of lupus vulgaris. Nevertheless, the general rule is that a superficially applied irritant produces a superficial dermatosis.

External irritants may be classified into the following groups: (a) chemical; (b) heat and cold; (c) actinic; (d) bacterial; and (e) mechanical. This order is chosen because the clinical types can best be explained in this way. The reaction of the skin to these different irritants is generally of the catarrhal type, which is known as "eczema." This term has, therefore, been

used freely to label lesions, but, as will be explained later, is used more rigidly in describing cases.

Ætiology and Pathology.—Chemical irritants applied to the skin may cause immediate, or primary, local necrosis, but only those reactions of the skin to irritants which do not cause local death of the tissues are dealt with in this section. Secondary local necrosis does, however, sometimes result from such reactions, as in the case of chronic leg ulcer following a varicose "eczema."

Chemical irritants applied to the skin produce different forms of reaction according to the intensity of the irritant. The reactions also vary considerably in degree according to the sensitiveness or susceptibility of the subject to varying irritants.

Recently a great deal of attention has been directed to the subject of "sensitiveness," or "allergy," of the skin. It is recognised that sensitivity may be congenital or acquired, and also that it may be specific for certain substances, or more or less general. For instance, certain individuals are congenitally sensitive to the "*primula obconica*," or Chinese primrose, and whenever they come in contact with this plant a dermatitis will result. On the other hand, persons who are not sensitive to this plant can be made so by rubbing the leaf into scarifications made in the skin, or by injecting into the skin, in appropriate doses, the active principle which has been isolated by Bloch. These are examples of congenital and acquired specific sensitiveness respectively.

General sensitiveness is less well defined, but again may be congenital or acquired. In this connection, congenital peculiarities of the skin, such as xeroderma, have to be considered.

If an intense irritant, such as a mustard plaster, is applied to the skin, the whole area to which it is applied becomes intensely red, owing to congestion of the papillary vessels; the epidermis becomes œdematous, and if the action is prolonged, small vesicles or even large bullæ develop under the horny layer. Lesions of this type are spoken of as *erythematous eczema*.

If a milder irritant is applied it may only attack the follicles, which are the most vulnerable part of the protective mechanism of the skin. In this case small follicular papules are produced, which in the more acute cases are surmounted by a small vesicle. These papules tend to group together on an erythematous base forming circumscribed patches. These are the *papular* and *vesiculo-papular eczemas*. With some irritants—croton oil, for example—follicular pustulation may also occur. The pus in this case is sterile, and the lesions dry up when the irritant is removed. This constitutes *pustular eczema*.

Lastly, there is a type in which the primary lesion appears to be produced by cracking of the horny layer. It is seen, for instance, on the face in children who dribble, and on the hands of those who use soap and water to excess. Under these conditions the horny layer becomes sodden with water, and this takes place more readily if alkalis, such as soap, are present. Then the skin dries quickly, especially when exposed to the wind, hot sun or a fire, and the horny cells tend to separate from one another, exposing the delicate mucous layer. In this type scaling first appears, followed rapidly by erythema. This is one form of *squamous* or *erythematous-squamous eczema*.

All these primary reactions are liable to undergo secondary changes.

Thus, fluid may exude from the surface from rupture of the vesicles and bullæ, producing a weeping eruption (*eczema rubrum*). Or, in the drier types, scaling may occur from irregular formation of the horny layer (this is the secondary type of *squamous eczema*). The moist cases may become infected with pus organisms, and the exudate may dry in the form of crusts (*eczema crustosum*). Thickening of the horny layer may occur, especially when the palms and soles are attacked, and this leads to cracking in the deeper folds of the skin (*eczema rimosum*). Occasionally in the lower extremities lymphatic obstruction and an overgrowth of the epidermis is produced (*eczema verrucosum*), or even elephantiasis may occur.

In order to classify these eczematous lesions a little more usefully it is advisable not to speak of a superficial inflammation produced by a known external irritant as an "eczema," but to call it a "dermatitis," qualified by the name of the irritant which causes it, as, for example, "formalin dermatitis." To use the word dermatitis without qualification is not so informing as to use the word "eczema."

For a large group of cases in which the external irritant cannot be traced, or in which it is one of those mild irritants to which every one is exposed, such as the air, or the friction of clothes, and also for certain cases in which the lesions are disseminated over the body, the term "eczema" is still used for want of further ætiological knowledge.

DERMATITIS FROM CHEMICAL IRRITANTS

These irritants are so numerous that it is impossible in a work such as this to attempt to give a complete description of them. They may, however, roughly be divided into the following classes: (1) Due to animal poisons; (2) due to plants; (3) due to chemical agents used in medicine; (4) due to chemical agents used in trades; and (5) due to decomposition of body secretions.

1. Superficial dermatitis due to ANIMAL POISONS is rare, most of the reactions being of the deep type, as they are injected by the stings and bites of insects. Some forms of caterpillar, *e.g.* the woolly-bear, however, occasionally produce an eczematous reaction.

2. PLANTS are probably responsible for more cases than are diagnosed. The *Rhus toxicodendron* or poison ivy produces the most marked symptoms, but this plant is rarely found in this country. The symptoms are an acute erythematous dermatitis, usually with much bullous formation, attacking chiefly the exposed parts, face and hands, but also affecting the moist parts of the body, the genitals, axillæ and flexures. Japanese lacquer, which is made from one of the *Rhus* family, may also give rise to a dermatitis in susceptible individuals. The commonest plant in this country to produce a dermatitis is the *Primula obconica*, but other species of the primula may attack susceptible individuals. The lesions are similar to those mentioned above, but much less severe. Among other plants which may produce a dermatitis are *Daphne mezereum*, oleander, rue, parsnip, daffodil, and chrysanthemum, while handling certain woods, such as teak, satin-wood and ebony, may produce similar effects. In all doubtful cases of "eczema" it is well to look for the presence of one of these irritating plants.

3. Of the CHEMICAL AGENTS USED IN MEDICINE some, such as cantharides, mustard, croton oil, chrysarobin and iodine, are used to produce varying degrees of dermatitis. Others may produce it unintentionally, among these being boric acid, iodoform, sulphur, carbolic acid and perchloride of mercury. Formalin, much used in pathological laboratories, produces a damaging effect on the horny layer, followed by a squamous and fissuring dermatitis. Sulphur, used in the treatment of scabies, is a common cause of an erythematous-squamous dermatitis on the flexor aspects of the limbs, on the abdomen and back, and is associated with intense irritation. Further, surgeons, students and nurses are apt to develop a dermatitis of the hands from the use of various antiseptics.

4. TRADE DERMATITIS.—This is very common, and the lesions produced are often specific. They are very numerous, for a description of the majority of which special works should be consulted. Among the more common are the soap and water dermatitis seen in washerwomen and in those engaged in household duties. This may take the form of a dry fissuring dermatitis on the back of the hands and forearms, or a papulo-vesicular dermatitis in the same situation. Its ætiology has already been discussed. Grocers and bakers are subject to a vesicular dermatitis of the hands, from handling sugar and dough: the so-called grocer's and baker's itch. French polishers, photographers, leather workers, etc., are frequently subject to dermatitis from articles used in their trades. These affections are usually of the papulo-vesicular type.

An acute erythematous dermatitis affecting the exposed parts of the body has been seen in those engaged in making explosives, also a more acute form caused by "mustard gas." The lesions in this case closely resemble those produced by poison ivy, the same distribution being observed.

Tar and oil acne.—Tar and various oils commonly give rise to a folliculitis, with a central keratotic plug like a comedo, associated with perifollicular inflammatory papules and pustules, and found on those parts of the body which come into contact with the irritants.

Grouped comedones.—In infants who have been rubbed with camphorated oil an eruption often occurs about the chest, neck and chin. The lesions are small black follicular plugs, closely placed, and are often associated with troublesome inflammatory complications.

Hair dyes.—An acute dermatitis of the face, particularly affecting the eyelids, is seen in persons who use certain hair dyes, especially those containing *paraphenylenediamine*.

Fur dyes.—Certain cheap furs, chiefly rabbit skin dyed with some of the phenylenediamine group of dyes, are responsible for a dermatitis involving the neck and chin. There is often a considerable latent period between the first wearing of the fur and the appearance of the eruption.

5. INTERTRIGO.—Decomposition of sweat and other body discharges may set up a dermatitis, usually of the erythematous type. This is best seen in fat women who are not too cleanly in their habits, the lesions being found under the breasts, in the folds of the abdomen and groins, and on the genitalia. A similar condition is often seen about the napkin region of infants. An erythema first appears, the horny layer of the skin, becomes sodden and is removed by friction of the parts, and a raw oozing surface results.

Jacquet's erythema infantum.—In some children an eruption consisting of pea-sized papules occurs about the prominences of the buttocks, thighs and abdomen under the napkin; the depths of the folds escape. Occasionally these lesions spread beyond the napkin area and frequently they are capped by a vesicle or crust. These cases appear to be due to ammoniacal urine and must be carefully distinguished from congenital syphilis. The condition is spoken of as "infantile erythema of Jacquet."

DERMATITIS FROM HEAT AND COLD

The erythema, followed often by blistering, as a result of a burn or scald, is well known. Similar but usually milder and more transient erythema may follow exposure to cold. The application of carbon-dioxide snow to the skin for purposes of treatment is a good example of the effects of extreme cold.

ERYTHEMA AB IGNE.—Frequent exposure to the fire produces a curious reticular erythema, followed by pigmentation.

ERYTHEMA PERNIO (CHILBLAINS).—Chilblains are frequently seen after exposure to cold. They occur chiefly in children and old people, and particularly in those whose peripheral circulation is sluggish. The lesions are salmon-pink to purplish, varying in colour, which occur chiefly on the fingers, toes and the lower part of the legs, and which itch intensely. The lesions may ulcerate and produce extremely indolent sores. When the helices of the ears are attacked, as they sometimes are in old people, considerable loss of tissue may result. The treatment for this condition is the same as for "Acrocyanosis" (see p. 1079).

TRENCH FOOT.—A somewhat analogous condition was met with in the trenches in France during the War of 1914–1918, in men who had to stand for long periods in the wet and cold. The feet became swollen and painful, the skin was reddened and blistering, and even necrosis occurred. The condition took a long time to subside.

DERMATITIS FROM ACTINIC RAYS

ERYTHEMA SOLARE.—Light, whether from the direct rays of the sun or from artificial sources, produces marked inflammatory changes in the skin. The sun's rays produce first a transient erythema which subsides, leaving pigmentation; but in other cases a more persistent erythematous dermatitis occurs, associated with oedema and thickening of the skin and scaling or blistering: this is the so-called "erythema solare."

TROPICAL SKIN.—Exposure to tropical sun for many years may lead to atrophy of the skin with pigmentation and warty formation.

PRURIGO ESTIVALE.—A rather rare condition occurs in some children in which it appears that the sun's rays are an exciting cause. It consists of the appearance on the face and hands of small very itchy papules which appear during the summer and disappear in the winter. They are not always limited to exposed areas, but these regions are always the most severely attacked. The condition is a very persistent one, and any treatment, except protection from the sun's rays, has little or no effect.

HYDROA ÆSTIVALE.—This is a much rarer condition than the preceding one and occurs in persons suffering from hæmatoporphyria congenita, though not in these cases only. The lesions are blisters, which appear on the parts exposed to the sun's rays, and scars are left when the blisters disappear.

X-RAY AND RADIUM DERMATITIS.—X-rays produce much more persistent forms of dermatitis than the sun's rays. If a slight overdose of X-rays is given, an erythema develops in the course of 1 to 3 weeks, which then gradually subsides. If a larger dose is given, the erythema may come on earlier and blistering may occur, which takes weeks to get well. Finally, in the still more intense burns, necrosis of the skin, with the formation of an extremely indolent ulcer, develops. In other cases, atrophy of the skin, with telangiectases and pigmentation, occurs, which may, after many years, break down into an indolent and painful ulcer, and this may in turn become epitheliomatous.

Radium may produce a similar series of changes.

ECZEMA

A description of the more specialised superficial dermatosis produced by mechanical and bacterial irritants will be left until those eruptions for which we reserve the term "eczema" have been discussed. The diagnosis and treatment of the dermatoses dealt with in the preceding paragraphs, together with that of eczema, will be considered at the end of this section. The reason for this is because it is probable that the lesions of eczema are produced, in part at least, by chemical, thermal and actinic irritants, and that pathogenic bacteria do not play an active part. Mechanical irritants do play a secondary rôle, and irritating chemical substances produced by saprophytic organisms are also concerned, but the latter are really chemical and not bacterial irritants.

Under the term "eczema" are included—(1) Certain cases of dermatitis, probably due to chemical irritants, the identity or nature of which has not been discovered. (2) Cases in which individuals are so susceptible to irritants that they react to mild stimuli that would not ordinarily be classed as irritants, such as a slight exposure to the sun, a cold wind, the warmth of a fire, or even to the friction of the clothes. (3) Cases which, having commenced with a simple dermatitis caused by an irritant, fail to get well on its removal and the patient becomes hypersensitive, so that fresh patches are produced, either in the neighbourhood of the original lesion or in other parts of the body. In fact, another factor is present which is spoken of as "sensitiveness."

Ætiology.—Many views are held as to the causes of this "sensitiveness." First there is the possibility of an inherited susceptibility or diathesis. This may be nothing more than some congenital peculiarity of the skin of which a recognisable form is xeroderma, already discussed. Abnormalities of secretion and of circulation alter the resisting power of the skin. So, probably, do certain toxic states, such as gout and rheumatism; deficient elimination, as occurring in nephritis; chronic infections, as in pyorrhœa and tonsillar sepsis; digestive disturbances and alcoholism. Disturbances of the nervous system, such as teething in infants, uterine troubles and the neuroses, may play a part.

More important, however, is the possible absorption of toxins from a local focus of dermatitis producing a hypersensitiveness or "allergic" condition, so that a violent local reaction occurs in the skin if the secretions from the original focus are brought in contact with other areas.

Pathology.—In eczema and superficial dermatitis the anatomical changes are most marked in the epidermis and papillary layers. There is congestion of the papillary vessels, with overgrowth (acanthosis) and œdema (spongiosis) of the mucous layer, and wandering cells may be present throughout the epidermis. Ballooning of the cell of the mucous layer occurs and minute vesicles appear. The horny layer is improperly formed, the cells retaining their protoplasm and nuclei, with the result that they shrink up on reaching the surface, from evaporation of water in the protoplasm, and so scaling is produced. This pathological condition is called *parakeratosis*.

Symptoms.—The general type of lesion found in eczema has already been described (p. 1397). It now remains to describe some of the common types of case met with, and this is conveniently done by referring to the regions of the body affected.

FACE AND SCALP.—Eczema of the face of a very definite type is frequently met with in infants. It usually occurs in the first year of life, and is more frequently met with in the winter months. It begins with a red irritable spot on one or both cheeks, and spreads fairly rapidly, so as to involve the whole face and often the scalp. In the more severe cases lesions are found on other parts of the body. The initial lesion is a red swollen patch or a group of follicular papules which later develop into an eczema of a scaly or weeping type. Itching is always intense but paroxysmal, the child rubbing the parts violently with the hands or against the pillow. In the moist variety septic infection may occur and thick yellow-green crusts form on the surface. Infantile eczema is often very resistant to treatment and may last from six months to a year, even under careful treatment; it usually dies out, however, during the second year of life.

This type of eczema occurs about three times as frequently in males as in females, and is often followed later in childhood by a condition frequently known as "flexural eczema," which is dealt with below.

Another type, also seen in infants, commences on the scalp, usually as a scaly or crusted patch, which becomes eczematized, usually as a result of scratching, and tends to spread down to the forehead and face.

In slightly older children a generalised eczematous condition of similar distribution, but of a septic type, is seen in association with nasal and aural discharge. Small follicular pustules are often present, and blepharitis is common. This is really an eczematized impetigo—that is, a direct bacterial infection, and not of the same nature as the first named, which is non-bacterial in origin.

Another common form of eczema met with on the face in children consists of sharply circumscribed scaly patches, always dry and with a surface like crêpe. These patches occur in the region of the mouth and nose, and can generally be traced to dribbling, running at the nose, the habit of licking the lip or smearing the face with a wet finger, or to the use of strong soaps. Some cases, however, are of streptococcal origin (p. 1423). The condition is produced by rapid drying of a sodden horny layer as described above, and goes by the name of "pityriasis simplex."

In adult life, especially in oldish people, an acute erythematous eczema of the face is apt to develop. It usually appears quite suddenly. The whole face becomes acutely red and swollen; the œdema of the eyelids often being so great as completely to close the eyes. In severe cases blistering may occur; but usually the acute œdema subsides and scaling ensues. This stage may either clear up completely or a chronic eczema characterised by redness, thickening of the skin, and scaling may follow. These acute cases are often associated with a similar condition of the hands and forearms, in fact the parts exposed to the air are most likely to be affected, and this condition is particularly prone to occur in cold weather. In some cases also a history can be obtained of a coexisting septic dermatitis elsewhere, frequently on the leg, and in these cases it is possible that some absorption from this has rendered the patient susceptible. Once a patient has had an attack he is always liable to recurrences, and great care must be taken to avoid exposure to extremes of temperature. Cases of this type may be limited to the eyelids, and a troublesome and chronic condition develops.

Occasionally the papulo-vesicular type of eczema is met with on the face; it is not infrequent on the forehead, generally in men under the hat-band, and often occurs in those who perspire freely.

Eczema of the scalp is usually of bacterial origin. This also applies to the ears (see *Seborrhœic Dermatitis* p. 1427).

"**FLEXURAL ECZEMA.**"—This is a well-recognised condition, which occurs usually in children, but may continue into adult life, and occasionally commences after puberty. It is a very specialised condition, and has been variously named "*Besnier's prurigo*," or "*flexural prurigo*." It frequently follows facial eczema in infants, and is also frequently associated with asthma. These three conditions form a syndrome and are manifestations of an underlying congenital condition named by Czerny the "*exudative diathesis*." The lesions are those produced by friction, and vary from moist eczematous patches to patches of chronic lichenification. The areas affected are chiefly the flexures of the elbows and knees, less frequently the backs and fronts of the wrists, the back of the hands, the sides of the neck and the face. Other parts of the body may be affected, and an extensive eruption is sometimes present. The condition is essentially a curious form of pruritus, the cause of which is not yet established. Experiments made with a view to demonstrating protein sensitiveness are still inconclusive. In the majority of cases the condition tends to die out before puberty.

UPPER LIMBS.—The hands and forearms, also being exposed to the weather, are subject to eczema. The erythematous type frequently complicates that of the face, and runs a similar course.

Papulo-vesicular eczema of the backs of the hands and the forearms is very frequent. It is generally produced by external irritants, whose nature can often not be determined. The lesions usually consist of rather sharply circumscribed red patches covered with numerous vesicles which rupture and leave oozing, pitted raw areas of the size of a pin's head. Sometimes the whole patch is considerably swollen with œdema. There is a great tendency in this type for fresh patches to appear in the neighbourhood, and even on distant parts.

A chronic form is sometimes met with in the palm, chiefly along the deeper folds. It begins as an ill-defined red patch, and subsequently marked

thickening of the horny layer takes place. Owing to its inelastic nature skin cracks and deep fissures are produced, which are very painful and very chronic. This type is spoken of as *eczema rimosum*.

An acute vesicular form is also seen on the hands, chiefly on the palms and sides of the fingers, especially in people whose hands perspire freely. Owing to the thickness of the horny layer on the palm, these vesicles are very deep-seated, and appear like sago-grains in the skin. At first they do not rupture, but run together and form large blebs which can often be seen to be purulent. The attacks usually come on quite suddenly, and the feet are often attacked simultaneously; they occur chiefly in the hot weather when sweating is profuse. This condition is called *dysidrosis* or *cheiropompholyx*, and was originally thought to be due to obstruction of the sweat ducts, with the formation of retention cysts. This is now known not to be the case, and that the vesicles are produced by an inflammatory exudate. It is probable that the skin is made sodden by excessive sweating, and this renders it susceptible to the attack of some external irritant. A similar condition has been observed from handling aurantia, a substance used in explosives, which shows that the condition may be produced by an external irritant. A number of these cases are due to fungus infection, the so-called "eczematoid ringworms" of the hands and feet (see p. 1437).

TRUNK.—Eczema on the trunk is nearly always secondary to patches starting elsewhere, if seborrhœic dermatitis and dermatitis due to irritants such as sulphur are excluded. There is, however, one type to which reference should be made. In people who sweat much, and especially in infants, an eruption of small vesicles, each surrounded by an inflammatory zone, sometimes appears on the trunk. The lesions appear to be formed around the sweat duct openings. This condition is called *miliaria rubra*, or *prickly heat*, and is probably of the same nature as the vesicular eczema of the hands, the mouths of the sweat follicles being softened by the sweat and some irritant, possibly a bacterial irritant, causing an inflammation.

The nipples are sometimes the seat of an eczema; but this is nearly always of external origin, either from careless treatment during suckling, or from injury from stays.

GENITALS AND ANUS.—The moist areas of the genital and anal regions are liable to be attacked. These are not infrequently secondary to a pruritus, a traumatic dermatitis being produced. On the anus and vulva, thickening of the skin, called *lichenification*, is most common, and has been already mentioned. The scrotum is occasionally the seat of an erythematous dermatitis very distressing to the patient, and very intractable. A considerable number of cases of eczema in the genito-crural region are due to infection with fungi, or yeast-like organisms, and in order to exclude these a careful microscopic examination of the scales should be made (see p. 1436).

LOWER LIMBS.—A special form of eczema is very common on the lower part of the legs. It is associated with chronic vascular stasis. It is generally met with in middle-aged or old people, but is frequently seen in younger persons who suffer from varicose veins, hence its designation *varicose eczema*. It begins either from an infected abrasion which does not heal, or from scratching an itchy leg. Once started the inflammation spreads, as the condition of the skin does not favour resolution. The extension is often due to ill-devised dressings which further lower the resisting power of the skin and

favour the retention of discharges. An extensive weeping or crusted dermatitis, therefore, follows, and this is rarely confined to the leg on which it starts, the other soon becoming infected, probably from contact in bed. Owing to the poorly nourished condition of the skin, ulceration is very prone to occur, and thus the chronic varicose ulcer, so familiar to the out-patient department of any hospital, develops. These cases are particularly prone to spread to other parts of the body by the mechanism referred to above.

The feet are subject to the same type of acute vesicular eczema as the hands.

THE NAILS.—The matrix of the nails may be involved in an eczematous process affecting the hands and feet, and may either show a marked irregularity of growth, with roughening of the surface of the nail, or the nail-plate may be pushed up from the nail-bed by parakeratosis beneath.

Diagnosis.—Keeping in view the types of eczema already described, the diagnosis of the lesions should present little difficulty; to determine the cause, however, is not so easy. Efforts must, however, first be directed to try and discover an irritant, and if it cannot be found, or if it appears to be one of those mild irritants which do not normally produce a skin reaction, the cause of the patient's susceptibility must be investigated. These causes have already been discussed and require no repetition. As to the nature of the irritant, some help is obtained by the type of reaction and by its distribution; for instance, in an acute eczema affecting the face and hands, exposure to the wind or sun or to some irritant, as the primula, is suggested. With trades certain parts of the body tend to be especially exposed. Lesions affecting the exposed parts and the moist parts of the body suggest some strong volatile irritant, such as rhus poisoning.

Acute erythematous eczema has occasionally been mistaken for erysipelas; but the absence of a sharp line of demarcation, a slowly spreading edge and high fever, should render the diagnosis simple. Acute giant urticaria of the face is unassociated with redness or vesication. The lesions of erythema multiforme are smaller, more sharply defined, and deeper-seated.

The squamous forms have to be distinguished from seborrhœic dermatitis; this is often difficult, but the characteristic features of this latter disease will be considered later.

Ringworm of the glabrous skin tends to occur in circumscribed circular patches or rings, and the fungus can easily be found under the microscope. A special form occurring in the groins is characterised by its bilateral symmetry, its sharp spreading edge, and the presence of fungus in the scales.

Pityriasis rosea in extreme forms may lead to confusion, but the acute generalised onset, and the presence of some of the typical oval lesions, with a collarette of scales attached about a millimetre from the free edge, will usually settle the diagnosis.

Psoriasis is rarely confused, owing to its characteristic distribution on the extensor aspects of the limbs, its usual sharply defined patches, and the dry silvery scaling, seen even in the smallest papules. A few cases, however, of isolated patches made up of small aggregated psoriasis papules may be very difficult to distinguish from localised patches of squamous eczema.

The moist forms have to be distinguished from impetigo contagiosa. In

this disease, however, the vesicles are larger but rarely seen, while the presence of isolated crusted lesions of varying size, with little or no inflammatory zone surrounding them, is characteristic.

The vesicular eczemas of the hands and feet may be caused by a ringworm fungus. This should always be suspected in the chronic spreading cases, and must also be looked for in the acute cases. The diagnosis is made by finding the mycelium of the fungus in the walls of the vesicles—a task not always easy. A curious type of fissuring eczema between the toes is almost invariably caused by a ringworm fungus.

The eczemas found in the course of animal parasitic affections, such as pediculosis and scabies, will be dealt with later.

Prognosis.—This is always uncertain. Most cases of dermatitis due to an external irritant applied on a single occasion get well readily when the irritant is removed. Those caused by the repeated application of the same irritant, as in trade dermatitis, are apt to be more persistent, while recurrent attacks may be extremely troublesome. Once the skin has been damaged subsequent attacks are more common and more resistant to treatment.

Those cases in which some underlying susceptibility exists are always apt to be resistant to treatment.

Treatment.—**PROPHYLACTIC.**—This depends on the search for the irritant, and its removal. The latter is not always possible in case of trades; but much can be done to insist on scrupulous cleanliness. It must not, however, be forgotten that the use of strong soaps, soda and turpentine to remove traces of a man's occupation are often the cause of the dermatitis. In cases where these substances have to be used, by washerwomen, etc., the use of a cold cream or some glycerine preparation to replace the grease of the skin will prevent a good deal of trouble.

Eczematous subjects should protect themselves from the sun, cold wind, and heat of the fire.

LOCAL TREATMENT.—This applies equally to the cases of dermatitis due to known irritants, and to those we have labelled "eczema." The main treatment in the early stages, after removing the cause, is to protect the skin and to provide soothing application to allay the inflammation. The use of soap and water will generally have to be forbidden.

In the early and acute stages lotions are most suitable, grease in any form being badly tolerated. Calamine lotion can be applied frequently and allowed to dry on the skin, the powder it contains forming a protective dressing over the surface; it is best used in the acute erythematous and papulo-vesicular form in which there is not much oozing. In the weeping cases, lead lotion applied on linen and kept moist is more suitable; it forms an insoluble albuminate of lead which acts as a protective layer. If, however, much sepsis is present, it is well first to use a mild antiseptic, baths of 1 in 4000 potassium permanganate, or lotions of 1 in 4000 perchloride of mercury, or 1 in 1000 acriflavine being very suitable. If these lotions dry the skin too much 3 per cent. glycerin may be added.

As soon as the acute stage has subsided oily preparations are better. It is well to begin with one containing a considerable percentage of water, the lin. calaminæ (B.P.C.) or linimentum calcis being the type. Ichthammol, 2 per cent., may with advantage be added in most cases, and if the itching is severe 1 per cent. to 2 per cent. phenol. Later the water can be given

up and either pure oily preparations as *lin. calaminæ co.* (B.P.C.), or ointments used. These latter are not satisfactory if there is much discharge ; but this can be checked by painting the surface once every second or third day with 2 per cent. to 3 per cent. silver nitrate in *sp. æther. nit.*

Once the chronic stage has been reached pastes are the best means of applying medicaments. Zinc paste consists of zinc oxide, 25 ; *pulv. amyl.*, 25 ; *paraff. moll.*, 50 parts, and makes a firm dressing when spread on linen or lint. It not only affords good protection, but allows a certain amount of absorption to take place.

If the chronic cases do not respond to treatment stimulating preparations are required, and can be incorporated in the zinc paste. *Ac. pyrogall.*, 1 per cent. to 2 per cent. ; coal tar, 1 per cent. to 5 per cent. ; or oil of cade, 5 per cent. to 10 per cent., are useful, and where there is thickening of the horny layer 1 per cent. to 3 per cent. *ac. salicylic.* should be added. Chronic dry cases, and even moist ones, if not septic, often do well if painted with crude coal tar which is allowed to dry on. X-rays, 120 r ($\frac{1}{3}$ of a skin unit), repeated 3 or 4 times at weekly intervals, are extremely valuable in resistant cases, and cause rapid disappearance of the lesions.

In septic cases the crusts should be removed by warm oil or starch-poultices, and a weak mercurial or flavine lotion first applied, and afterwards a zinc paste containing 3 per cent. ammoniated or yellow oxide of mercury.

The gelatine paste of Unna is very useful in the chronic eczemas of the leg, after any sepsis has been removed by antiseptic dressings. Certain chronic eczemas of the legs do well when strapped with varicosan or elastoplast bandages recommended by Dickson Wright, and this method is particularly valuable when ulcers are present.

For facial eczema of infants, 3 per cent. crude coal tar in zinc paste, spread on a mask and continuously applied, is of great value ; or the special tar paste devised by White of Boston, U.S.A., may be employed. The same paste is the most satisfactory application in cases of flexural eczema of the non-infective type.

GENERAL TREATMENT.—The patient must be examined for any conditions liable to lower his general resistance. Septic foci, such as pyorrhœa, or tonsillar sepsis, should be removed. In the more acute cases it is advisable to put the patient on milk diet, and to keep him in bed. In the less severe cases a light diet, the avoidance of alcohol, strong coffee and tea, hot and highly seasoned dishes, shell-fish, salted meats and cheese, should be prescribed. Constipation should be dealt with, while intestinal fermentation may be met by the exhibition of salol or bismuth salicylate, grs. 10 to 15 ; *ichthammol*, min. 2 to 5 ; or menthol, gr. 1 in capsules three times a day. In gouty subjects alkaline waters and colchicum are indicated.

In the infantile facial cases, the children are usually overfed and some reduction in diet is often required.

In debilitated cases cod-liver oil is of value, while arsenic and iron are helpful when anæmia is present. In acute cases *vin. antimoniale*, min. 5, t.d.s., has been much recommended.

Sleep is often disturbed, and will require sedative drugs to allay itching, and in the worst cases hypnotics : bromides are useful for the former, while for the latter sulphonal, methylsulphonal and chloral hydrate are among

the best. Morphine should be avoided, owing to the prolonged nature of the cases and to its tendency to increase itching.

Desensitisation.—As has been noted above, of recent years it has been realised more and more that many cases of eczema are dependent on the sensitiveness to specific irritant. The offending substance can sometimes be determined by the reaction produced when it is applied to the skin, or in doubtful cases a series of substances can be applied under pieces of strapping and can be introduced by puncture or scarification, all tests being carefully controlled. In the so-called “patch test,” when the offending substance is applied under strapping, a local eczematous reaction appears; when puncture or scarification is employed, a wheal is produced.

If the cause of sensitiveness is thus discovered it is possible, in some cases, to desensitise the patient by injecting intradermically an extract of the offending substance in minute and gradually increasing doses. Further, in cases where no specific substance can be determined, it has been found possible to desensitise patients by the injection of non-specific protein substances.

A method much in vogue at the present time is to withdraw 5 to 20 c.c. of blood from a vein of the patient and to inject either the whole blood or the serum from it into the gluteal muscles. Another method is to inject 5 to 10 c.c. of sterile milk on several occasions, at 2 to 3 days' interval, intramuscularly. Peptone is also used by some, and may be given either intravenously or intramuscularly.

DERMATITIS FROM MECHANICAL IRRITANTS

Acute dermatitis due to mechanical irritation is best seen in the redness and blisters found on the hands after rowing or on the seat after riding, in those unused to these exercises. The chronic form shows itself as a thickening of the horny layer as seen in the callosities on the hands and feet. The form of dermatitis of mechanical origin, however, which requires special attention here is that produced by the fingers and finger-nails.

1. SCRATCH ERUPTION

Constant friction applied to a localised area produces changes in the skin of a characteristic type. The skin becomes thickened and loses its elasticity; the folds and lines are much exaggerated, and the angular areas of skin intervening become prominent and shiny, resembling the papules of lichen planus; the colour may be the same as the normal skin, or red, but generally purplish, and sometimes the surface is finely scaly or warty. In old-standing cases much brown pigmentation may be present. This condition is spoken of as “*lichenification*,” and is seen at its best in localised pruritus, already described on p. 1403.

When general irritation is present the scratch lesions are more diffuse. The finger passing over the skin causes contraction of the *arrectores pili* muscles and the follicles are erected; the next sweep of the finger-nail scrapes the top off the erected follicle and a spot of blood appears, which dries as a blood-stained crust. In bad cases, linear excoriations are produced,

consisting of a line of blood-stained crusts. If sepsis supervenes, typical impetigo contagiosa lesions are produced, and these are particularly common in children; in other cases "eczematisation" occurs—that is, the inflamed papules group together to form a patch or patches, which may be dry and scaly or may weep. Patches of lichenification may also be found mixed with other scratch lesions, while in the most severe cases, ecthymatous lesions, boils and linear ulcers may occur.

2. CALLOSITIES AND CORNS

These are localised overgrowths of the horny layer, the result of local mechanical irritation. A corn differs from a callosity in that the central portion shows a much greater degree of overgrowth than the periphery, and forms an inverted horny cone which presses on the sensitive dermis, producing much pain. A corn may develop from a callosity, but frequently arises independently.

Symptoms.—*Callosities* are seen as a painless thickening of the horny layer over the ball of the foot and on the palms of the hands, in the latter situation especially in manual workers. They may also occur in other situations.

Corns may be of two kinds—(1) the hard and (2) the soft. The former are painful, horny elevations, chiefly seen on the feet, and especially in people who wear badly fitting boots. The common sites are on the dorsal surface of the little toes and on the plantar surface of the great toe and over the head of the first metatarsal bone. If the surface layers are removed with a razor a central "core," often stained black or dark brown from hæmorrhage, will be seen.

Soft corns are found on the lateral aspects of the toes in the interdigital spaces. They are usually lentil-sized raised swellings, covered with sodden epidermis, and intensely painful. Soft corns are invariably found associated with interdigital ringworm.

Treatment.—*Callosities* require no treatment. The principal point in the treatment of corns is to remove injurious pressure; this can be done firstly by fitting suitable boots, and secondly by taking pressure off the corn by wearing a ring of spongiopiline around it. The surface horny layer should be pared down with a sharp knife or razor and 10 per cent. salicylic acid plaster applied or salicylic acid collodion painted on, the softened horny layer being removed daily. Soft corns are treated as for interdigital ringworm (see p. 1438).

DERMATITIS ARTEFACTA

This is the name given to self-inflicted lesions of the skin. These are usually found in hysterical individuals, who produce them in order to induce sympathy, or in persons who are endeavouring to exact compensation or to avoid some unpleasant duty.

Symptoms.—The lesions are produced by various means, such as friction, the application of strong acids, or alkalis, or of blistering fluid, by heat or by the aid of some sharp instrument. All stages from simple erythema to actual destruction of the skin may occur. They may be single or multiple,

but are found on parts of the body easily accessible to the hands, and especially to the right hand (in left-handed people to the left hand). The lesions are very characteristic, especially those in which a liquid agent has been used. They have very sharp edges and the outline is angular, unlike that seen in any ordinary skin eruption; and not infrequently irregular patches near the main lesions have the appearance of having been produced by a spilt liquid. In addition, it may be noted that in the case of the malingerer the artefact may simply consist of keeping open an already existing lesion.

In hysterical cases anæsthesia of the palate has been frequently noted.

Treatment.—For effective cure the patient must be kept under observation, and caught in the act of producing the lesions. This may put a stop to further activities. Otherwise, occlusive dressing and mental treatment are required.

DERMATITIS FROM BACTERIAL IRRITANTS

Many different organisms are capable of producing dermatitis of external origin, and the eruptions produced are usually characteristic of the organism causing them. These will be described under the organisms concerned.

1. PYOGENIC INFECTIONS

It is not always possible on clinical examination to determine whether a given lesion is produced by the streptococcus or the staphylococcus. It used to be held that the superficial infective vesicular lesions were due to the streptococcus, while the follicular pustular lesions were of staphylococcal origin. While this appears to be true for the latter, it is now recognised that certain vesicular lesions may be of staphylococcal origin.

(a) IMPETIGO CONTAGIOSA

Symptoms.—This is an affection chiefly seen in children. It affects mainly the exposed parts, such as the face and hands. The initial lesion is a small pea-sized clear vesicle, which, owing to its superficial position between the horny and mucous layers, has an extremely thin wall and ruptures very easily. Before rupture, however, the fluid often becomes turbid, and if cultured in this condition contains both streptococci and staphylococci. If cultured, however, in the very early stages, pure growths of streptococci may usually be obtained. Once ruptured, fluid exudes freely from the base of the blister and dries as a crust. The crusts vary in thickness and character according to the amount of secondary infection, being thin and amber-coloured if little secondary infection is present, but thick and greenish if it is considerable. Usually the lesions are numerous; they are asymmetrical and obviously spread by local inoculation.

When the lesions occur in folds, such as at the angles of the mouth or nose and behind the ears, a troublesome fissure is likely to form, and generally crusting is absent, the fissure being surrounded by a moist, sodden, red area.

The disease is very contagious and children inoculate one another freely, any slight abrasion being sufficient to allow the entrance of the infecting

organism. One particularly common cause is pediculosis capitis, and in this case the scalp is usually first affected. In all cases of impetigo of the scalp or back of the neck, search should be made for pediculi.

Occasionally the blisters do not rupture early, but spread centrifugally, flattening down in the centre as they progress, and leaving a ring-like bullous margin (*impetigo circinata*). In other cases a large number of bullous lesions appear very rapidly, with little or no crust formation (*impetigo bullosa*). *Staphylococcus aureus* can usually be grown in pure culture from cases of these types.

Any of these varieties may occur in adults, but the crusted form has generally smaller crusts than in children. One of the most frequent areas to be affected in adults is the beard region, and impetigo contagiosa is one of the forms of so-called "barber's rash."

Diagnosis.—This is usually easy. The presence of scattered crusts, with little or no surrounding erythema, and the occasional small, very thin-walled blister, and an asymmetrical distribution chiefly on the exposed parts, is unlike any other condition.

Treatment.—In most cases the treatment is easy. The crusts should be removed by bathing in warm water, by fomentations or by warm olive-oil compresses, or in bad cases by starch poultices, and the raw surface covered with 2.5 per cent. ammoniated mercury ointment. In most cases it is advisable to incorporate the ammoniated mercury in Lassar's paste and spread this on lint and tie it on. The acutely spreading bullous form is treated in the first instance by pricking the blisters and dressing with 1 in 1000 acriflavine lotion. In very resistant cases, injections of mixed strepto- and staphylococcal vaccine have proved useful, but are rarely required.

(b) PEMPHIGUS NEONATORUM

Synonym.—*Impetigo Neonatorum*.

This is a form of bullous impetigo seen in newly-born infants and is characterised by the presence of varying-sized blisters on the skin.

Ætiology and Pathology.—*Pemphigus neonatorum* has precisely the same cause as *impetigo contagiosa*, but produces its characteristic features on account of the ease with which the horny layer separates from the underlying mucous layer in small infants. Infection is usually conveyed on the fingers of the mother or nurse. Pure cultures of *Staphylococcus aureus* can usually be obtained from the bullæ in their early stages.

Symptoms.—The eruption usually appears in the first few days of life. A clear blister appears, which rapidly increases in size, and others soon occur in the neighbourhood. There is little or no tendency to crust formation, though the blisters frequently rupture, the raw surface being protected by the loose blister wall which lies over it. Blisters vary in size from a pea up to a florin or larger, and in severe cases may be very numerous, covering practically the whole surface of the body. The lesions may commence on any part of the body, but are frequent about the napkin area. In the most severe forms the horny layer is so rapidly separated over large areas of the body that blister formation is not an obvious feature. This variety is known as *dermatitis exfoliativa infantum* or "Ritter's disease," and ends fatally in a large proportion of cases.

Diagnosis.—The pemphigoid syphilide must be distinguished from pemphigus neonatorum. In the former condition the eruption is symmetrical, is chiefly found on the prominences of the buttocks, on the palms and soles; other symptoms of syphilis are present, such as wasting, snuffles, fissures at the angles of the mouth, and other syphilitic skin eruptions. A Wassermann reaction in doubtful cases settle the diagnosis.

Prognosis.—Mild cases respond rapidly to treatment, but in the more rapidly spreading cases the prognosis is always grave.

Treatment.—The bullæ should be opened and their contents absorbed with cotton-wool. Strips of lint soaked in 1 in 1000 acriflavine lotion should be applied, and changed three times a day or more often if necessary. It is important that the blister edge should be removed by forceps, so that the lotion may act on the spreading edge of the lesions. The child should be well wrapped up to prevent loss of heat.

(c) ECTHYMA

In this condition local gangrene of the skin occurs and an ulcer, surrounded by a deep inflammatory zone and covered by a crust, is produced. The lesions are not always of pyogenic origin, but may be brought about in various ways; but as they have some resemblance to impetigo contagiosa, it will be well to describe them here.

Ætiology and Pathology.—The type seen in children is often of streptococcal origin and begins as an impetigo. Scratching or a debilitated condition of the patient allows of a more violent reaction, and necrosis occurs. The frequency with which ecthyma is associated with urticaria papulosa, scabies and pediculosis points to trauma as an ætiological factor. The large round adult type, referred to below, is nearly always preceded by a boil, which is a staphylococcal infection, while the linear type can be shown to be produced by violent scratching, to which is added secondary pus infection.

Symptoms.—All varieties are most often seen on the legs and buttocks. The lesions are usually discrete and few in number, but there are exceptions. They have the appearance of impetigo contagiosa lesions, but there is a wide congested area around the crusts, and these latter are not "stuck on" but firmly fixed. On removal an ulcer the size of the crust is found. This type is usually found in children. Another variety is seen in adults especially, in association with pediculosis vestimentorum, and was seen very frequently during the War of 1914–1918. Two types are seen: the large circular type, which has the characters of those mentioned above, but the individual lesions are larger, and the linear or gutter-shaped type, in which long ulcers, often 2 or 3 inches in length and covered with a thick crust, are present.

Diagnosis.—This has to be made from the ecthymatous syphilide, usually a late tertiary manifestation. In this condition there is a tendency to grouping of the lesions, and they are of a more chronic type. Other syphilitic manifestations, a positive Wassermann reaction, and rapid response to anti-syphilitic remedies will settle the diagnosis.

Treatment.—Local treatment is similar to that of impetigo contagiosa. The crusts should be removed by baths or starch poultices, and Lassar's paste with 3 per cent. ammoniated mercury tied on. In the adult cases, after

the sepsis has been removed by 1 in 4000 perchloride of mercury or 1 in 1000 acriflavine dressings, Unna's paste should be applied and changed every 2 or 3 days until healing takes place. Where debility and malnutrition are present, suitable internal treatment must be resorted to, cod-liver oil and malt, and the preparations of iron, arsenic and the phosphates being most useful.

(d) PITYRIASIFORM DERMATITIS

Certain forms of circumscribed, dry, superficial dermatitis, with fine branny scales, are sometimes seen in association with impetigo contagiosa and appear to have the same origin. In fact, all stages between the two conditions can be traced. The name *impetigo pityroides* is sometimes applied to this type of case. In other cases dry scaly patches are found without any impetigo contagiosa lesions, and streptococci have been isolated from them. These cases are sometimes indistinguishable from the scaly patches which occur on the faces of children, and which are described in the section on Eczema (p. 1412), where it is suggested that moisture and soap play the chief part in their production. It would thus appear that the streptococcus may produce lesions clinically identical with those produced by these physical and chemical causes. It has been thought also that some forms of circumscribed scaly dermatitis found about the neck and trunk, and also in the flexures of the limbs, and which have in the past been loosely grouped as seborrhœic dermatitis, are probably of streptococcal origin, but further investigation is necessary in order to group them clearly.

These lesions are often resistant to treatment. They are frequently associated with a good deal of itching, and are often followed by secondary changes due to friction, namely, "lichenification" and "eczematisation."

Treatment.—The early cases sometimes respond well to applications of dilute ammoniated mercury ointment; others, however, do best on ac. salicyl., grs. 15; liq. picis carbonis, min. 15; past. zinci ad 1 ounce. Once a condition of lichenification is established, the treatment should be on the lines laid down for local pruritus (p. 1403).

(e) FOLLICULAR IMPETIGO OF BOCKHART

This is the name given to a superficial pustular eruption of staphylococcal origin seen in connection with the hair follicles.

Symptoms.—The lesions consist of small beads of pus situated quite superficially at the mouths of the hair follicles, each being surrounded by a narrow red zone. Generally the hair can be seen penetrating the centre of the pustule. There is no tendency for the lesions to run into one another, each remaining quite distinct. Usually groups of them occur in localised areas, but sometimes their distribution is very extensive, cases occurring in which almost every stout hair is surrounded by a pustule. The most frequent sites are the fronts of the thighs, the legs, the genitals and the backs of the forearms. A very troublesome variety is seen on the scalp of children between the ages of 2 to 5, the infection being usually derived from a discharging ear or nose. The whole scalp is affected, and the condition is combined with a superficial septic dermatitis which affects also the face, and often spreads to other parts of the body. Ciliary blepharitis is a frequent complication.

This is the condition which was formerly described as *pustular eczema*. The majority of localised cases occur in conjunction with other forms of pyoderma; they are seen in scabies and pediculosis, and also in people suffering from boils.

Treatment.—The general health of the patient must be attended to, and all local foci of sepsis dealt with on surgical lines. A search for parasitic infestation must be made and appropriate measures adopted. In the localised cases, the pustules should be punctured, and 1 in 1000 acriflavine lotion applied; if this proves too irritating, lead or calamine lotion should be used, the accumulation of powder from the latter being bathed away daily with a weak alkaline lotion. Mercury lotions are better avoided, as they tend themselves to produce follicular pustulation.

The more extensive cases are very resistant to treatment. Shaving the affected areas, followed by the application of mild antiseptic lotions or sedative lotions and alkaline baths, is sometimes effective. Staphylococcal vaccines should be tried in addition, and in some cases stannoxyl (an oxide of tin) and injections of collosol manganese have given good results. In the pustular eczema of the scalp, the crusts should be removed with warm oil or starch poultices, and the lotions indicated above or acriflavine 1 in 1000 in liniment. calcis applied. At the same time nasal and ear discharges must be appropriately treated. These cases take a considerable time to cure, but the results repay the attention necessary.

(f) FURUNCLE

Boils or furuncles are deep-seated infections of the hair follicles with the *Staphylococcus aureus*.

Ætiology and Pathology.—The exciting cause of a boil appears to be the presence of virulent staphylococci in the hair follicles which occasion an intense reaction sufficient to cause local necrosis. This is the more liable to occur where the skin is thick, owing to the pressure exerted on the dense fibrous-tissue bundles and the consequent obstruction to the circulation. Scratching, which conveys the causative organism to the follicles and damages their orifices, predisposes to boils, as is seen by their frequent occurrence in parasitic affections. Lowering of tissue resistance, such as occurs in diabetes and in other conditions of lowered vitality, is also a predisposing cause. In other cases it is probable that a condition of allergy and hypersensitiveness to the staphylococcus is present, and this probably accounts for the constant recurrences which occur.

Symptoms.—Boils may attack any part of the body where hair follicles are present, but are most commonly seen on the neck, back and buttocks, regions where the skin is thick and exposed to pressure and friction. The lesions are usually single or few in number, but they tend to recur with great persistence, and recurrences may continue for a long period. The patients attacked are often in a low state of health. Boils are particularly liable to occur in diabetics, and the urine of patients should always be examined for sugar. A boil commences as a deep, tender infiltration, which rapidly increases in size so as to form a painful red swelling, up to an inch in diameter, which projects above the surface of the skin. Later, a small pustule appears in the centre of the swelling and this eventually bursts, exuding a small quantity of pus, which relieves the pain. Later still, a small

slough separates from the centre of the swelling, and when this has come away the boil heals, leaving a pitted scar. Some boils, however, subside without bursting. Sometimes the earliest lesion is a superficial pustule, which is followed by the rapid formation of an inflammatory zone. Later, infiltration of the deeper tissues follows, and a slough forms and is discharged, as in the first-mentioned type.

Treatment.—The general condition of the patient must first be dealt with on general lines. Certain internal remedies, such as Vitamin A, yeast and calcium sulphide, have occasionally proved of value. Vaccines and toxoid of *Staphylococcus aureus* produce results in some cases, but cannot be looked on as a specific. Injection of collosol manganese, or injection of manganese butyrate, suggested by McDonagh, beginning with 0.5 c.c. and repeated twice a week, in slightly increasing doses, for 3 to 4 injections, produces dramatic results in certain cases; others, however, are completely resistant. Stannoxyl, given by the mouth 2 to 3 tablets t.d.s., has also been claimed to produce excellent results.

Local measures are of great importance. Before the boils have ruptured, applications of kaolin poultice (antiphlogistine) are helpful in relieving pain and causing absorption of the exudate. Short-wave diathermy and small doses of X-rays may also produce a similar result. After rupture, dressings of 1 in 4000 perchloride of mercury, or pasta magnesiæ sulphatis (B.P.C.) are indicated. Boric acid fomentations should be avoided, as they spread the infection. Early or deep incision into boils is better avoided as it tends to spread the infection.

(g) CARBUNCLE

A carbuncle is a boil or group of boils in which the subcutaneous tissue has become involved in the infective and necrotic process.

Symptoms.—Usually only a single lesion is present. It may at the commencement appear like an ordinary boil, but the spread is rapid, and soon a large, red, indurated, painful area is produced. The lesion may attain a diameter of many inches. After a few days numerous points of pus appear on the surface of the swelling, and these burst and exude pus. The bridges of tissue between these openings may subsequently break down and reveal a large slough, which may take several weeks to separate if not removed by surgical means. Fever and other constitutional symptoms are generally present.

Treatment.—The general treatment is the same as for boils. Local surgical treatment may be required, and consists either of complete excision of the carbuncle, with the surrounding inflammatory tissue, or of making a crucial incision and removing the slough, the open wound being packed with bismuth-iodoform-paraffin paste or an appropriate antiseptic dressing.

(h) SYCOSIS BARBÆ

This is a staphylococcal infection of the hair follicles of the beard region, and is one of the three forms of "barber's rash," the other two being impetigo contagiosa of the beard region and ringworm of the beard.

Ætiology and Pathology.—The disease is produced by inoculation of staphylococci into the hair follicles in the beard area, either primarily or as a secondary infection to an impetigo contagiosa. Many cases doubtless start from infection conveyed in the barber's shop, but a considerable number also occur in patients who shave themselves. Scratches from the razor subsequently inoculated by the patient's finger are probably as common as direct infection from a barber's brush or razor.

Symptoms.—The disease usually begins at one spot in the beard or moustache area by the formation of pustules around the hairs. These pustules tend to occur in groups, and become surrounded by an inflammatory zone. As the deeper parts of the follicles become infected, nodules form and the whole affected area becomes swollen and œdematous. Pus discharging from the ruptured pustules dries and forms crusts. Subsequently some of the hairs loosen and can be pulled out without pain. Each hair on removal is seen to be surrounded by a swollen and transparent root-sheath, and often a bead of pus escapes from the follicle. The disease is progressive, and eventually the whole beard and moustache area, and not infrequently the eyebrows and eyelashes, may be attacked. It tends to become chronic, and in old-standing cases a large number of the hairs are lost, leaving a smooth, red, atrophied patch, not unlike lupus vulgaris, to which the name *lupoid sycosis* has been given. The presence of some follicular pustules and the absence of lupus nodules are, however, sufficient to separate the two conditions.

Diagnosis.—In addition to the diagnosis from lupus vulgaris just mentioned, sycosis has to be differentiated from impetigo contagiosa, and from ringworm. From the former the diagnosis is made by the involvement of the deeper structures of the skin and the presence of pus in the hair follicles; from ringworm by the absence of fungus in the scales and hairs (see *Tinea barbæ*, p. 1436).

Treatment.—The acute cases should be treated much in the same way as other acute inflammations of the skin, without any attempt being made directly to attack the organism responsible for the disease. Frequent bathing in warm water or oil should be used to remove crusts, or boric-starch poultices may be used. Lead lotion, or 1 in 1000 acriflavine lotion, should be applied on lint and changed frequently. When the acute stage has subsided the parts should be kept clipped short with scissors. Hairs from infected follicles should be epilated and the skin dabbed with 5 per cent. ichthammol in calamine lotion or 1 in 4000 perchloride of mercury. As an adjunct staphylococcus vaccines or toxoid may be given. The local application of a vaccine has sometimes proved useful. In some cases excellent results have been obtained by the use of "quinolor" ointment (Squibb). Epilation by X-rays has been recommended, but the results are not very satisfactory, recurrence being frequent. Small doses of X-rays are, however, helpful.

2. ANTHRAX INFECTIONS

These lesions, which resemble in some respects those produced by the staphylococcus, are dealt with elsewhere (p. 57).

3. DIPHTHERIA INFECTIONS

The lesions produced by the diphtheria bacillus are rare, but are seen sufficiently often to require notice. Diphtheritic infection of wounds is a well-known condition, but does not need to be considered here. In children suffering from diphtheria, gangrenous patches occasionally develop, chiefly on the trunk, from which cultures of the organism may be obtained, but the organism may also attack the skin of otherwise healthy persons.

Symptoms.—A single lesion usually occurs, but there may be more than one. It begins as a clear blister, like an impetigo vesicle, and ruptures very easily; on about the second day a considerable red zone is present around the original lesion, and a central slough has formed, comparable to a small burn. This condition persists for some time, if not treated, and eventually the slough separates and the spot heals. Constitutional symptoms may be present. The writer has recently seen a case of paronychia from which a pure culture of the Klebs-Loeffler bacillus was obtained.

Treatment.—Prompt injection of diphtheria antitoxin, with the application of a local antiseptic dressing, is all that is required.

4. INFECTIONS BY THE SEBORRHŒIC ORGANISMS

Three organisms are commonly found in cases of seborrhœic dermatitis, but the part each plays is not yet conclusively proved, so that it will be convenient to group the seborrhœic conditions under one heading. The organisms found are the *acne bacillus*, the *bottle bacillus* (pityrosporon of Malassez), and the *Staphylococcus epidermidis albus*. The first named is a small bacillus which is found chiefly after puberty and is present in very large numbers in the comedo of acne vulgaris. The bottle bacillus is a yeast-like organism which buds and often shows itself as a flask-shaped body, and is found most plentifully in seborrhœic dermatitis of the scalp. The white skin staphylococcus is found pretty universally over the skin.

(a) SEBORRHŒIC DERMATITIS

Under this term we include a chronic scaly condition of the scalp, formerly called seborrhœa sicca, and also certain "eczematous" lesions of the face, chest and back, and occasionally on other parts of the body, which are characterised by the presence of more or less circumscribed reddish patches covered by greasy scales.

Ætiology and Pathology.—The histological changes in the skin are those of a chronic superficial dermatitis. The three organisms mentioned above are met with in the scales in all adult cases, but the origin and spread of both seborrhœic dermatitis of the scalp and the figurate type on the body suggest that the views of Sabouraud and Whitfield, that the bottle bacillus is the chief ætiological factor, are correct. Further, the ease with which most lesions clear up under treatment by sulphur supports these views. There is no doubt that an underlying seborrhœa is the main factor in causing the activity of these organisms.

Symptoms.—Seborrhœic dermatitis of the *scalp* is the well-known

"scurfy head," and is an extremely common affection, most individuals having it to a greater or less degree. It probably begins in early infancy, and is sometimes seen as a ringed lesion on the scalp of young infants. These lesions have been shown by Whitfield to contain the bottle bacillus in large numbers. These rings may disappear spontaneously, but the infection, which has presumably been conveyed from the mother or nurse, persists and lights up again later in life in certain individuals, especially in those prone to seborrhœa (see p. 1400). In the adult the affection consists of a diffuse branny scaling on the scalp, usually unassociated with any obvious inflammation of the skin. On close examination the scales are seen to be formed around the hairs, indicating that the inflammation is perifollicular. Varying degrees of scaliness are met with; in some cases it is scarcely perceptible, in others it consists of thick, greasy masses. Symptoms are generally absent, but occasionally a good deal of irritation is present, which leads to scratching, and small crusted lesions are then found among the scales. Sometimes a more acute inflammation supervenes, and the scalp becomes red and hot, and an exudate of fluid may occur, producing crusting. In these cases the inflammation usually extends for a centimetre or so beyond the hairy margin. The persistence of scaly seborrhœic dermatitis is considered by some to be an ætiological factor in producing that form of baldness known as alopecia prematura, which is characterised by the recession of the hair from the forehead and baldness on the crown of the head. It is probable, however, that other factors, such as heredity, also play a part in this condition.

The *face* may also be affected, especially the eyebrows, forehead, nasolabial folds, beard and mastoid regions. Here the lesions are dry, reddish or pale patches, surmounted by greasy scales or crusts. On close examination it can usually be seen that the lesions are follicular in origin and that the patches are formed by the aggregation of these follicular papules. The ears may be affected, especially the retroauricular sulcus and the concha, and some forms of blepharitis appear to have a seborrhœic origin. The lesions on the face are very liable to become infected with pus organisms and become thickly crusted. An intractable scaly inflammation of the lips, *cheilitis exfoliativa*, is also considered to be of seborrhœic origin.

On the *chest and back* ringed or figurate lesions are frequently seen, but here follicular papules may occur. The centre of the sternum and the interscapular area are the common sites. Occasionally patches occur among the pubic hairs.

Some authorities include under this heading cases in which circumscribed pinkish or red circular or oval patches, covered by fine branny scales, occur on the trunk and limbs. They are resistant to treatment, especially to the remedies useful in seborrhœic dermatitis, and there is some evidence that they are forms of streptococcal dermatitis. This type is prone to attack the flexures of the limbs, chiefly the axillæ and groins, as are other streptococcal infections.

Diagnosis.—All cases of scurfy head in children should be considered to be ringworm until careful examination has excluded this cause. The presence of stumps and the demonstration of the fungus will settle the diagnosis. In body ringworm the distribution is irregular, the lesions are sharply circular, and the scaling is not greasy. Fungus can be found in the scales.

Impetigo contagiosa of the small crusted type has a close resemblance to seborrhoeic dermatitis, especially on the face. The presence of some definite impetigo vesicles and crusts, and the history of its unilateral spread, may help to clear up the diagnosis.

Pityriasis versicolor occurs in the same regions as the body form of seborrhoeic dermatitis, but it has a fawn colour and no inflammatory reaction, and the fungus can be found in the scales. Pityriasis rosea can be distinguished by the presence of oval lesions with a collarette of scales within the edge of the lesion, by its acute onset and by its symmetrical distribution.

Prognosis.—The figurate variety on the trunk can always be kept under by appropriate treatment, but frequently recurs. The face is more resistant to treatment, and when much septic infection has taken place may take a long time to cure. On the scalp constant treatment is necessary, and a permanent cure can scarcely be hoped for, as the organisms invade the follicles. With proper hygiene and appropriate treatment, however, the condition can be kept quiescent.

Treatment.—*Scalp.*—Frequent washing is necessary to remove the scales and accumulated dirt. Unless there is any acute inflammation present, men should wash the scalp two or three times a week or even daily with *sp. sapon. kalin.* (B.P.C.), or *ext. quillaie liq.* (B.P.C.); sulphur or tar soap may be used. In women the washing should be done once a week. After drying, in severe non-inflammatory cases, an ointment containing 3 per cent. each of *ac. salicylic.* and precipitated sulphur in a basis of gr. 120 soft paraffin and gr. 360 cocoa-nut oil should be rubbed into the scalp. Resorcinol, thymol, anthrasol, thiol or ammoniated mercury 3 per cent. may be used as alternatives or in various combinations. In the milder cases lotions are preferable. Resorcinol or chloral hydrate min. 60, *sp. vin. rect.* fl. oz. 1, *aquam ad* fl. oz. 8, is a useful lotion. Resorcinol should not be used in fair or white-haired patients owing to its staining properties. In the acutely inflamed cases, washing with soap should be avoided, though crusts may be bathed away with warm water. After removal of the crusts, *ichthammol* gr. 30 in 1 fl. oz. of *lin. calcis* should be applied, the hair being cut short if necessary.

On the *face*, sulphur and salicylic acid ointment may be used in the chronic cases; if, however, sepsis is present the crusts must be removed and calamine or *ichthammol* liniment applied.

On the *body*, sulphur and salicylic acid ointment is usually all that is required.

(b) ACNE VULGARIS

This condition is characterised by the presence of greasy plugs, known as comedones, in the pilo-sebaceous follicles—particularly those on the face, shoulders, chest and back—often associated with perifollicular inflammation. It is an extremely common affection in its milder forms and by no means rare in its severest types.

Ætiology and Pathology.—The disease occurs chiefly in individuals between 15 and 25 years of age, and is seen in both sexes. There can be no doubt that it is dependent on the development of the sebaceous glands which occurs at puberty, and that there is, in addition, an individual pre-

disposition, probably inborn, to develop the affection. The affected individuals suffer from seborrhœa.

If a comedo is examined it is found to consist of epithelial cells, sebaceous material, and the three organisms which are associated with seborrhœic inflammations and, in addition, a small acarus, the *demodex folliculorum*, is sometimes found. In the greater mass of the comedo, the *acne bacillus* occurs almost pure, the other organisms being found chiefly near the mouth of the follicle. Accumulated evidence seems to show that the *acne bacillus* is the chief exciting cause of the comedo, but that secondary suppuration may be due to the activity of staphylococci, though this has been denied by Sabouraud. The bottle bacillus and the *demodex* appear to play no active part in the production of the disease. The excessive oily secretion of the skin, with the patulousness of the follicles which accompanies this condition (the so-called "keroze" of Darier), offers a field for the activities of the *acne bacillus*, which flourishes in the sebaceous secretion. The actual comedo is formed by exfoliated epithelial cells—produced by an inflammatory hyperkeratosis of the follicle—mixed with sebum.

Symptoms.—The earliest lesions are the comedones or "blackheads." These are small, black spots which are seen filling the dilated orifices of the pilo-sebaceous follicles, most frequently on the face, but also in the other sites mentioned above. If pressure is exerted on a follicle, a cocoon-like plug can be squeezed out, which is of a cream colour, except for the portion which fills the mouth of the follicle, where it is black. Isolated comedones are extremely common, but when large numbers of them occur the term *acne punctata* is applied to the condition. Frequently, however, the presence of these follicular plugs predisposes to an acute perifollicular inflammation, and the comedo becomes surrounded by a red zone; later, a small pustule may occur in the centre. This lesion is generally painful. When these inflammatory lesions predominate, we speak of the case as one of *acne papulosa*, or *pustulosa*; but it must be noted that all varieties tend to be present together. In some cases the inflammation does not start superficially around the follicular orifice, but deeper, in the region of the sebaceous gland. Here we find first a deep-seated lentil- or pea-sized nodule, often painful, which gradually increases in size, reddening the skin as it pushes upwards, and then sometimes bursts at once and discharges a small quantity of turbid yellow fluid with the remains of the comedo; or it may attain the size of a filbert, and present signs very similar to those of a sebaceous cyst. Sometimes the nodules disappear without rupturing. This type is usually spoken of as *acne nodularis*, and is particularly liable to appear on the back. It is the most persistent type, and often leads to much keloidal scarring.

Diagnosis.—Rosacea, especially the acneiform type, when it occurs in young people, may sometimes be mistaken for acne vulgaris, and indeed the two conditions may occur together. In rosacea, vascular congestion is the prominent symptom; the lesions are generally localised to the centre of the forehead, the nose and central portion of the cheeks and the chin—they are painless and the comedo is absent.

Acneiform lesions produced by the internal administration of bromides and iodides may simply be an exaggeration of a pre-existing acne vulgaris; but if not, the lesions tend to be more grouped and to produce tumour-like swellings. Other eruptions characteristic of these drugs may also be present.

The acne produced by the irritation of tar and paraffin is usually localised to the forearms and lower limbs.

Prognosis.—The condition tends to die out between the ages of 20 to 30; but the nodular type may often continue till a considerably greater age. Though improvement is sometimes slow, treatment materially hastens a cure. Bad scarring is sometimes left in severe cases.

Treatment.—As the causative organism is situated deeply in the follicle, it cannot be reached by the ordinary anti-parasitic remedies. Treatment must, therefore, be directed to emptying the follicles. In the mildest cases this is best done by frequent washing with soap and hot water. This is followed by gentle squeezing massage over the affected areas in order to empty the grease from the follicles. Comedones should also be squeezed out, preferably with a comedo extractor. After this a mild sulphur preparation, such as calamine lotion containing 2 per cent. or 3 per cent. of potass. sulphurata or sublimed sulphur should be dabbed on, or if this dries the skin too much 3 per cent. precipitated sulphur in ung. aquosum may be used. Ointments, however, should be avoided as far as possible, as they tend to block up the follicles. This treatment must be persisted in for a considerable period. More drastic treatment consists in exfoliating the skin with a resorcinol paste or with the mercury-vapour lamp.

Vaccines have not given very satisfactory results, though staphylococcal or mixed acne and staphylococcal vaccines have been of some value in the pustular cases.

Very satisfactory results have been effected by means of X-rays. It must be remembered, however, that atrophy and telangiectases sometimes occur many years after treatment. It should therefore be reserved for the more resistant cases and especially those which are tending to develop scars, and should only be given by those who have special experience.

In addition to local treatment the patient's general health must be attended to, constipation rectified, and such conditions as dyspepsia, anæmia and menstrual disturbances treated. A low carbohydrate diet should be advised.

(c) ACNE VARIOLIFORMIS

Synonym.—Acne Necrotica.

An inflammatory condition of the hair follicles, accompanied by local necrosis, and leaving pitted scars resembling those seen in variola.

Ætiology and Pathology.—The disease is seen chiefly in middle-aged persons of both sexes. It is believed to be of bacterial origin, and is attributed by Sabouraud to the acne bacillus. It occurs in seborrhœic individuals.

Symptoms.—The affection occurs chiefly on the scalp and forehead, but is occasionally seen on the face, neck, chest and back. The lesions usually come out a few at a time, and the attacks may persist for long periods; but there are generally intervals of complete freedom. Often change of residence has the effect of stopping or determining an attack.

The lesions at the commencement are pinhead-sized vesicles situated at the mouth of the hair follicles. These increase in size to that of a lentil or pea. The vesicles rapidly dry up without bursting, and scabs are formed. These are seen to be depressed below the surrounding skin. When the

scabs fall off after a week or so a small punched-out scar remains. The onset is usually accompanied by a good deal of itching or burning.

Diagnosis.—The condition has to be distinguished from the scattered crusts which occur in seborrheic dermatitis of the scalp as a result of scratching. The diagnosis can be made by the pre-existing vesicular lesions in the case of acne varioliformis, and by the scarring left.

Treatment.—These cases are often resistant to treatment. The general health should be attended to, and often change of air is very beneficial. Local anti-seborrheic remedies should be applied, such as salicylic acid and sulphur ointment (3 per cent.), ammoniated mercury ointment (10 per cent.), or lotions of potass. sulphurat. and zinc sulphate.

(d) ACNE KELOID

A hypertrophic inflammatory condition occurring on the back of the neck just below the hair margin.

Ætiology and Pathology.—The disease occurs in young adult males. Very little is known of the cause. It occurs at a point where the collar rubs the back of the neck, and friction appears to play a part in its production. The condition has been studied closely by Adamson, who can find no evidence of previous comedo formation. Though he considers that it is produced by a combination of trauma and bacterial infection, he does not consider that the acne bacillus or the *Staphylococcus pyogenes* plays any part in its formation.

Symptoms.—The condition commences with small firm nodules, which gradually increase in size and eventually merge into one continuous mass, closely simulating a keloid.

Treatment.—Adamson recommends X-rays as the only satisfactory method of treatment.

DERMATITIS DUE TO FUNGI

1. RINGWORM

Ætiology and Pathology.—Tinea or ring worm is the name given to certain inflammatory affections of the skin produced by the growth in it of certain of the hyphomycetes or moulds. These fungi grow for the most part in the horny layer of the epidermis or its appendages, the hairs or nails, and by their growth produce an inflammatory reaction. The fungi which are commonly seen in this country belong to three genera—the *microsporon*, the *trichophyton* and the *epidermophyton*, the latter of which is characterised clinically by not attacking the hairs. To these must be added a fourth, which, though of the same family, is not usually included under the term "ringworm," namely *favus*, the fungus of which belongs to the genus *Achorion*. The fungus of ringworm is transmitted to man either from another human being or from certain animals, some fungi being only found in man. The types which are common in one country are not necessarily so in another, and in tropical zones a very large variety occur which are not considered here, but are dealt with fully in works on tropical medicine. The *microsporon*,

or small spored ringworm, attacks almost entirely children under the age of about 16, while the epidermophyton is not frequently seen in young children. The trichophyton, however, attacks children and adults indiscriminately. The genera can usually be distinguished without difficulty from one another both clinically and by examining the hair or scales under the microscope in liq. potassæ. The different species can, however, only be distinguished by their cultural characteristics. The same ringworm fungus grows differently on different media, and in order to compare cultures the fungus is by tacit agreement grown on what is known as Sabouraud's "proof medium," for the reason that this observer has collected and illustrated in his book, *Les Teignes*, a very large number of the known ringworm fungi. The common nomenclature of the fungi is that adopted in this work.

Ringworm is found on the scalp, where it almost universally travels along the hairs into the hair follicles, on the beard region, where the hairs are often but not always affected, or on the glabrous skin, where it usually remains confined to the surface horny layer. The nails are also sometimes attacked.

(a) RINGWORM OF THE SCALP

Ætiology and Pathology.—Ringworm of the scalp, *tinea tonsurans*, is essentially a disease of childhood, the adult scalp being so rarely attacked as to be considered a curiosity. In this country about 90 per cent. of cases of scalp ringworm are produced by the microsporon fungus, the large majority of these being produced by a human species, *Microsporon audouinii*, the rest (not more than about 5 per cent.) by the microspora of the cat, dog and sometimes other animals. Another 10 per cent. or so of cases are due to trichophyton fungi of which there are several species. The microsporon fungus first attacks the horny layer on the surface of the scalp; it reaches the hair shaft at the mouth of the follicle and grows down on and beneath the cuticle of the hair, destroying the cuticle and fibrillating the hair, and finally terminates in a fringe of mycelial processes just above the expansion of the bulb of the hair. The mycelial processes on the surface of the hair give off small round spores, which are packed so closely together that, when examined in liq. potassæ under a $\frac{1}{2}$ -inch objective, they are seen to form a thick mosaic sheath round the hair. As a result of the damage produced, the hairs first lose their elasticity and then fracture. This fracture usually takes place about $\frac{1}{2}$ -inch above the mouth of the follicle.

Symptoms.—*Microsporon ringworm.*—In the bulk of cases the disease begins with a small circular scaly patch on the scalp. Very soon the hair on the patch is noticed to be thinning. Several patches may appear simultaneously. On close examination with a lens, these circular patches are found to be covered with fine, branny scales of a greyish colour, the follicles are prominent, giving the patches a nutmeg-grater-like appearance, and numerous broken hairs are seen. At the edge some hairs may be found unbroken but bent at sharp angles, as though a sort of greenstick fracture had occurred. These hairs and the stumps are often covered with a whitish powder, which is the spore sheath referred to above.

On pulling one of the stumps with forceps the former will come away, but usually breaks off above the hair bulb, leaving the latter behind; a great deal of perseverance is necessary to remove the stump intact. The hair

thus removed and examined in liq. potass. has the appearances mentioned above, and in addition the fibrillation of the hair will be noted, especially the irregular fracture of the distal end. In old-standing cases the regular circular outline of the patches may be lost, the whole scalp having a moth-eaten appearance, and stumps being scattered irregularly over large areas. The microsporon ringworms contracted from animals have similar appearances.

Endothrix ringworm.—The fungus of this type is not contracted from animals. The clinical appearances may be similar to microsporon ringworm, but two other types are seen. In some cases no patches are present, but a general thick scurfiness of the scalp occurs. On very careful search with a lens isolated stumps may be found scattered all over the scalp. In other cases sharply defined bald patches occur, which on inspection show no stumps, but every follicular opening is filled with a small black spot. By the careful use of pointed epilation forceps, such as those devised by Whitfield, one or more of these spots may be removed, and on examination the fungus can be demonstrated. This type is called *black-dot ringworm*, and has to be distinguished from alopecia areata.

When the stumps from an endothrix ringworm are examined in liq. potass. under the microscope the spore sheath is found to be absent, the fungus being entirely inside the hair and the cuticle intact. The fungus itself consists of longitudinally running mycelial filaments, which are divided up into small square, round or oval segments, the whole having a ladder- or chain-like appearance.

Ectothrix ringworm.—The fungus which produces this type is of animal origin, and generally produces a much more inflammatory type of lesion than the other varieties. In the majority of cases suppuration occurs, the fungus itself being responsible for pus formation. These suppurating ringworms are spoken of as *kerion celsi*. The affected area is much swollen and red, and often raised considerably from the surrounding skin. The swelling is boggy to the touch, and often gives the sensation of fluctuation, which to the uninitiated suggests abscess formation. On the surface pus is seen to exude from numerous follicular openings, but broken hairs are also seen. These if examined in liq. potass. show fungus, both within and without the hair; the cuticle is destroyed and the mycelium has similar characters to the endothrix fungus, the spores being arranged in chains and not packed together, as in the microsporon type.

Diagnosis.—This is usually simple, the presence of the stumps containing fungus being diagnostic. In cases where stumps are few in number, great help can be obtained by examining the child's scalp under a mercury-vapour lamp, screened by what is known as "Wood's glass." In microsporon ringworm the affected stumps fluoresce brilliantly and can be readily seen. This method is particularly valuable in determining whether a case is cured after treatment. Fluorescence does not occur in endothrix ringworm. From favus the diagnosis is made by the presence of yellow favus cups. From seborrhœic dermatitis the diagnosis should not be difficult if it is always remembered that a scurfy head in a child must always be considered to be ringworm until this has been excluded. Great care has, however, to be taken to make a thorough search for stumps in the endothrix cases. In alopecia areata a smooth, shiny centre with, perhaps, a row of scattered stumps at the periphery of the patch is found. These stumps, however, are club-shaped, are very thin

as they enter the scalp, and when pulled out always come away with a shrunken bulb attached. No fungus can be seen on microscopic examination.

Treatment.—The cardinal fact to remember in the treatment of scalp ringworm is that up to the present no means has been discovered of killing the fungus in the hair follicles. It is, therefore, necessary to epilate the hairs in order to obtain a cure. This can be done by three methods: by X-rays; by producing sufficient inflammation in the affected areas to make the hairs fall out; or by the administration of thallium acetate internally. This second method is the way Nature cures some cases. In kerion the suppuration is sufficient to loosen the hairs, and all that is necessary is to assist this process by hot fomentations and epilation with forceps. In the ordinary microsporon type, however, the production of the necessary inflammatory reaction is not so easy. Various irritants have been used—the most satisfactory of which is croton oil. The application of this, however, requires great care, and is not suitable for ordinary out-patient practice. Probably the best application available at present is an ointment of equal parts of common salt and soft paraffin. The scalp is shaved and washed daily with soap and water, the healthy portion then smeared with a weak mercurial ointment, such as 2·5 per cent. white precipitate ointment, and the salt ointment rubbed vigorously into the ringworm patches. After a time the patches inflame, and the hairs loosen and fall out. Cure by this method in fairly localised cases takes 2 or 3 months if the treatment is vigorously and conscientiously carried out; otherwise it may take 12 or 18 months to effect a cure. X-ray treatment is generally preferable. The method used is that devised by Adamson and Kienbock, and consists in treating the scalp from five different points with an epilation dose, the points being so arranged that the whole scalp is uniformly irradiated. A modified four-area method introduced by Schreuss is gradually becoming more popular. This should cause all the hair to fall out in 3 weeks, and a complete cure should take place.

It has recently been shown, chiefly owing to the work of Buschke and his associates, that, if thallium acetate in a single dose of 8 mgrms. per kilo body weight be administered orally, the scalp hair will fall out after about 18 days, leaving the eyebrows and eyelashes unaffected. This method has now been used in a large number of cases of ringworm, and gives satisfactory results. It does, however, often produce well-marked toxic symptoms, chiefly severe joint pains and gastro-intestinal disturbance, and some fatal cases have been reported as a result of accidental overdosage. Its final beneficial results are probably not equal to those of X-rays. It should only be given to children who are perfectly healthy.

(b) RINGWORM OF THE BEARD

Symptoms.—Ringworm of the beard, *tinea barbæ*, occurs in two types: (1) the superficial, scaly type, and (2) the suppurative type. The former begins as a small scurfy patch, which spreads slowly in ring fashion and resembles the scaly type on the scalp. The hairs are usually attacked, and if removed fungus can be demonstrated in them and also in the scales. The fungus is usually of the endothrix type, and as such is transmitted from man to man. It is not infrequently caught in the barber's shop, and is one of the three forms of "barber's rash."

The suppurative type produces an irregular lumpy swelling of the affected part. The "lumps" are soft and boggy to the feel, and pus may be seen exuding from various follicular openings; the case bears a close resemblance to *kerion celsi*, but has not the same sharp circular edge, being more irregularly distributed. The hairs are attacked by the fungus, which in this case is generally of the *ectothrix* type, and is usually transmitted from animals, being frequent among grooms and cattlemen.

Diagnosis.—The scaly variety must be distinguished from seborrhœic dermatitis and the pityriasisform type of streptococcal infection. This is easily done by the presence of fungus in the hairs and scales of ringworm.

The suppurative type may be confused with the staphylococcal sycosis; but the latter never forms the tumour-like masses which are seen in ringworm, while again the presence of fungus will settle the diagnosis.

Treatment.—The same principles apply as in scalp ringworm. For the scaly type X-rays form the most certain form of treatment. Alternatively 3 per cent. salicylic and 5 per cent. benzoic acid ointment should be rubbed in daily and the hairs epilated, a few at a time, with forceps. The hair should be kept out short.

With the suppurative variety hot fomentations and epilation with forceps should be used.

(c) RINGWORM OF THE GLABROUS SKIN

This can be divided into four types. (1) *Tinea circinata*, the small ring- and disk-like patches seen about the face, neck, body and limbs; (2) *tinea cruris*, *eczema marginatum* or *dhobie itch*, seen chiefly as sheet-like patches in the inner side of the thighs, and on the perineum and scrotum; (3) the *eczematoid ringworms* of the hands and feet; and (4) the *pustular body ringworms*.

Symptoms.—1. *Tinea circinata*.—This condition may occur by itself or in combination with scalp ringworm. In the latter condition it usually occurs on the neck or face. In the microsporon cases of human origin the lesions take the form of small disks, usually not larger than a threepenny bit, which show little tendency to grow, and no tendency to clear in the centre or to form rings. The patches are of a pale pink colour, and are covered with branny scales, in which mycelial filaments can be demonstrated by examining them under a $\frac{1}{8}$ -inch objective in liq. potassæ. The other varieties of microsporon and the *endothrix trichophyton*s show a much greater tendency to form rings and to attain a larger size. In these cases the earliest spots are similar to those described above, but as they spread the centre loses its scaliness, becomes a paler colour, and eventually the skin resumes its normal character. The spreading edge presents the same branny scaling, and often small pinhead-sized vesicles and pustules. As before, mycelial filaments can be demonstrated in the scales. Itching is often present to a greater or less degree. In rare cases these rings are very numerous, and concentric rings may form. This is well seen in some tropical varieties, such as *tinea imbricata*, where the whole body is covered with concentric ring-formations.

2. *Tinea cruris*.—This condition, also known as *dhobie itch*, is produced by the genus *Epidermophyton*. It is so named because of the commonly held view that clothes are infected by the washerman or *dhobie*. Originally a tropical type, it is now extremely common in this country, being much more

often seen in private than in hospital practice, and almost entirely in males. It is usually seen as a superficial, flat, brownish-red patch situated bilaterally on the inner surface of the thighs in their upper third. The patches usually meet on the perineum, and often involve the whole scrotum, and sometimes spread forward into the groins. The patches, which were formerly known as *eczema marginatum*, have a very sharply defined margin, which is very slightly scaly, but no vesicles are present. Scrapings from the scales show a chain-like mycelium. The patches, though usually confined to this region, are occasionally seen on the umbilicus and in the axillæ, and are frequently associated with one type of eczematoid ringworm seen between the toes. There is generally intense itching felt in the patches.

3. *Eczematoid ringworms*.—There are several varieties of this type seen. The commonest is that which occurs between the toes. It occurs first between the little and fourth toe, and is generally bilateral. The skin in the web of the toe becomes thickened, whitish and sodden, and fissuring is prone to occur. It may spread to adjoining interdigital spaces, and on the dorsum and sole of the foot. In severe cases this area becomes covered with vesicles or large blebs, which may become purulent. It is not always easy to demonstrate the fungus in the thickened skin between the toes; considerable time must be given to soaking in liq. potassæ the skin removed, and many slides may have to be made before the search is rewarded.

The most common type seen on the hands consists of rather sharply circumscribed patches of a vesicular dermatitis. They may occur on any part of the hand or fingers, and are generally single and unilateral. They spread slowly, and are itchy. The lesions are usually produced by the trichophyton fungus. The demonstration of the fungus is necessary to distinguish them from other forms of localised dermatitis. When the lesions occur on the palm much thickening of the horny layer is produced, and cracking in the deeper folds may take place.

In another form an acute dermatitis which may involve both hands and feet, is set up, as has been shown by Whitfield. The cases are clinically indistinguishable from the type of acute dermatitis known as dysidrosis or cheiropompholyx, and in all such cases a careful examination must be made for a ringworm fungus.

4. *Pustular body ringworms*.—These occur in sharply defined patches, chiefly on the limbs and neck. The patches are of dull red colour, and sharply raised from the surrounding skin; they have a soft boggy feel, and pus can be seen exuding from the follicles. The fungus in this case is usually of the ectothrix variety.

Diagnosis.—This is only difficult in the acute eczematoid varieties, when it must be distinguished from cheiropompholyx and the localised forms of dermatitis and eczema. This can only be done with certainty by demonstrating the fungus. The circinate patches have to be distinguished from seborrhœic dermatitis and the scaly streptococcal lesions, and on the face from pityriasis simplex. The presence of fungus, and the ease with which patches respond to Whitfield's ointment, as well as the tendency to ring-formation, and asymmetrical distribution, will enable a diagnosis of ringworm to be made.

Treatment.—This is simple in the flat body patches and in tinea cruris. The number of antiparasitic remedies is large but the most satisfactory is

fuchsin paint (Castellani). This is painted on the patches once or twice daily, and can be used in the most inflamed cases. Other preparations include ung. ac. benzoici co (B.P.C.) (Whitfield's Ointment) and liq. iodi mitis (B.P.). Most patches will clear up in a week or two with these remedies, but it is well to continue them for some days after the lesions have disappeared in order to prevent recurrence.

Eczematoid ringworm of the toes can be treated with Whitfield's ointment, the sodden epidermis being removed daily after washing with soap and water; it is well to use this intermittently for 2 weeks at a time, powdering the toes well with a bland powder during the alternate 2 weeks, in order that the keratolytic action of the salicylic acid may subside and give a better indication of the results of treatment. In resistant cases 10 per cent. chrysarobin in spirit may be used in addition. These cases are always resistant to treatment, which requires to be carried on for long periods.

The acute eczematoid ringworms of the hands are often made worse by strong parasitocides, and it is generally better to start treatment with a wet dressing of 1 in 4000 potassium permanganate, subsequently trying small areas tentatively with the preparations mentioned above. The suppurative type can also be treated with 1 in 1000 acriflavine, or 1 in 4000 perchloride of mercury dressing, and subsequently with Whitfield's ointment, if not cured by the former methods. Resistant cases respond well to X-ray treatment.

(d) RINGWORM OF THE NAILS

This is fortunately not a very common affection, but occurs with sufficient frequency to be on the look out for it. It may be caused by the *endothrix* or *ectothrix* fungus.

Symptoms.—Usually several but not all the nails are affected. The disease usually commences under the free end of the nail, and travels slowly upwards. The nail bed becomes much thickened, and the epithelium sodden, and can be scraped away. As the disease spreads the nail becomes a greenish-grey colour and separated from its bed; the growing edge can be seen as a yellowish line above the discoloured and separated nail. In other cases the nail becomes soft or brittle and breaks up, exposing the underlying sodden nail bed. Very rarely the sides and base of the nail may be primarily affected. The toe nails are frequently affected in interdigital ringworm of the feet.

Diagnosis.—The diagnosis has to be made from eczema, psoriasis and syphilis. This can only be done with certainty by finding the fungus. Portions of nail near the growing edge should be taken and soaked for some hours in liq. potassæ. The under surface is then scraped and mounted, and a search made; and this may require several preparations before the mycelium is found. Cultures can often be made direct from pieces of nail; but contamination is very frequent.

Treatment.—The nail must be removed, either surgically or by softening in strong potash and scraping it away. Afterwards one of the stronger anti-trichophytic remedies can be applied. Norman Walker recommends covering the affected nails with lint soaked in Fehling's solution and applying a rubber finger-stall for 24 hours or longer, so as to remove the nail completely. The solution must not be applied to the surrounding skin.

2. FAVUS

Favus is a disease due to the growth of a fungus allied to ringworm, belonging to the genus *Achorion*. It differs from the former in forming thick, yellow, circular cups which cause local scarring and atrophy of the hair follicles. It is a much rarer disease in this country than formerly; but cases are still occasionally seen.

Symptoms.—Favus attacks the *scalp*, the glabrous skin and the nails, and has been recorded on the mucous membranes. On the scalp it appears as a collection of pea-sized or slightly larger circular yellowish crusts standing up from the skin and having a central depression, through the centre of which the hair projects. This is the *favus cup* or *scutulum*. Very large areas of the scalp may be involved in the process, the whole having a honey-combed appearance. Where the disease has been cured, scars and permanent alopecia are left. In section the yellow cup is seen to be made up of masses of mycelium radiating from the centre. Favus on the *glabrous skin* shows a somewhat similar appearance, a collection of bright yellow cups forming a massive crust, the whole being surrounded by an inflammatory zone. When seen in this country the lesions are generally very few in number and on the exposed parts, but in some countries where the disease is common the whole body may be covered with great masses of favus scutula. Favus of the glabrous skin in this country is often of mouse origin, and a different species to the scalp favus. Favus of the *nails* has somewhat similar characteristics to that of ringworm of the nails.

Treatment.—The only satisfactory treatment for favus of the *scalp* is X-rays. The risks of alopecia mentioned in the treatment of ringworm need not be considered here, as alopecia will result in any case from the disease. As a preliminary to X-Ray treatment the crusts should be removed and the scalp cleaned up with appropriate antiseptic applications.

In *body* favus the scutula must be removed, and the patches treated with either Whitfield's salicylic and benzoic ointment or a 4 per cent. chrysarobin ointment.

Favus of the *nails* is treated in the same way as ringworm of those parts.

3. MONILIA INFECTION

A good deal of attention has recently been paid to lesions closely resembling those produced by the ringworm fungi, but attributable to the growth of yeast-like organisms resembling those found in thrush. The lesions are chiefly found in moist situations, such as the groins, under the breasts and between the toes. The same fungus has been found to be responsible for a sodden condition between the fingers, to which the name *erosio blastomycetica interdigitalis* had formerly been applied. It has also been found in the nail folds, producing a curious bolster-like swelling of these structures, and has also attacked the nails themselves.

The *treatment* of these conditions is similar to that employed in ringworm.

4. TRICHOPHYTIDES

Of recent years a variety of generalised eruptions have been described in association with cases of fungus affection. These have been shown to

be produced in a way analogous to that in which the tuberculides are produced in cases of tuberculosis (see p. 1463). In certain fungus affections the skin becomes sensitive to the toxin of the fungus, as can be demonstrated by intradermal injection of extracts of the fungus concerned. It is presumed that either the fungus itself or its toxins enter the circulation and that eruptions at distant sites are thus produced.

The eruptions vary considerably in type; the lichenoid variety, consisting of numbers of pinhead-sized papules scattered over the trunk and analogous to the lichenoid tuberculide, is the commonest, but eczematous, scarlatiniform, morbilliform and urticarial eruptions have been described, and also lesions resembling erythema multiforme and erythema nodosum. Many of the vesicular eruptions of the palms and soles, associated with interdigital ringworm of the feet, are believed to be trichophytides.

The eruptions are described as microsporides, trichophytides, epidermophytides, favides and levurides, according to the nature of the primary affection, the last named being associated with monilia infections.

The diagnosis rests on the presence of an existing or recently pre-existing fungus infection, together with a proved cuti-sensitiveness to the toxin of the appropriate fungus.

No special treatment is required beyond that required for the primary affection, together with palliative treatment of the lesions.

5. TINEA VERSICOLOR

This is a superficial infection of the horny layer with the *Microsporon furfur*, and is frequently seen among hospital out-patients.

Symptoms.—It usually forms very thin, greenish-yellow patches or a continuous sheet over the chest and abdomen; but may cover larger areas of the body. It is said to occur chiefly in people who wear thick woollen underclothing and perspire freely. If the patches are scraped scales can be removed, and these examined in liq. potassæ show thin mycelial threads with large round spores among them.

Treatment.—The treatment is the same as for other body ringworms, salicylic and benzoic acid ointment or a sulphurous acid lotion causing rapid cure. The underclothing should, however, be sterilised, or reinfection will occur. Precautions against over-clothing should also be taken.

6. ERYTHRASMA

This is an uncommon disease in this country, and is due to the infection of the horny layer with an extremely small fungus, the *Microsporon minutissimum*.

Symptoms.—The affection occurs as superficial, reddish-yellow patches and plaques, more or less symmetrically arranged, chiefly in the groins and axillæ.

Diagnosis.—The malady is to be distinguished chiefly from tinea cruris, and this can readily be done by noting the size of the mycelial elements under the microscope. In erythrasma they are so small as to require a $\frac{1}{12}$ -inch objective, and under it appear as small bead-like chains, with masses

of spores intermingled, while in tinea cruris chain-like mycelium can easily be seen under a $\frac{1}{8}$ -inch objective.

Treatment.—The treatment is the same as for pityriasis versicolor.

7. LEPOTHRIX

This is a not very uncommon affection of the axillary hairs in which they become surrounded with dark reddish concretions.

According to Castellani, this affection is caused by a bacillary-like fungus, *nocardia tenuis*, acting in symbiosis with a red pigment-forming coccus, *micrococcus castellanii*.

The treatment consists in dabbing the affected hairs twice daily with alcoholic formalin (2 per cent.), and rubbing in at night a 2 to 5 per cent. sulphur ointment. Calamine lotion may be used to allay any irritation caused by the treatment (Castellani).

DERMATITIS DUE TO ANIMAL PARASITES

The affections of the skin due to animal parasites are of a mixed variety, but for general purposes may be classed under the superficial dermatoses. Animal parasites produce their effects on the skin either by puncturing and injecting an irritating substance or by burrowing in the skin; but what have chiefly to be taken into consideration are the secondary effects produced by the irritation these creatures produce. In tropical countries the number of animal parasites which produce skin lesions is very large; it is proposed here, however, to consider only those seen commonly in this country.

1. BITES AND STINGS

The common flea, the bed-bug, gnats and the pediculus family are the common biting insects seen in this country, while of the stinging insect bees, wasps, hornets and ants may be mentioned. Excluding pediculi, which require more detailed description, the lesions produced by all these insects are wheals of varying size, depending on the particular insect, and also on the susceptibility of the person attacked. The lesions are familiar to all, and require no detailed description.

Treatment.—As most of these stings are due to an acid irritant, the application of weak solution of ammonia and other alkalis gives most relief. In the case of the bee the sting should be removed if still in the skin.

2. PEDICULOSIS

Three forms of pediculi attack man: the *Pediculus capitis*, the *P. vestimentorum* or *corporis*, and the *Pediculus* or *Phthirius pubis*.

The first two are merely varieties of the same species—the *Pediculus humanus linnaeus*.

PEDICULOSIS CAPITIS.—**Ætiology.**—This condition is caused by a small insect, 2.5 to 3 mm. long, with an oval body consisting of a narrow thorax and wide abdomen, to the former of which are attached six legs, each being provided with a hook-like extremity, with which it hangs on to the hairs. The head is small, oval, and provided with two antennæ, a powerful mandible

and a proboscis with which it punctures the skin in order to suck the host's blood. This variety is found among the scalp hairs, chiefly in female children of the lower classes. Pediculi breed with great rapidity, laying their eggs on the hairs. The eggs are contained in a chitinous, ovoid cell, with a movable lid or operculum, and are known as nits; they are laid from the scalp outwards, and each is stuck on to the hair by a drop of cement extruded by the female as she moves along the hair. Nits can only be removed by unthreading them from the hairs.

Symptoms.—Itching is the only symptom produced by the *P. capitis*, and this is due to an irritating substance injected by the insect when it bites. A large number of infested individuals feel no itching; they are, however, a source of danger, as they infect others. If the itching is severe, scratching follows, and this frequently causes impetigo contagiosa, which is most marked at the back of the scalp, but may spread to the vertex, eventually involving the whole scalp and matting the hair down among thick crusts. Similarly it may spread to the back of the neck and shoulders, and involve large areas of the body. Even when impetigo is absent, the presence of scratch marks on the back of the neck and shoulders is almost diagnostic of *P. capitis*.

Diagnosis.—All cases of impetigo of the scalp, especially in children, should be examined for pediculi. The diagnosis is easily made by finding the pinhead-sized, white, shiny oval bodies attached to the base of the hairs, and in bad cases the transparent little insects themselves can be seen scuttling about among the hairs.

Treatment.—The insects are easy to kill, but the nits are more resistant. The favourite method is to saturate the scalp with paraffin and tie it up for 12 hours; this has the disadvantage of being messy, and is not free from danger if the head is brought too near a naked light. Whitfield's method of saturating the hair with 1 in 40 phenol and then tying the hair up in it for half an hour is very efficacious, especially when much impetigo is present. After this, the crusts can be removed and the nits combed out, and a weak ammoniated mercury ointment applied.

PEDICULOSIS VESTIMENTORUM.—**Ætiology.**—The causative parasite has exactly the same anatomical character as the preceding, but is usually slightly larger, up to 3 to 4 mm. in length. It is not very common in civil life, being only seen in the habitués of the casual ward and the common lodging-house. In war-time, however, it becomes one of the chief causes of sick wastage of armies, being almost universal in its incidence and causing an enormous amount of skin disease.

The insect lives chiefly in the clothes, coming on to the body in order to feed; it is chiefly found, therefore, in those parts of the clothing which come into most intimate contact with the body. In civil life the *Pediculus vestimentorum* is rarely seen, but its nits may be found in the seams of the under-clothing of infected persons. Occasionally in heavily infested people nits may be found on the axillary, pubic and perineal hairs.

Symptoms.—The skin lesions in this condition are mainly those produced by scratching. Closely placed, small, red macules may occasionally be seen, the results of the insect bites, but this is unusual. The scratch eruption has a characteristic distribution and type. In civilians, it is most marked about the back of the shoulders and around the waist and upper part

of the buttocks. In soldiers, it is even better marked on the legs and about the knees, owing to wearing the puttee. The lesions in earlier cases are papules, surmounted by hæmorrhagic crusts and linear excoriations. In cases of longer standing, areas of eczematization and lichenification occur, and the skin becomes irregularly pigmented. Septic complications are not very common in civil life, but in the field are the rule. Boils and linear, gutter-shaped ulcers, described under ecthyma on p. 1432, are extremely common under these latter conditions, chiefly on the legs.

Diagnosis.—This has chiefly to be made from scabies, but the presence of the burrows and the distribution of the rash—described in detail in the article on that disease (p. 1444)—should enable a diagnosis to be made. From senile pruritus the diagnosis can only be made by finding lice or their nits.

Treatment.—Disinfection of the clothing and bedding of the infected person is all that is required, except in those who harbour nits on their hairs, in which case the latter should be cut short or shaved. Most local sanitary authorities will carry out the necessary disinfection if duly notified; the methods employed scarcely come within the scope of this work. Local lesions can afterwards be treated with sedative lotions and creams, and impetiginous lesions as already described (p. 1421).

PEDICULOSIS PUBIS.—**Ætiology.**—The *Pediculus* or *Phthirus pubis* has a different appearance from that of the above-mentioned varieties; the body is shorter, wider and almost triangular in shape. It is usually about 1.5 mm. long and about the same width, and is provided with six legs, which are more curved than in *Pediculus humanus* and are also provided with hook-like extremities. This louse can move with considerable rapidity along the hairs, but has very limited powers of movement on a flat surface. When found among the hairs it is seen clinging with its legs to two adjacent hairs. Its eggs are laid in the same manner as with other varieties.

The pubic louse is found almost exclusively in the pubic and perineal hair, but in severe cases the hair in front of the abdomen, chest and thighs may be infested, as may also the axillary hairs, the beard, the eyebrows and eyelashes. It is extremely rare on the scalp. It is usually transmitted during coitus.

Symptoms.—There are two main symptoms, itching and the presence of small bluish stains on the skin. The itching is often intense and may lead to loss of sleep, but is localised to the area attacked. Scratch lesions are not very common, doubtless owing to the protection afforded by the stout pubic hairs; they do, however, occur. The bluish stains found on the skin in regions infested by the crab-louse are now known to be produced by the bites of the insect. They are 4 to 10 mm. in diameter, not raised above the skin, and do not disappear on pressure. They are known as *maculæ cæruleæ*.

Diagnosis.—This is made by finding the louse and its nits attached to the base of the hairs.

Treatment.—The best results are obtained by clipping the hair short and rubbing in 5 per cent. betanaphthol ointment. Ung. hydrarg., phenol lotion (1 in 40), and petrol are also used, but the former of these may set up a severe dermatitis if not carefully used. On the eyelashes, the insects and their nits should be removed by forceps.

3. SCABIES

Ætiology.—Scabies is a disease caused by a spider-like, acarine parasite, the *Sarcoptes scabiei*. The acari form a large group of animal parasites which attack man and the lower animals. The parasite generally found in man (var. *hominis*) is a special variety and is not contracted from animals. Various other acari, however, which attack animals may also attack man, but they do not produce identical symptoms.

The *Sarcoptes scabiei*, commonly spoken of as the acarus, is a minute round body, just visible to the naked eye, and of white shining appearance. The body bears eight legs, which differ in the two sexes. In both sexes the two anterior pairs bear suckers; in the male the third pair bear long bristles and the fourth pair bear suckers, while in the female both hind pairs bear bristles. The female is larger than the male, and burrows in the horny layer of the skin to lay her eggs. If undisturbed the female may live for 2 to 3 weeks and lay up to about 30 eggs. The eggs are laid in the burrow and the young hatch out there, the complete cycle from egg to mature acarus being completed in about 10 days. The larvæ, however, hatch out in 3 to 3½ days.

The female acarus has certain favourite sites for burrowing, namely, the genitals, the fronts of the wrists, the web and sides of the fingers, the ulnar border of the hand, the backs of the elbows, the anterior axillary folds, the nipples in women, the umbilicus, the sides of the gluteal cleft and lower part of buttocks, the front of the knees, the ankles and the dorsum of the feet. In infants the palms and soles are also frequently affected.

Symptoms.—The eruption of scabies is of two kinds—the acarine burrows and the follicular papular eruption. The burrows occupy the sites named above. They are seen most clearly on the hands, where they usually form thin, sinuous lines, from a millimetre up to a centimetre in length and occasionally even longer. The burrow is generally easily seen, as dirt accumulates in it, but quite often it can only be recognised by a lens. The oldest part of the burrow has a splay mouth, while at the other end the small white body of the acarus, with a black spot in its fore part, can be easily seen with a lens and often with the naked eye. Frequently a clear vesicle or vesicles are seen beneath the burrow, but as a rule on the hands no redness is present unless secondary infection has occurred. When blisters are present, secondary infection is frequent, and pustular, weeping and crusted areas are produced. In other sites vesicles are not common, but a large pea-sized papule usually underlies the burrow, and the burrow itself and its acarus are not so easily seen; these lesions are frequently seen on the penis, scrotum and anterior axillary folds, and are usually diagnostic.

The follicular papular eruption is arranged in smaller or larger circles round the areas where the burrows occur. The main distribution is on the anterior aspect of the body, from the nipples to the knees, and in a semi-circle around the anterior axillary fold. The back is free, except in severe cases, down to the top of the gluteal cleft, but scratch lesions occur on the lower part of the buttocks, where ecthyma is often a complication, and on the back and inner parts of the thighs. On the limbs the eruption occupies both front and back of the forearms, up to about the centre of the arm, and

also occurs around the ankles. The lesions are first pinkish or whitish elevations of scattered hair follicles, but soon they become covered with bloodstained crusts from scratching. Linear scratch marks are rare in scabies. It is not clear what produces the scratch eruption in scabies, but the work of J. W. Munro suggests very strongly that the follicular lesions are produced by the acarine larvæ, and the distribution of the lesions adds confirmation to this view.

In old-standing cases almost the whole body may be affected, though the face and scalp are practically never attacked in adults; but in small children even these areas may suffer.

Diagnosis.—In well-marked cases no difficulty arises, as the burrows can be seen, but in treated cases the diagnosis may be very difficult and a diagnosis from pediculosis may have to be made. Also the two conditions may occur together. The distribution and character of the rash will usually settle the point, but a careful search with a lens for burrows and acari should always be made.

Treatment.—This depends more on the carrying out of detail than on the actual parasitocides used. Three things are necessary, namely, the opening of the burrows by scrubbing, the subsequent application of a parasiticide to the body, and the disinfection of clothes and bedding. The body should be soaked in a hot bath, then rubbed with soft soap, and finally scrubbed with a brush, particular attention being paid to areas where burrows occur. After this sulphur ointment is rubbed in all over the body (face and scalp excepted), and the patient again dresses, his clothes having been disinfected in the meanwhile. On the two following days the ointment is again rubbed in, but no bath given (as it tends to increase the liability to sulphur dermatitis), on the fourth day nothing is done, and on the fifth day the patient has a bath—to wash off the ointment—and puts on clean things, all dirty linen being sent to the wash. If any dermatitis from the sulphur arises, lin. calaminæ, to which 2 per cent. liq. pic. carb. is added, may be smeared on, and if there is much sepsis appropriate treatment can then be applied. Another satisfactory method is to paint the body, after the bath and before drying, with a lotion composed of benzyl benzoate, soft soap and industrial spirit, in equal parts. This is allowed to dry, and the painting repeated at once. Twenty-four hours later a cleansing bath is given. Other preparations which have been used with success are ung. potassii polysulphidi (B.P.C.) (Marcoussen's or Danish ointment), 5 per cent. betanaphthol, and 12 per cent. balsam of Peru ointment.

B.—THE DEEP INFLAMMATORY DERMATOSES

Under this heading are included those inflammatory conditions which start in the dermis or hypoderm, and only involve the epidermis secondarily. It is often easy to decide clinically whether an inflammation starts in the dermis or in the hypoderm, and strictly these conditions should be described separately; but as the same exciting cause may often produce either condition, it is simpler to describe them together.

The causative irritant may reach the point attacked in three ways—namely, (1) through a crack or puncture in the epidermis, (2) by the lymphatics, or (3) by the blood stream. In the first group are included those cases in which certain chemical poisons are introduced into the skin by the bites and stings of insects (already dealt with on p. 1441), and cases in which micro-organisms are introduced into abrasions, as in the case of erysipelas from the streptococcus (p. 19), syphilitic chancre from the *Spirochæta pallida* (p. 193), soft sore from Ducrey's bacillus, lupus vulgaris and lupus verrucosus from the tubercle bacillus (pp. 1460, 1462), and actinomycosis, sporotrichosis, etc., from certain fungi. The second group includes certain lesions produced by bacterial irritants, such as are seen in the lymphangitis abscess in tuberculosis and the sporotrichial gummata. The third group includes the drug eruptions and other dermatoses, which are labelled toxic eruptions and which are presumably due to chemical poisons circulating in the blood, and also eruptions due to the circulation of micro-organisms, such as are seen in the syphilides and tuberculides. For convenience of description it is proposed to deal with the majority of deep inflammatory dermatoses under two headings—(1) the toxic eruptions, and (2) eruptions produced by living organisms. It must be understood, however, that in the present state of knowledge the ætiology of many of those included in the former group is still very obscure. There is also a third group of dermatoses whose characters make it difficult to place them in either group, and these have, therefore, been described as (3) *dermatoses of unknown origin*.

TOXIC ERUPTIONS

It is practically impossible to produce experimentally in animals any of the toxic eruptions, owing to the fact that no animal has a skin comparable to that of man. Consequently, all our experimental knowledge of toxic eruptions has to be derived from the observed effects of drugs and food-stuffs on the human skin. It is, therefore, proposed to consider first the eruptions produced by these substances.

1. DRUG ERUPTIONS

These fall into two great classes—those produced by non-protein-containing and those produced by protein-containing drugs. Extracts of organs given by the mouth rarely, if ever, produce eruptions and are, therefore, not specially considered.

(a) NON-PROTEIN-CONTAINING DRUGS

These include all the ordinary galenicals.

Two classes of eruption are produced by non-protein-containing drugs.

(1) Non-specific eruptions, which may be produced indiscriminately by many different drugs, and (2) specific eruptions, which are peculiar to certain drugs.

(1) NON-SPECIFIC ERUPTIONS

Symptoms.—These are generally erythematous, urticarial or purpuric. The erythematous rashes may be scarlatiniform, morbilliform, or, more

rarely, of the erythema multiforme type ; sometimes the lesions are vesicular. Urticarial lesions are usually of the simple urticaria type, but occasionally the giant forms are seen. Purpuric lesions are often erythematous at the start and develop hæmorrhages later. It is difficult to classify drugs into any special groups by the reactions they produce, but it may be noted that the under-mentioned types of eruption may be produced by the drugs named :

Erythematous.—Acetanilide, alcohol, arsenic, aspirin, barbituric acid and its derivatives, balladonna, benzoic acid, cantharides, capsicum, chloral, chloralamide, chlorbutol (chloretone), chloroform, copaiba, cubeba, digitalis, ipecacuanha, mercury, opium, phenazone (antipyrine), pilocarpine, phenacetin, quinine, rhubarb, salicylic acid and the salicylates, stramonium, strychnine, sulphonal and turpentine.

Urticarial.—Antimony, arsenic, barbituric acid and its derivatives, benzoic acid, chloral, copaiba, digitalis, opium, phenacetin, pilocarpine, quinine, salicylic acid and the salicylates, santolin, turpentine and valerian.

Purpuric.—Arsenic, chloral, chloroform, copaiba, ergot, hyoscyamus, iodoform, mercury, phosphorus, quinine, salicylic acid and the salicylates, stramonium and sulphonal.

(2) SPECIFIC ERUPTIONS

Symptoms.—Certain drugs give rise to eruptions which are characteristic of the drugs. Arsenic, bromides, iodides, phenazone (antipyrine), phenolphthalein, mercury, silver and gold are the most important.

Arsenic.—In addition to simple erythematous and urticarial lesions, an acute generalised exfoliative dermatitis may develop. This is especially seen after injections of arsphenamine (salvarsan). Herpes zoster also occurs. Pigmentation, especially about the trunk, though it may be more or less generalised, is seen in chronic arsenical intoxication. It usually presents a fine reticular pattern. Hyperkeratosis occurs chiefly on the palms and soles ; it may be diffuse or occur in localised, corn-like projections. Occasionally these localised hyperkeratoses develop into epitheliomata. Excessive sweating of the palms and soles (hyperidrosis) may occur, and the nails may become striated and brittle.

Bromides.—Two main types of specific eruption are seen. Bromide acne is a follicular hyperkeratosis, often closely resembling acne vulgaris and seen in the same situations, but often more extensive, involving the legs and arms, as well as the face, chest and back. It is seen chiefly in epileptics who have taken bromide for some time. The other form is the so-called "anthracoid" form, which is most commonly seen in infants and children ; in the former the drug is often conveyed in the mother's milk. Nodules and tumours varying in size from a pea up to an inch or two in diameter are found, chiefly on the face and legs. The tumours are of a deep red colour and studded with minute pustules ; in the larger lesions the surface is often crusted, and in some cases ulceration occurs. The lesions may develop and persist for a considerable time after the drug has been discontinued.

Iodides.—The most typical lesions produced by iodides are papules which look like vesicles and bullæ, but when pricked only blood escapes. They are sometimes spoken of as "pseudo-bullæ." They are common on the face and extremities, and often appear after taking quite small doses of iodides,

and within a very short time, even as quickly as 24 hours. They are most common in patients suffering from nephritis. These lesions may increase rapidly in size and produce large tumour-like masses, studded with pustules or with a crusted or ulcerated surface, and when occurring in patients who are seriously ill may hasten a fatal termination from septic absorption. In the early stages the cases have been mistaken for small-pox. An acne similar to that produced by bromides is also seen.

Phenazone (Antipyrine).—In addition to producing the more generalised types of eruption phenazone may produce large erythematous reddish or purplish patches, situated discretely over the body, of sharply circular outline and giving rise to a sensation of burning. When they subside they leave a very marked pigmentation, which disappears very slowly.

Phenol-phthalein.—This drug, now largely used as an aperient and contained in many proprietary remedies, occasionally produces an eruption similar to the last named. The patches are of dull purplish colour and come out on face and limbs, but often also affect the mucous membrane of the mouth. The lesions belong to the group known as "fixed eruptions," as they tend to recur at the same site if the drug is repeated.

Mercury.—This drug occasionally gives rise to a severe erythema in the groins and axillæ, and also on the palms and soles. There may be also purpuric spots and vesicles. Other symptoms of mercurialism, such as nephritis and ulcerative stomatitis, may also be present.

Silver.—Long-continued ingestion of this drug produces a peculiar slaty-grey pigmentation of the skin, generally universal, but most marked on the exposed parts. It scarcely comes under the heading of inflammations, but is included for the sake of convenience.

Gold.—Injections of gold may give rise to a general exfoliative dermatitis, similar to that of arsphenamine.

(b) PROTEIN-CONTAINING DRUGS

These include serums and vaccines. Vaccines rarely cause marked cutaneous eruptions, but when they occur they are of similar nature to those produced by sera. Serum eruptions form a very interesting group, as it is reasonable to suppose that their method of production is closely analogous to that of those toxic eruptions whose ætiology is obscure. For it has been assumed that these are due either to absorption of poisonous proteins produced by the body or to the toxins of pathogenic bacteria which are present in the body.

Serum eruptions are supposed to be an anaphylactic phenomenon and, though this seems to be a reasonable explanation in those cases where the rashes follow a second injection of a foreign protein given at least 14 days after the first, it does not fit in so well for cases in which the eruption follows the first injection of serum. In these cases a specific allergy must be assumed to exist.

Symptoms.—Any of the above-mentioned non-specific eruptions may develop, but there is a greater tendency for the lesions to be of the *erythema multiforme* type. Often the rash is very extensive, the trunk, face, and limbs being covered with disk-like, sharply circumscribed, infiltrated red lesions; these often become bullous, and hæmorrhages may occur in the

centre of the lesions. In other cases ringed lesions occur, which spread peripherally and clear in the centre—*erythema gyratum*. Not infrequently these lesions are associated with fever, and pain and swelling in the joints, and gastro-intestinal disturbances, such as diarrhoea and vomiting and albuminuria. In other cases the lesions are more of an urticarial nature, with transitory, very itchy wheals and swelling of the skin of the face. These eruptions usually come out about a week or 10 days after the injection of the serum and clear up in about the same time.

Similar eruptions sometimes develop in persons vaccinated against small-pox, though in addition a true vaccinal eruption, in which the lesions have the characters of the vaccine vesicles, may develop.

Urticarial lesions develop in certain individuals who are sensitive to certain food-stuffs, after ingestion of these substances. They will be considered more fully under Urticaria (p. 1451).

(c) THE TREATMENT OF DRUG ERUPTIONS

The first thing is to stop the drug causing the eruption. In the erythematous and urticarial types local soothing lotions are indicated, of which the most useful are lotio evaporans (B.P.C.), or phenol min. 120, lot. calaminæ (B.P.C.) ad fl. oz. 12. Alkaline and bran baths are often very useful.

In the serum eruptions, calcium chloride or lactate is recommended and may be given in 10-grain doses three times a day. Quinine, grs. 1 or 2 three or four times a day, is sometimes of value. In the more severe cases the patient must be kept in bed.

2. THE ERYTHEMATA

The term erythema may be used to signify any transient redness of the skin, such redness being frequently produced by external irritants; and these have already been dealt with under the superficial inflammatory dermatoses. When, however, the term is used to describe a composite clinical picture, two main types have to be considered, namely, the non-infiltrative and the infiltrative.

(a) THE NON-INFILTRATIVE ERYTHEMATA

These include two classes, the congestive and the inflammatory. The congestive type has little or no dermatological importance. It is seen in blushing, which is a pure vasomotor phenomenon, and also in such transitory rashes as that seen during ether administration. The inflammatory type includes the rashes seen in scarlet fever, measles and rôtheln, drug eruptions just referred to, and certain other toxic conditions.

Ætiology.—One of the commonest varieties of erythematous rashes is that occurring as the result of septic absorption from a wound, and many of the cases of so-called surgical and puerperal scarlet fever belong to this group. They also occur in ptomaine poisoning and in other infections of the gastro-intestinal tract, but there always remain a number of cases in which no cause can be found.

Symptoms.—Erythematous rashes are usually of either the scarlatiniform, morbilliform or figurate type. The scarlatiniform cases differ from

true scarlet fever in the absence of other typical signs, such as high temperature combined with rapid pulse, the date of appearance of the rash, the presence of sore throat and the characteristic appearance of the tongue. The morbilliform erythemata differ from measles in the absence of coryza, conjunctivitis and respiratory catarrh. The temperature is atypical and Koplik spots are not found. The figurate variety consists of patches or groups of disk-like lesions which tend to spread peripherally and clear in the centre so as to leave circinate and gyrate patterns. It is seen most commonly in drug eruptions, and can hardly be confused with anything else. In all these varieties as the eruption subsides there is a tendency to scaling, usually of the fine branny type, and this may often be extensive; the glove-like scaling of the palms and soles, seen in scarlet fever, may also occur in the simple types of erythema.

Hæmorrhage may sometimes take place into the erythematous patches, especially when these occur on the lower limbs. Most cases of simple purpura really belong to the erythema group. The mucous membranes may be affected similarly to the skin, and other general symptoms, such as diarrhoea, vomiting, fever and albuminuria, may be present.

Treatment.—Attempts must be made to find and remove the cause. All possible septic foci, such as pyorrhœa, tonsillar sepsis and chronic appendix trouble, should be dealt with. The bowels should be made to act freely and a light diet ordered. Cases should generally be put to bed, and always when there is fever or any marked constitutional symptoms. In cases of streptococcal infection, sulphapyridine may be given.

(b) THE INFILTRATIVE ERYTHEMATA. ERYTHEMA MULTIFORME

Ætiology.—Erythema multiforme may occur as a drug eruption, and especially as a serum eruption, as already noted, but it generally appears without any special cause being determinable. It occurs especially in young adults and may often recur, some cases doing so year after year at regular intervals. Individual attacks may clear up rapidly, but in many cases fresh crops appear, and the disease may go on for weeks or months. The joint swellings which sometimes accompany the skin lesions led to the supposition that the condition was of rheumatic origin, but it is probable that these are only a manifestation of a similar affection of the synovia of the joints.

Pathology.—Microscopic examination shows a dilatation of the vessels of the dermis with a leucocytic exudation. There is much local œdema. The epidermis is œdematous, and fluid may accumulate beneath the horny layer or less frequently beneath the epidermis.

Symptoms.—The lesions of erythema multiforme differ from those mentioned in the last section in forming raised infiltrated lesions, which vary in size from a pea to a five-shilling piece or larger, and which have a well-defined distribution. They are usually found on the backs of the hands, wrists, and forearms and on the face, but are not infrequently found on the palms, and may also involve the trunk and lower limbs. In the milder cases they consist of red papules and patches with a sharply-defined border and are usually completely circular in outline. In the more severe forms hæmorrhages occur in the centre, or they become surmounted by bullæ. There is some tendency to slow peripheral extension, with clearing up of the centre,

so that ringed lesions may be formed. In these a play of colours may be noted, the outer red ring surrounding a purple hæmorrhagic ring, which in its turn surrounds a brownish pigmented centre; these lesions are sometimes called *erythema iris*. In rare cases the bullæ so predominate as closely to resemble a pemphigus. The subjective symptoms are often slight, but sometimes itching and burning occur. Lesions may appear on the mucous membranes. Pain and swelling in the joints are not infrequent, and gastro-intestinal disturbance may occur, as may also fever and albuminuria.

Treatment.—Care must first be taken to remove any possible cause. Of the drugs which are of value are calcium lactate, the salicylates and quinine. The former is best given in 240 minim doses on alternate nights. Local treatment is not really necessary, but calamine lotion or linament may be applied to relieve itching or burning.

Erythema nodosum is closely related to *erythema multiforme*, and is dealt with on p. 296.

3. GRANULOMA ANNULARE

This is a very chronic, raised, ringed eruption, of dead white colour, seen chiefly on the dorsum of the hands.

Ætiology and Pathology.—The cause of this condition is unknown, but various intermediate types between it and *erythema multiforme* have been noticed, and this suggests a relationship with the latter condition. Histologically a dense cellular infiltration, associated with degeneration of the collagen bundles, is found in the deeper layers of the dermis, especially in the neighbourhood of the sweat coils.

Symptoms.—The earliest lesions are small white nodules, seen most commonly on the back of the finger joints. Such lesions often occur in groups. They spread slowly, the centre of the group becoming flattened, and surrounded by a raised, white festooned margin, which can be seen to be made up of nodules of the same character as those of the original lesions. The disease is very slowly progressive, and may last for months or years if not treated. The lesions are generally localised to the hands and wrists, but are occasionally seen in other parts of the body, such as the nape of the neck, the buttocks, elbows and knees. In some cases subcutaneous fibrous nodules have been described in the neighbourhood of the elbow joints and elsewhere. Subjective symptoms are generally absent.

Treatment.—Internally, quinine and the salicylates may be given. X-rays will often cause the lesions to disappear, but no other local treatment has much effect.

4. THE PURPURAS

The purpuras form a group which are closely related to the erythemata and are very often erythematous at the start. In them, however, the blood vessel walls are damaged and hæmorrhage occurs into the dermis. They may be of toxic or of bacterial origin. They are considered in detail on p. 810 *et seq.*

5. THE URTICARIAS

The urticarial eruptions are characterised by the presence of wheals, or localised areas of œdema. These are usually transitory in character and

are accompanied by severe itching. Several different forms are recognised—(1) Simple urticaria, (2) factitious urticaria, (3) giant urticaria, (4) papular urticaria, and (5) urticaria pigmentosa.

Ætiology.—As is pointed out above, urticaria can be produced by the ingestion of certain drugs and by injections of foreign sera. It can also be produced by the ingestion of certain food-stuffs in susceptible persons; for example, porridge, strawberries, shell-fish, eggs and milk. It also occurs after the consumption of decomposing food. As far as is known it is not due to the direct attack of any micro-organism, though syphilitic urticaria has been described. It is clear that it may be produced not only by protein poisons, but by non-protein poisons circulating in the blood. The actual mechanism by which the lesions are produced is not altogether clear. At one time it was thought to be a pure vasomotor neurosis, and that the poisons mentioned acted on the vasomotor centres; but in recent years it has been demonstrated that the lesions are true inflammations, and, therefore, it is probable that the action of the poison concerned is a local one. At the same time external stimuli, such as friction, seem, in many cases, to play a part in determining the points where the poison acts. Lewis believes that the lesions are produced by the liberation of a histamine-like substance from the tissue cells. It must also be noted that urticarial lesions may be directly produced by the injection of poisons into the skin. This is well seen in the bites and stings of insects, and the stings of plants, such as the nettle. In a large number of cases of urticaria, however, it is difficult to find any cause, and these are usually considered to be auto-toxic. The auto-toxin may be generated in the intestinal tract, or in infected foci, such as septic tonsils and teeth and inflammatory trouble in the pelvis. In the giant urticaria cases there is usually a considerable functional element present, and these cases are considered to be vasomotor neuroses. Nothing is known of the ætiology of urticaria pigmentosa.

Symptoms.—*Simple urticaria.*—This is the form most frequently met with in adults. It may occur in an acute or in a chronic form. In the former the eruption appears suddenly, is often accompanied by general symptoms, such as fever, diarrhoea and vomiting, and subsides more or less rapidly. In the chronic type the eruption appears in crops; the individual lesions run a more or less rapid course, but fresh crops continue to come out at intervals and the condition may persist for many weeks, months or even years.

The lesions of simple urticaria are in their earliest stages pale pink papules or patches, varying in size from a pea to an inch or two in diameter. In a short time—sometimes a few minutes, at others an hour or so—the central part of the patch becomes a dead white colour, is firm to the touch and raised a millimetre or two from the surrounding skin. The lesions are intensely itchy. They may be few in number or very numerous; sometimes the whole body may be covered with patches of all shapes and sizes, and figurate patches are common. The eruption is most common on the trunk, but any part of the body, including the mucous membranes, may be affected.

Factitious urticaria.—This is a condition of the skin in which the slightest trauma, such as a slight scratch, will bring out a wheal. This condition is often present in simple urticaria, but frequently exists apart from any spontaneous eruption. The condition is sometimes referred to as *dermatographism*, as it is possible to produce letters in urticarial wheals on the patient.

Giant urticaria.—In this condition the lesions are not so much wheals as circumscribed patches of oedema. They are particularly liable to occur about the face—the eyelids, cheeks and lips often swelling up quite suddenly—burning or itching being an accompanying phenomenon. The mucous membranes are not infrequently attacked, and in a few cases sudden cedema of the larynx may produce dangerous asphyxial symptoms. The lesions are very prone to recur, and these recurrences may persist for years. This condition is sometimes spoken of as *angio-neurotic cedema* (see p. 1073).

Papular urticaria.—This type may be a distinct disease or only a variant of simple urticaria, but is that commonly seen in infants and young children. It is not very commonly seen in breast-fed infants, though it does occur, but otherwise is chiefly seen in the first two years of life; it may in some cases, however, persist, with intermission, up to about 7 years of age. The lesions appear, just like those of the adult form, as pink oval patches, usually about $\frac{1}{2}$ inch in diameter, but instead of the bulk of the whole patch being converted into a wheal only a central pinhead-sized wheal is produced. Itching is intense and often paroxysmal. When the lesion is scratched the central papule becomes inflamed and a bloodstained crust is formed on its summit, and it persists after the surrounding pink zone has disappeared. The cases usually present discrete pinhead-sized papules, covered with bloodstained crusts, suggesting a parasitic origin. Not infrequently vesicles surmount the wheals and occasionally quite large bullæ are found.

The eruption comes out in crops, especially at night, and chiefly on the extensor aspect of the lower limbs, the buttocks and the extensor aspect of the forearms, but may occur almost anywhere on the body. The children seem to suffer little in general health, though sleep at night is often lost, its effects being often more obvious in the parents.

Urticaria pigmentosa.—This is a rare condition and it is still a question whether it should be grouped with the other urticarias. It is chiefly a disease of infancy and childhood, but a certain number of adult cases are on record. The condition may appear within the first few days of life, and in a few cases lesions are said to have been present at birth.

The lesions usually appear as wheals, $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter; and as these disappear a yellowish-brown colour is left, and the lesion may remain raised above the surrounding skin or may flatten down to a pigmentary macule. These pigmented lesions generally persist for years. Itching may or may not be present. Sometimes marked factitious urticaria can be elicited. It would appear that occasionally simple urticarial lesions may leave behind pigmentation, but the condition described above is something quite distinct. In true urticaria pigmentosa there is generally a great increase in the mast-cells of the dermis which is absent in ordinary urticaria leaving pigmentation.

Diagnosis.—Simple urticaria may be confused with dermatitis herpetiformis and with the premycotic stage of mycosis fungoides. In the former, small deep-seated vesicles usually occur; but as they are not always present the diagnosis may be difficult. From the latter a diagnosis cannot be made with certainty, though when occurring in old people and persistent, mycosis fungoides must be considered.

The giant forms must be distinguished from erysipelas and acute erythematous eczema. In the former, high temperature is present, the lesion has

a sharply defined, slowly spreading margin, and is often blistered. In the latter, the eruption is extensive and symmetrical, the skin is red, and vesicles are frequently present. Chronic erysipelas of the lip is a persistent, slowly increasing condition and usually arises from a persistent crack in the lip, while urticaria has a sudden onset and disappears again.

Papular urticaria in children is most frequently confused with scabies. The diagnosis can be settled by the presence of burrows and the finding of acari in the latter condition. In the vesicular form cases may be confused with varicella, but the course of the eruption is quite different.

Urticaria pigmentosa is not likely to be confused with any other condition.

Treatment.—If a cause can be found it must be removed: articles of diet known to cause the eruption must be avoided and all possible septic foci dealt with. If the cause is not clear, various types of food must be stopped one by one in order to exclude a possible source of trouble. Recently a cuti-reaction to various foodstuffs has been devised to detect the causative agent, and it is possible to desensitise patients from the particular poison to which they are susceptible. Apart from this, mild purgation and the administration of intestinal antiseptics, such as salol, ichthammol, creosote and calomel, can be recommended. Some patients improve on tonic drugs, such as iron, arsenic and quinine. Calcium chloride or lactate given over prolonged periods is efficacious in some cases. In others complete freedom from work and even rest in bed are necessary. In cases of unknown ætiology, non-specific protein therapy, such as injection of whole-blood, milk or peptone, is of great value (see p. 1418).

In the papular form in children excessive intake of sugar plays a part in a proportion of cases, and by a rigid cutting down of jams, sweets, etc., relief is often obtained.

In the giant form nerve sedatives, such as valerian and the bromides, are of value.

Locally, anti-pruritic lotions are most useful. Solution of coal tar and subacetate of lead, min. 120 of each to fl. oz. 8 of water; or liq. potass. hydroxid. min. 60, glycerin min. 60, to fl. oz. 8 of water, may be used and can be applied frequently; alkaline and bran baths also give considerable relief. In children a teaspoonful of liq. picis carbon. added to a warm bath before going to bed is a valuable remedy, and in some cases sulphur ointment grs. 15 to 1 ounce has proved useful; but lotions are usually better tolerated.

There is no known treatment which affects urticaria pigmentosa.

6. PRURIGO

Prurigo of Hebra is a condition which is rare in this country, but is not uncommon in Eastern Europe. It is apparently closely connected with papular urticaria, but most authors consider it a distinct affection. It begins usually in the first or second year of life, by the appearance of intensely itchy, pinhead to lentil-sized papules on the extensor aspects of the limbs, chiefly on the legs and forearms; these soon become covered with bloodstained crusts, and eventually the whole of the skin of the affected area becomes thickened (lichenified), pigmented and excoriated. The lesions may eventually involve the whole of the limbs, but the flexures usually escape. The trunk, neck and face may become eczematized and lichenified. The glands

in the groins and axillæ become much enlarged. The milder cases (prurigo mitis) may eventually respond to treatment and get well about the time of puberty, but the more severe cases (prurigo ferox) persist throughout life, the patient eventually succumbing to the disease.

Treatment.—There is no specific treatment. Baths and sedative lotions and ointments, together with sedative drugs, to relieve itching and to ensure sleep, should be employed.

7. DERMATITIS HERPETIFORMIS

Dermatitis herpetiformis or Duhring's disease is a condition characterised by the appearance on the body of crops of erythematous or urticarial papules or patches, usually surmounted by herpetic vesicles or bullæ, and giving rise to intense itching.

The evidence for placing this disease among the toxic eruptions is not conclusive, but is sufficiently suggestive to make it justifiable. In this connexion may be mentioned its occurrence as one of the rarer toxæmias of pregnancy (hydra gestationis), and its close clinical resemblance, in some cases, to the urticarias and the erythemata.

Ætiology and Pathology.—The disease may occur at any time of life ; it is rare in infancy and childhood, although cases have been reported ; it is fairly common in young adult life, but most cases occur in middle life. Both sexes are pretty equally attacked. A special form occurs in association with pregnancy, and is apt to recur with each pregnancy. The malady does not appear to be associated with the presence of any particular micro-organism in the body, and cultures from vesicles and bullæ are sterile in their early stages. Blood cultures are also negative.

It has been considered by some to be due to some nervous derangement, and it is true that it is sometimes associated with neuroses ; it is probable, however, that these are a product rather than a cause of the disease. It is probable that the condition is produced by an unknown toxin.

Microscopically lesions show a dense, cellular infiltration of the superficial part of the dermis, chiefly around the vessels. There is always considerable superficial œdema, which in the vesicular and bullous cases collects under the epidermis, lifting it from the underlying dermis ; the fluid of the vesicles contains a large number of eosinophil corpuscles.

Symptoms.—The eruption is essentially polymorphous, that is to say, all the types of lesions mentioned above may be present in the same case at the same time. Most commonly the lesions look like irregularly figurate urticarial wheals which are surmounted by numerous shotty vesicles. In other cases the lesions are more frankly erythematous, while in others larger vesicles or bullæ form, either with or without an underlying erythematous or urticarial patch. In all cases, except when the bullæ are large, there is intense itching in the lesions, with the result that they are scratched, and small bloodstained crusts or excoriations are seen mixed up with the other manifestations of the disease. The limbs and the trunk are most frequently affected, but any part of the cutaneous surface may be involved, though rarely the palms and soles, and the mucosæ are attacked in a considerable percentage of cases. There is a great tendency for the lesions to recur, fresh crops coming out at frequent intervals, and the disease may

persist for years; the writer has under his care a case of over 40 years' duration.

The general health usually suffers very little, in spite of the fact that sleep is often disturbed. Gastro-intestinal symptoms, such as diarrhoea and vomiting, may occasionally occur, and in fatal cases lesions have been found in the gastro-intestinal tract, which possibly account for the above-mentioned symptoms. An increase in the eosinophil corpuscles in the blood occurs in the majority of cases.

Diagnosis.—From erythema multiforme it can be distinguished by the irregular distribution, the shape and the polymorphic character of the lesions in conjunction with the intense itching which occurs; from urticaria by the presence of vesicles and bullæ, which are almost unknown in simple urticaria, though not infrequent in the papular variety, and by the persistence and recurrence of the lesions; and from pemphigus by the itching, the polymorphous character of the lesions, and the comparatively slight effect on the general health.

Treatment.—This calls for much patience on the part both of the patient and the physician. In the first place a careful examination, both clinical and bacteriological, must be made to find any focus of disease. Of the internal remedies most reliance has been placed on arsenic, and in some cases the eruption ceases when a certain dose is reached; but this is by no means always the case. Arsenic may be given by the mouth as Fowler's solution or Asiatic pill, or by injection, the cacodylate of soda, enesol and arsphenamine (salvarsan) being the favourite preparations. The dose should be small to start with, and increased to the limit of tolerance; and the drug should be discontinued if no definite result is obtained. Aperients and saline lavage of the bowel are satisfactory in severe cases; in others quinine, salicin and sodium bicarbonate have proved successful. In any case attempts should be made to check the itching. Alcohol and coffee should be stopped, and also all excess of sugar in the diet. Phenacetin and phenazone are useful, and sedatives may be given occasionally at night. The injection of certain non-specific protein substances, such as the patient's own blood, horse serum, sterilised milk or peptone, has occasionally produced a cure.

Local remedies consist mainly of anti-pruritic applications. The most useful is a lotion containing 2 to 3 per cent. phenol, 1 per cent. liq. potass. hydroxid., or 2 to 5 per cent. liq. picis carbonis. Alkaline and bran baths are valuable, especially in the bullous cases—in which cases also weak (5 per cent.) sulphur ointment sometimes acts well.

8. PEMPHIGUS

An inflammatory condition of the skin, characterised by the eruption of blisters usually occurring in crops, and associated with constitutional symptoms.

Four different varieties are recognised: (1) acute pemphigus, (2) chronic (pemphigus vulgaris), (3) pemphigus foliaceus, and (4) pemphigus vegetans. Acute pemphigus is now known to be a definite bacterial infection, and should not strictly be included in this group; but it is placed here for convenience of description. In the other three varieties the cause is unknown, but it is believed that they are of toxic origin.

(a) ACUTE PEMPHIGUS

Ætiology.—This is a rare condition which occurs almost entirely in butchers, and appears to be due to inoculation of some abrasion with a pathogenic micro-organism, the diplococcus of Pernet and Bulloch. The lesions are not produced by local spread, as in impetigo contagiosa, but are distributed through the blood stream, and symptoms of acute toxæmia occur simultaneously with (or even before) the appearance of the eruption. The diplococcus can be obtained from the bullæ, as well as from the blood.

Symptoms.—The disease commences acutely with fever, malaise, nausea and vomiting. Bullæ then appear suddenly on the apparently normal skin; they are usually very numerous, and as big as a pigeon's or hen's egg. They may burst, leaving a red oozing surface. The lesions usually involve the mucous membranes of the mouth and throat, and even of the intestinal tract, causing pain and difficulty in swallowing, diarrhœa and vomiting, and secondary bronchitic and broncho-pneumonic complications. In a large proportion of cases a general septicæmic condition supervenes and the patient dies, but in a few milder cases the lesions dry up and the patient recovers. Rarely cases may become chronic.

Treatment.—The cases are so infrequently seen that little can be said on this point. The main principles are to keep the patient in bed, puncture the vesicles, and apply mild antiseptic dressing, e.g. 1 in 4000 potassium permanganate, with a thin layer of paraffin gauze between it and the skin, or 1 in 1000 acriflavine emulsion. Quinine is recommended as the best internal remedy. No observations have been made, as far as the writer knows, with regard to specific therapy.

(b) CHRONIC PEMPHIGUS

Symptoms.—The affection, which is also rare, is characterised by the appearance of crops of bullæ in various parts of the skin and mucous membranes, each bullæ appearing on the skin without any pre-existing erythematous or urticarial lesion. The eruption is not associated with itching, but the general health suffers, thus differing from dermatitis herpetiformis. Further, the bullæ are usually sterile in their early stages, though both the *Staphylococcus pyogenes albus* and the *Bacillus pyocyaneus* have been found; but these are almost certainly secondary infections.

The bullæ usually dry up in the course of a week, leaving an erythematous and scaly patch, which subsequently disappears; but fresh crops of blisters constantly come out, and this state of affairs may persist for months and years. The general tendency is for the disease to continue, while the general health steadily deteriorates, and finally death supervenes.

Diagnosis.—This has to be made from dermatitis herpetiformis and from the bullous form of erythema multiforme. The main points in the diagnosis of the former have been considered in a preceding paragraph. In erythema multiforme the lesions are especially distributed on the extremities, and some of them show the definite coin-shaped erythematous patches. The attack usually lasts only a week or two.

Treatment.—This is very unsatisfactory. Arsenic in full doses has

given good results, and quinine and salicin are also recommended. Suramin (antrypol, germanin Bayer 205) appears to give good results in some cases. Gastro-intestinal antiseptics and colon lavage are useful. Intramuscular injection of horse serum, or of the patient's own serum, may be tried. Local treatment is the same as for acute pemphigus.

(c) PEMPHIGUS FOLIACEUS

This is probably only an extensive type of chronic pemphigus.

Symptoms.—Bullæ appear frequently and over large areas of the body, and as a result set up a condition resembling generalised exfoliative dermatitis. When this stage is reached fresh bullæ are not properly formed, owing to the permeability of the improperly formed horny layer, abortive flaccid lesions constantly appearing on the affected areas. Crusting, scaling and a tendency to warty formations, together with much pigmentation, are present. The trunk, neck, face, scalp and limbs may all be attacked, but usually the hands and feet are much less affected.

The general health is usually much affected; but this is not always so. The writer has had under his observation a case of 20 years' duration who, apart from the skin condition, was otherwise well. Usually, however, cases end fatally in a year or two.

Diagnosis.—The only condition likely to be confused with this is generalised exfoliative dermatitis; but in this latter condition bullous formation is absent.

Treatment.—This is the same as for chronic pemphigus.

(d) PEMPHIGUS VEGETANS

Symptoms.—The lesions in this type frequently first appear in the mucous membrane of the mouth, but may appear on other parts of the body. When fully developed they are chiefly localised to the flexures of the axillæ, elbows, groins, knees and around the anus and vulva. The initial lesion is a flaccid blister, which on rupture develops fungating granulations from its base, which discharge much fetid secretion. The patient's health suffers rapidly from septic absorption, and he usually succumbs in the course of a few months.

Diagnosis.—The condition when fully established is characteristic, but in the early stages the lesions might be mistaken for syphilitic mucous tubercles. The diagnosis can be settled by the presence of other syphilitic lesions, by finding the spirochæta pallida, and by the Wassermann reaction.

Treatment.—No treatment is known to influence the course of the disease. Local antiseptics are required for the lesions, weak perchloride of mercury and eusol being the most likely to be satisfactory. Otherwise treatment is on the same lines as for pemphigus chronicus.

9. DERMATITIS EXFOLIATIVA

Synonym.—Pityriasis Rubra.

This is a generalised inflammation of the skin, characterised by redness and profuse scaling. There are many types of this condition, and divers

causes. It is customary to divide dermatitis exfoliativa into primary and secondary varieties. The former occurs without any pre-existing dermatosis, while the latter really represents the generalisation of some other skin inflammation, such as eczema, seborrhœic dermatitis or psoriasis. When such diseases generalise there is a tendency for them to take on the character of a primary exfoliative dermatitis, presently to be described.

The primary variety occurs in its most striking form in salvarsan poisoning, and may also occur in such diseases as leukaemia and mycosis fungoides—diseases closely related to one another. There still remain, however, a number of cases in which no cause can be found, and which we are probably justified in considering as toxic eruptions.

Symptoms.—The cases due to arsphenamine may be taken as typical of the group. In these the eruption usually commences as bright, scarlatiniform patches on the flexor aspect of the forearms and on the abdomen and chest. The rash spreads rapidly, so that in a day or two the whole body is covered. At first it is found to consist of distinct pinhead-sized macules, chiefly around the follicles, but soon it becomes one continuous red sheet. Almost immediately the eruption begins to scale; the scales may be of the fine branny type, or large like fish scales. The amount varies in different cases, but is often very considerable, the bed having to be swept out several times a day. The onset is not always as described; sometimes the initial eruption is an urticaria, or even like an acute erythematous eczema, involving the face and forearms, and in a case recently under the writer's care it was erythematous-vesicular at the commencement. Once the eruption is fully established it usually takes 2 to 3 months to disappear. In an uncomplicated case the rash gradually subsides, scaling ceases, and the skin assumes its normal colour, though some thickening may remain for a considerable time. The flexures of the limb and the neck usually are the last to clear. The hair is frequently completely shed, but grows again later, and the nails may also be lost temporarily, though this is less frequent. An irregular thickening of the nails is, however, more common. At the onset there may be fever, malaise and intestinal disturbance; later, in the course of 3 to 4 weeks, bronchitis and broncho-pneumonia may supervene, and sometimes cause a fatal issue. Nephritis may also occur, and may cause permanent renal changes. If careful nursing is not provided the skin may become infected, and septic absorption may occur. There is always much enlargement of the lymphatic glands, which are soft and spongy, and conjunctivitis is sometimes present.

Most of the idiopathic cases run a similar course; but several different varieties have been described, among which the condition known as pityriasis rubra of Hebra is apt to be associated with visceral complications and with skin atrophy, and runs a very fatal course.

Diagnosis.—The primary cases must be distinguished from those due to leukaemia and mycosis fungoides. In the former the blood picture will probably clear the diagnosis, and in the latter the severe itching, which usually accompanies it, and which is usually absent in the simple exfoliative dermatitis cases.

Prognosis.—This is always uncertain, and should be very guarded. Cases may clear up in 2 or 3 weeks, or may persist for years, with gradually increasing prostration ending in death.

Treatment.—In the arsphenamine cases sulphur appears to be the best remedy. It may be given as sodium thiosulphate by intravenous injection. The main internal remedies must be directed to maintaining the general health, and to countering complications as they arise. Complete rest in bed is indicated in all cases, however mild they may at first appear, and the warmth of the body must be maintained. Warm bran baths may be given if no fever is present, and dusting the skin with talc powder makes the patient comfortable. Local septic complications must be dealt with by mild antiseptic creams or pastes.

ERUPTIONS DUE TO BACTERIA AND FUNGI

Having dealt with the deep inflammatory dermatoses produced by toxins circulating in the blood, it is now necessary to consider those which are caused by living organisms reaching the skin by the same channels. Three of these form a very important group of dermatoses, namely, tuberculosis, syphilis and leprosy. The two latter have been dealt with on pp. 193 and 121 respectively. There are, however, certain others which require notice. It is probable that the eruptions of certain specific fevers may be due to the presence of the infecting organisms in the tissues; this is known to be the case in the rose spots of typhoid fever. The erythematous and purpuric eruptions sometimes seen in malignant endocarditis and other septicæmic and pyæmic conditions are also probably due to the direct action of the streptococcus, while it will be necessary later to describe the cutaneous manifestations produced by the gonococcus when it enters the blood stream.

1. TUBERCULOSIS CUTIS

The tubercle bacillus may attack the skin in several different ways. The commonest variety is a superficial granulomatous formation known as lupus vulgaris; this variety can apparently be produced both by local inoculation and through the blood stream. Lupus verrucosus, a variant of this type, is generally a local inoculation, and is accompanied by warty overgrowths. Miliary tuberculosis of the skin may accompany general miliary tuberculosis, and local tuberculous ulcers may also form, but are chiefly seen on the mucous membranes. In addition, an infection of the skin may occur when a tuberculous abscess, either from a suppurating lymphatic gland or bone, bursts through the skin, and this is spoken of as scrofuloderma. There are also groups of cutaneous and subcutaneous tuberculous lesions, produced by bacilli circulating in the blood, which are called tuberculides, and include several varieties, the lichenoid or lichen scrofulosorum, the acneiform or acne scrofulosorum, the papulo-necrotic, and the gummatous (erythema induratum or Bazin's disease).

(a) LUPUS VULGARIS

Pathology.—The lupus nodule is composed of a group of ordinary miliary tubercles, such as are seen in the lungs and elsewhere. It consists of groups of epithelioid cells, surrounding giant cells, often with peripherally arranged

nuclei, the whole being surrounded by a dense mass of round cells. Tubercle bacilli have been demonstrated in the lesions, and inoculation into guinea-pigs will produce tuberculous lesions.

Symptoms.—Lupus vulgaris usually attacks the face ; but it is not uncommon to find patches on other parts of the body. In this latter site it may be symmetrical. Usually on the face it is asymmetrical. It is most frequent on the nose or cheek, and frequently begins in childhood. The earliest lesion is a small, dull-red pinhead-sized spot, to which other similar spots are soon added, the whole being surrounded by an erythematous zone. On pressure with a lens, however, these original spots can be distinctly seen as yellowish points or nodules compared in appearance to apple jelly. They are very soft, and if a pointed match is applied to one of them it sinks easily into the nodule. These patches may slowly spread so as to involve considerable areas, and may persist for a long period without ulceration. In other cases, however, ulceration may supervene, and considerable destruction of tissue take place, especially on the nose, which is often completely destroyed up to the edges of the nasal bones. The bone itself is not attacked ; but the cartilage may completely disappear. If healing occurs, a soft, superficial scar is produced ; but fresh nodules are liable to appear in it. Any part of the face may be attacked ; but the scalp usually escapes. The glands in the neck may enlarge and occasionally break down, but this is uncommon. Facial lupus vulgaris is frequently complicated by similar lesions in the mucosa of the nose and mouth, and these situations are sometimes the primary seat of the disease. On the mucous membranes the nodules are not visible, but sharply defined, raised, rather warty-looking patches occur. It is commonly seen on the inner aspects of the cheek, gums, palate and nasal mucosa ; but the pharynx and larynx may be involved.

On the body the patches may attain great dimensions ; they often spread at the margins and heal in the centre, forming irregular gyrate patterns. As a rule they are of the non-ulcerating type ; but in some cases ulceration occurs. In some cases, too, considerable contraction of the skin results, leading to deformity ; in others the lymphatic vessels become blocked, and a condition of elephantiasis may supervene.

The disease usually spreads very slowly, and lasts a great number of years. Some cases remain practically stationary almost indefinitely. A few, however, spread rapidly and defy treatment.

In old-standing cases there is a definite tendency to the development of carcinoma. This is of the squamous type, and does not as a rule give rise to secondary carcinomatous glands. It can, therefore, generally be removed locally.

Diagnosis.—Lupus vulgaris is most easily confused with lupus erythematosus (p. 1466). In the former definite nodules are present, and there is a tendency to ulceration ; the disease usually begins in childhood and persists for many years. In the latter the disease is non-ulcerative, has a great tendency to be symmetrical, rarely appears before adult life, and is especially liable to occur in the middle-aged. The scalp is often attacked, while this is rare in lupus vulgaris. In lupus erythematosus thick adherent scales, fixed down by epithelial plugs, form, and the scar is often covered with pits of varying size ; in lupus vulgaris the scale is of a very superficial type and easily removed, and there are no plugs.

From tertiary syphilis the diagnosis may occasionally be difficult, and the two conditions may be combined. The absence of nodules, the tendency to form sharply cut, rather deep ulcers, the presence of a positive Wassermann reaction, and the rapid response to anti-syphilitic remedies will generally settle the diagnosis.

Rodent ulcer is distinguished by its appearance fairly late in life, by its firm rolled edges of pearly white colour, and by its tendency to involve the bony structures of the face.

Treatment.—Of prime importance is the general health of the patient. Good ventilation, sunlight, warm clothing and plenty of good food are necessary if cases are to do well. In the absence of a good supply of natural sunlight excellent results are obtained by exposing the body to the rays of the carbon-arc or mercury-vapour lamps. As to drugs, cod-liver oil, malt, iron and arsenic are often of considerable value.

Local measures should also be taken to destroy the lesions. Small patches may be excised; but this method is only rarely applicable. If the patches are non-ulcerating, and not too extensive, excellent results are obtained by the Finsen light; but this method is rarely available, and is very slow. The nodules may be destroyed by the application of 20 per cent. ac. salicylic plaster with cresote, or 10 per cent. pyrogallie acid ointment rubbed in daily until a violent reaction is produced. Adamson has recommended rubbing acid nitrate of mercury into the patches, and this gives very satisfactory results. In the larger ulcerating patches a preliminary scraping, followed by the application of acid nitrate of mercury or zinc chloride stick, is usually satisfactory. The use of X-rays and CO₂ snow has nothing to recommend it, and the former is very dangerous if given over prolonged periods. On the other hand, some satisfactory results have been reported after treatment with "Grenz" rays.

(b) LUPUS VERRUCOSUS

This is due to the local inoculation of the tubercle bacillus, and is chiefly seen in those who handle infected meat, and in those who conduct autopsies. It is also known as *verruca necrogenica* or *post-mortem wart*.

Symptoms.—The lesions usually occur on the hands, chiefly the dorsum and on the knuckles. The earliest lesion is a small, red, firm papule, which spreads slowly. The centre soon becomes raised and warty; but there is always a well-marked inflammatory zone around this warty growth. Serum and pus may exude between the papillæ of the wart, and the whole may be crusted. The lesion is generally single, and may attain several inches in diameter. Rarely numerous lesions are present.

Treatment.—Small lesions are best excised. Destruction by the actual cautery, or by diathermy, may be practised in some cases. In this variety a pastille dose of X-rays may flatten down the warty growth, and this may be followed by the use of salicylic acid or mercurial plasters, or by painting with acid nitrate of mercury.

(c) LOCAL TUBERCULOUS ULCERS

These occur usually as complications of tuberculosis of other organs. They are frequently present on the mucous membranes, or around the orifices

of the body. Little can be done except palliative treatment if they are numerous ; but isolated ones can be destroyed by one of the methods already described.

(d) SCROFULODERMIA

This term is applied to secondary infection of the skin from the bursting of deep-seated tuberculous abscesses. The lesions either take the form of a thick crust overlying an area of unhealthy-looking granulation tissue, or are purplish-red shiny areas surrounding a sinus.

The term is also applied to single or multiple subcutaneous abscesses, not infrequently seen in children, which contain pus, in which numerous tubercle bacilli can be found. They are sometimes called *tuberculous gummata*.

Treatment.—The treatment of the underlying condition is essentially surgical and where possible the affected skin should be excised, otherwise scraping, followed by painting with acid nitrate of mercury, is the best treatment.

(e) THE TUBERCULIDES

These lesions, which are due to the lodgment of tubercle bacilli in the peripheral capillaries, with the production of a local inflammatory reaction, differ from the foregoing tuberculous diseases of the skin in that there is no tendency for the individual lesions to spread. They are thus comparable to the secondary syphilides, and like them are of several different types.

THE LICHENOID TUBERCULIDE.—**Symptoms.**—This condition, also known as *lichen scrofulosorum*, is chiefly seen in young children with glandular tuberculosis. The lesions come out in crops, chiefly on the trunk, and are arranged in circular or oval groups, made up of pinhead-sized acuminate follicular papules. These lesions may be of the same colour as the normal skin, or of bright red colour. There is usually a small crust on the summit of each papule, or sometimes a small pustule. The disease lasts from a week or two to many months.

Diagnosis.—In lichen spinulosus there is less obvious inflammation, and a horny spine projects from the centre of the papule, which can be removed by forceps.

The small follicular syphilide occurs in adults, and is associated with other syphilitic phenomena.

Treatment.—No special treatment of the skin has any effect. The treatment is that for glandular tuberculosis.

THE ACNEIFORM TUBERCULIDE.—**Symptoms.**—This condition, also known as *acne scrofulosorum*, occurs in children and adolescents who are suffering from some form of tuberculosis, and chiefly affects the buttocks and thighs, but may be more extensive. The lesions are lentil-sized, acuminate, follicular papules and pustules, and are generally distributed discretely. They are of bright red colour and pustular or crusted.

Diagnosis.—The affection is sometimes difficult to diagnose from staphylococcal folliculitis ; but the individual lesions run a much slower course, and are usually more numerous and not painful.

Treatment.—This is the same as for the lichenoid tuberculide.

THE PAPULO-NECROTIC TUBERCULIDE.—Symptoms.—In this variety of tuberculide the lesions are small lentil- to pea-sized nodules starting deep in the dermis or in the hypoderm, eventually softening and bursting through the skin with the production of a small rather indolent ulcer. After healing, pitted scars are left. The lesions are usually numerous and come out in crops, which may continue to appear over a period of some years. The parts affected are chiefly the distal extremities of the limbs, *e.g.* the backs of the hands and feet, the sides of the fingers and the extensor aspects of the forearms and legs. Lesions in these regions have been named *folliclis*. Somewhat similar lesions have been described on the face and termed *acutis*, but it is still not quite clear that they belong to the same group. The eruption nearly always occurs in patients who have some other manifestations of tuberculosis, and occurs chiefly in young adults.

Diagnosis.—These cases can be distinguished from *erythema multiforme* by their deeper site of origin, and by their tendency to ulcerate and produce scars.

Treatment.—As for other tuberculides. No local treatment has any effect on the lesions.

THE GUMMATOUS TUBERCULIDE.—Symptoms.—This condition, which also goes by the name of *erythema induratum* or *Bazin's disease*, is not uncommon, and is almost entirely confined to the legs, especially the calves, and is usually bilateral. It occurs chiefly in girls and young women between the ages of 15 and 25. The initial lesion is a deep-seated nodule, from a pea to a hazel-nut in size, starting in the subcutaneous fat. The nodule slowly increases in size, involves the skin, which becomes purplish in colour, and eventually softens and bursts. The ulcer thus produced has a ragged edge and an unhealthy purplish-red base, often covered by a dirty greenish slough. These ulcers are very sluggish, and take weeks or months to heal. Fresh lesions are constantly forming, and a dozen or more lesions may be found simultaneously on the two limbs. There is often considerable pain in the lesions.

Diagnosis.—From syphilitic gummata.—In this condition the lesions are less numerous—indeed often single—and are rarely so symmetrical. The lesions are usually painless. The edge of the ulcer is sharper and more cleanly cut, and the base is cleaner, or has a characteristic wash-leather slough. Other stigmata of syphilis may be present, and the Wassermann reaction is positive.

In older patients, mainly women, similar nodules occur, which do not break down and ulcerate. These are probably of the same nature but have been called *hypodermic sarcoids*, and must not be confused with *cutaneous sarcoids* described under *sarcoidosis*.

Treatment.—The ulcers heal readily if the patient is kept in bed, but are liable to recur when she gets up again. General tonic treatment should be given, and in many cases arsphenamine has proved to be a potent remedy. Locally antiseptic baths and dressings are required.

2. LUPUS ERYTHEMATOSUS

An inflammatory condition of the dermis, usually chronic but occasionally running an acute course, characterised by the presence of circumscribed

red patches, with or without adherent scales, and which on recovery leaves scars.

Ætiology.—This is still unknown. It has for many years been thought to be due to toxins of the tubercle bacillus and is frequently associated with tuberculosis, but cases occur in which this disease cannot be traced. Lately a good deal of attention has been called to focal sepsis as a cause, but the evidence is no more conclusive than for tuberculosis. It is thought that it may be produced by more than one variety of toxin, or that it may be due to a specific organism as yet undiscovered. There can be little doubt that exposure to sunlight tends to bring out the lesions. The disease is chiefly found in middle age, but may begin before the age of 20. It is more frequent in women.

Pathology.—The chief change in the skin is an infiltration in the neighbourhood of the vessels of the dermis with round cells, which may destroy the hair follicles and sweat ducts. In the epidermis there is a hyperkeratosis, which is especially marked at the follicular openings, so that horny plugs are formed.

Symptoms.—Two main types are seen—(1) The erythematous, and (2) the scaly or fixed type.

1. *The erythematous type.*—This is less frequent and has a greater tendency to be generalised. It may run an acute course or may develop into the scaly type. The lesions are chiefly seen on the cheeks and form circumscribed disk-like lesions, raised and slightly infiltrated and of a pale red to a purplish-red colour. These often show patulous follicular openings on the surface. They may also occur as diffuse flat non-infiltrated sheets of redness. This type is very apt to be associated with lesions in other parts of the body, particularly the backs of the hands and fingers, the arms and forearms, the chest, neck and ears. Occasionally an almost universal eruption appears. The patches may sometimes become bullous and hæmorrhage may occur into the bullæ. These disseminated cases may be associated with acute visceral diseases, such as pneumonia, pleurisy and nephritis, and even in the absence of these high fever may be present. Usually in this type of case, if the patient survives, the eruption clears up without much scarring.

2. *The scaly type.*—This is by far the commonest variety, and is generally very chronic and localised, but may occasionally be acute and generalised. The lesions are chiefly seen on the nose, cheeks, ears and scalp, but are not uncommon on the backs of the hands. They are very apt to be distributed symmetrically in the shape of a bat's wing on the nose and two cheeks. The lesions are usually irregularly shaped red patches, often sunk below the surface of the skin, and covered with greyish scales, which are extremely adherent. When removed horny plugs are seen to penetrate into the epidermis, and when the patches clear up a depressed scar is left, often with numerous pits on its surface. When the scalp is attacked the hair is lost permanently. The mucous membranes may be attacked, the most frequent sites being the vermilion border of the lips and the palate.

Pain sometimes occurs in the patches and sometimes they itch, but generally no local sensations are present. The patient's health is usually below par, and there is often considerable neurosis, but severe constitutional symptoms are usually absent.

The course is exceedingly chronic, the patches often persisting for years in spite of treatment.

Diagnosis.—The condition is differentiated from lupus vulgaris by the absence of nodules, the symmetry of the lesions, the absence of ulceration and the age of the patient; from erysipelas, by the slowness of the spread and the absence of high fever; and from erythema multiforme, by the chronicity of the lesions and the presence of destructive effect on the skin shown by scarring.

Treatment.—The acute erythematous cases should be kept in bed and complications treated. All possible sources of focal infection should be removed. The drug which appears most to influence cases is quinine, which should be given in full doses. Local treatment is not usually required in the acute erythematous cases. In all cases, especially the very acute types, sulphapyridine is worthy of trial.

In the chronic scaly cases, quinine and general tonic treatment are indicated. Good results have been obtained by the intravenous injection of gold compounds, such as myocrysin, krysolgan, triphal, solganal, or sanocrysin. It is advisable to keep the doses of gold preparations small compared with those given for tuberculosis. Intramuscular injections of bismuth salts have also given good results. Rest in bed is always beneficial, and the patient should not be allowed to go out in a strong wind or in the hot sun, as these aggravate the condition.

Local treatment is chiefly directed to removal of the scales and the production of a mild inflammatory reaction in the patches. For the former *ac. salicyl.* ointment, 3 to 5 per cent., or plaster, 5 to 10 per cent., may be employed. For the latter 5 to 10 per cent. pyrogallie acid, painting with pure carbolic acid, or applications for a few seconds of CO₂ snow.

3. GONORRHOEAL KERATOSIS

The lesions in this condition are probably produced by gonococci circulating in the blood stream, although they have not been demonstrated.

They occur in patients suffering from gonorrhœal arthritis and other manifestations of general gonococcal infection, and usually appear on the palms and soles, though other parts of the hands and limbs may be affected.

The lesions are red patches covered with cone-shaped horny thickenings, and are generally numerous. In addition, a general hyperkeratosis of the palms and soles may occur.

Treatment.—General treatment for gonorrhœa is required, together with *ung. ac. salicylic.* locally.

4. SPOROTRICHOSIS

In addition to those bacterial conditions which attack the skin by way of the blood stream, a certain number do so by way of the lymphatics. Lymphangitis with abscess formation from pyogenic organisms is well known, and the same condition in tuberculosis has already been described under the title of "scrofuloderma." Actinomycosis is another such condition, and has already been dealt with (p. 185). Somewhat similar conditions to the

two last mentioned may be produced by certain fungi, of which the only one which requires special notice is sporotrichosis.

Symptoms.—Infection may take place through a crack in the skin, usually on the hand or foot. From this a lymphangitis starts, which spreads up the affected limb, and subcutaneous cold abscesses soon appear at points along the affected lymphatics. These eventually burst and leave indolent fungating ulcers, which show little or no tendency to heal spontaneously. A good deal of pus or yellowish fluid exudes.

Diagnosis.—Cases are usually diagnosed as tuberculosis, and a certain diagnosis can only be made by obtaining the fungus in culture. This should be done on Sabouraud's proof medium and incubated at room temperature.

Treatment.—The lesions usually disappear under large doses of potassium iodide administered internally.

ERUPTIONS DUE TO FILTRABLE VIRUSES

Certain affections of the skin are now known to be due to filtrable viruses. Among these are :

- (1) Herpes zoster (see p. 1583).
- (2) Herpes simplex.
- (3) Herpes preputialis.
- (4) Verruca vulgaris.
- (5) Molluscum contagiosum.

It is more convenient to deal with warts and molluscum contagiosum when discussing tumours of the skin (pp. 1481 and 1482), while zoster is described elsewhere (p. 1583).

1. HERPES SIMPLEX

Herpes Simplex or Herpes Febrilis is a condition to which some people, and especially children, are prone whenever they develop a slight febrile attack or even a slight cold.

Ætiology.—The disease is produced by a filter-passing virus which, when injected into rabbits, produces a fatal form of encephalitis, and is closely related to the virus of encephalitis lethargica.

Symptoms.—The lesions consist of small groups of vesicles, on an inflamed base, which come out chiefly in the neighbourhood of the mouth. They are irregularly distributed, have no relations to any nerve trunks, and are generally bilateral. They disappear in the course of a week or so, after crusting over, and leave no scars. In one type, seen especially in children, recurrent attacks occur on the cheek, often at regular intervals and without any special cause. These attacks also clear up and leave no scars. A recurrent type is also found affecting the buttocks in adults.

Treatment.—A bland protective ointment, such as zinc cream or Lassar's paste, or a dusting powder, such as bismuth subgallate, is all that is required. In recurrent cases, small doses of X-rays given during the quiescent period appear to diminish the liability to fresh attacks. Treatment by vaccines prepared from the virus have been tried, but results are uncertain.

2. HERPES PREPUTIALIS

This is the name given to small crops of two or three to half a dozen or more small vesicles which sometimes appear on the under surface of the prepuce. The vesicles quickly rupture and leave behind pinhead-sized ulcers which are painful. There is no tendency for these ulcers to increase in size. This latter feature helps to differentiate them from both syphilitic ulcers and soft sores.

Treatment.—This is the same as for Herpes Simplex.

ERUPTIONS DUE TO ERRORS OF METABOLISM

XANTHOMA

Xanthoma forms an interesting link between the inflammations due to chemical toxins circulating in the blood and those due to bacteria, for in this condition lesions of a granulomatous nature are produced around a deposit of a chemical substance in the tissues. Apart from this condition, all the granulomata whose nature is known are produced directly by bacteria; tubercle, syphilis and lepra are the best known examples.

Three clinical varieties of xanthoma are recognised: (1) xanthoma tuberosum, (2) xanthoma diabetorum, and (3) xanthoma planum.

Ætiology.—Xanthoma tuberosum and diabetorum occur in patients who for some reason or other have some disturbance of lipoid metabolism, often shown by an excess of cholesterol in the blood serum. This is why one form is seen in diabetics. The cholesterol becomes deposited in the tissues and causes a reaction, chiefly among the fixed connective-tissue cells of the dermis, particularly the endothelial cells, and a granuloma not unlike that seen in tuberculosis is produced. Histologically the tumours of xanthoma consist of large cells, arranged around the vessels, containing droplets of a cholesterol-fatty-acid-ester and some fat. Around these cells a varying degree of connective-tissue hypertrophy may occur.

Symptoms.—*Xanthoma tuberosum.*—In this condition numerous discrete yellowish nodules appear in the skin. These increase in size and may form tumours as big as an orange. They are most commonly seen on the extensor aspects of the limbs, especially on the elbows and knees, where they may form large firm tumours, but they may be seen on any part of the skin. The bones, tendons, viscera and mucous membranes may also be involved. The colour varies from a bright yellow to an orange or red. The disease is usually seen in young adults. It does not generally affect the general health, but is occasionally associated with jaundice. The lesions are very persistent.

Xanthoma diabetorum.—The lesions are usually smaller and more numerous; they are lentil-sized lesions and come out in crops, usually on the buttocks and extensor surfaces of the limbs. They disappear rapidly under appropriate treatment for diabetes.

Treatment.—In the diabetic cases, the underlying disease must be treated. For xanthoma tuberosum no definite treatment can be laid down, but a diet which contains as little fat as possible should be prescribed.

Xanthoma palpebrarum.—**Ætiology**.—This has been considered to be a fatty degeneration of the fibres of the orbicularis palpebrarum muscle, but in some cases an excess of cholesterin in the blood has been demonstrated.

Symptoms.—The lesions consist of flat, yellow, slightly raised patches, which are often symmetrically placed, on the eyelids near the inner canthus. They may be as small as a pin's head or may involve almost the whole eyelid. They produce no symptoms. They mostly occur in old people.

Treatment.—They can be destroyed by electrolysis or by caustics, or removed by excision.

C.—INFLAMMATORY DERMATOSES OF UNKNOWN ORIGIN

In this group are included certain dermatoses with well-defined characters which entitle them to be considered clinical entities, but whose ætiology is entirely obscure. The following diseases are included under this heading: psoriasis, parapsoriasis, pityriasis rubra pilaris, lichen planus, scleroderma and sclerema neonatorum. It must not be assumed that because these conditions are grouped together that they have any relationship to one another.

PSORIASIS

A very common condition characterised by the presence of red, scaly papules and patches of characteristic appearance on various parts of the body and unassociated with any disturbance of the general health.

Ætiology.—The disease frequently begins towards the end of the second decade, and is not infrequently seen in children from about 7 years of age and upwards, but is very rare in small children. Both sexes are equally affected. On the other hand, the first attack may occur in advanced age. It has been attributed to parasitic agencies, toxins of bacterial and metabolic origin, and to neuropathic causes, but there is very little evidence to support any of these views. There is no doubt that in some cases a strong family history can be made out.

Pathology.—Histological examination shows a great overgrowth of the epithelium, with downward growth of the interpapillary processes, and corresponding elongation of the papillæ. The horny layer is badly formed (parakeratosis), and collections of leucocytes can be found between the horny cells. There is a cellular infiltration around the papillary vessels and those of the subpapillary layer.

Symptoms.—The malady is a chronic one and may come and go throughout life. Usually attacks occur at quite irregular intervals, but in some cases they may appear at definite seasons—some appearing in the summer, others in the winter. The extent also varies greatly in different cases, some only having a few patches, others being covered with lesions.

The sites of predilection are the extensor aspects of the limbs—especially of the elbows and knees—the trunk—both back and front, but especi-

ally the waist region, the scalp and, more rarely, the face, nails and palms and soles.

The lesions begin as pinhead-sized papules, and are from their very beginning surmounted by a small silvery scale. The individual lesions usually spread centrifugally and may eventually attain great size. Usually, however, they join with other patches and so form plaques, which may, for instance, cover the whole back in one continuous sheet. The same type of scaling persists even in the largest patches, though in chronic treated cases the surface of these patches may appear to be highly polished; on scratching, however, with a sharp instrument the silvery scales are immediately apparent. The whole mass of scales can, with care, be removed in one continuous sheet, and underneath is found a shiny, dry red surface which, on examination with a lens, shows the dilated papillary vessels as tiny red points.

The arrangement of the lesions varies. In some cases the body and limbs are studded with lesions the size of a small pea or a threepenny-piece (psoriasis guttata); in others the lesions are larger (psoriasis nummularata); in some the centre of the lesions clears up, leaving rings (psoriasis circinata), and the rings may run together, forming gyrate figures (ps. gyrata). Occasionally the crusts are very thick (ps. rupioides), and this is especially the case on the scalp, where the hairs prevent the scales from falling off.

In some cases the lesions remain small and confined to the follicles (follicular psoriasis), and these may occasionally group into patches. They may also come out along scratches on the skin. When the nails are affected, either small pinhead-sized pits may be produced, or the whole nail may be forced up by lesions occurring in the nail bed, the nail eventually breaking up and thick masses of scales being found beneath it. The palms and soles are less frequently involved, but when they are affected circumscribed red patches form, associated with scaling and fissuring in the deep folds. In these regions also a pustular form of psoriasis has been described, consisting of sharply defined red patches studded with minute pustules, usually sterile, imbedded in the patches. The mucous membranes are not affected.

The lesions vary from a pale to a dark red in colour, and on clearing up usually leave little or no pigmentation, though in very chronic patches, especially on the legs, some pigmentation may remain for a time.

There are usually no subjective sensations, but occasionally itching is present. The general health is not affected.

Diagnosis.—This disease may resemble the secondary *papulo-squamous syphilide*. It differs, however, from this condition in the fact that the lesions are scaly from the start; that when the scales are removed no infiltration can be felt, and that the surface left is smooth, dry and studded with numerous small red points; that pigmentation is generally absent or little marked; that the lesions are mainly distributed on the extensor aspects of the limbs, and that the scalp may be extensively involved without loss of hair; and that other signs of syphilis, such as general adenitis and involvement of the mucous membranes, are not present. The Wassermann reaction and the effect of treatment will generally confirm a clinical diagnosis.

In *seborrhæic dermatitis* the scales are greasy, the patches spread by aggregation of follicular papules, and the scalp, face and centre of the chest and back are chiefly affected. The lesions respond quickly to sulphur, which is not the case with psoriasis.

In *eczema* itching is marked, when the scales are removed a moist surface is left, and the lesions are made up of aggregation of papules and papulovesicles.

In *psoriasis rosea* the scaling is usually in the form of a collarette, the lesions are of a pale pink colour, and the limbs are little affected, especially below the elbows and knees.

In *lichen planus* some typical papules can almost always be seen, the lesions have a characteristic lilac or purple colour, the flexor aspects of the limbs are most involved, and itching is generally intense.

Treatment.—Internal treatment is considerably employed, but it is difficult to estimate its value. Arsenic is the most valuable drug, and should be given in increasing doses up to the limit of tolerance, but should be entirely discarded if no effect is produced. Liquor arsenicalis, min. 3, t.d.s. and upwards, and pil asiatica are chiefly employed. Arsenic should not be given when lesions are coming out rapidly. In these cases salicin, gr. 15, t.d.s., and thyroid, gr. 1, t.d.s., have been recommended by Crocker and others.

Local treatment is the most efficacious, and chrysarobin gives the best results, but is messy, stains linen permanently, and is liable to set up a severe dermatitis if used carelessly. It is best applied in 5 per cent. to 10 per cent. ointment rubbed into the patches daily, after the scales have been removed in a hot bath with the aid of soft soap. Dithranol (derobin, cignolin), is a less messy chrysarobin substitute, and used as an ointment, in the strength of gr. $\frac{1}{2}$ to the ounce, is a very efficient remedy. If this treatment is carried out thoroughly for 3 or 4 weeks the patches will disappear. This treatment is best carried out in hospital or in a nursing home.

Pyrogallic acid ointment 10 per cent., oil of cade ointment 20 per cent., or an ointment consisting of solution of coal tar 12·5, ammoniated mercury 6·25, salicylic acid 6·25, simple ointment to 100. If the patches become inflamed they are best treated temporarily with linimentum calaminæ. Isolated resistant patches may be treated by X-rays, but this cannot be often repeated. For the scalp, the scales should be removed with soft soap, and the patches painted with equal parts of solution of coal tar and industrial spirit. This latter solution is useful in psoriasis of the nails, after the nail has been cut away and the scales removed.

PARAPSORIASIS

This is a term applied to certain rare forms of resistant erythematous lesions which occur on the body. Three types are recognised: (1) parapsoriasis en gouttes, (2) parapsoriasis en plaques, and (3) parapsoriasis lichenoides.

Ætiology.—Nothing is known of the ætiology of these conditions.

Symptoms.—*Parapsoriasis en gouttes* occurs as pea-sized or slightly larger red spots, covered by fine branny scales, chiefly on the upper part of the trunk. In some cases necrotic lesions occur.

Parapsoriasis en plaques occurs as symmetrical patches, oval or linear, of pale yellow or red colour, with a shiny surface or covered by fine scales, and occurring chiefly on the legs, thighs and lower trunk.

Parapsoriasis lichenoides forms a reticular pattern, chiefly on the extensor

aspects of the upper limbs. The lesions are red or purplish in colour and the surface is either shiny or covered with fine scales.

Treatment.—These cases are very resistant to treatment, but should be dealt with on much the same lines as psoriasis.

PITYRIASIS RUBRA PILARIS

This is a rare disease characterised by the appearance of follicular papules, with horny spines, which tend to involve the whole cutaneous surface and eventually produce a generalised dermatitis resembling pityriasis rubra of Hebra.

Ætiology.—This is very obscure. It is thought by some observers to be a follicular form of psoriasis, but at present there is no conclusive evidence. The disease occurs in both sexes and at varying periods of life, but sometimes in the very young.

Symptoms.—The lesions are of two types: red follicular papules with horny spines, which are chiefly seen on the extensor aspects of the limbs, and especially on the dorsum of the hands and fingers; and red scaly plaques or sheets, which involve the scalp, face and trunk. Either of these types may predominate. Where the former type occurs the skin presents the appearance of a nutmeg grater. Hyperkeratosis of the palms and soles develops, with fissuring of the deeper folds, and the nails become pitted and brittle. Ectropion followed by conjunctivitis may occur. The mucous membranes usually escape. The malady does not seriously affect the general health, and subjective symptoms are usually absent.

Treatment.—No specific treatment is known. Thyroid has been recommended, but the results are very uncertain. Bran and alkaline baths, followed by inunctions of ac. salicyl., grs. 10, glycer. amyl., adip. lanæ hydros., āā gr. 240, seem to give the best results.

PITYRIASIS ROSEA

A widespread eruption of pinkish macules and papules of round or oval outline, with branny scaling, which does not usually extend to the periphery of the lesion.

Ætiology.—This is still obscure. It was originally thought to be of parasitic origin, probably owing to the resemblance of the lesions to ring-worm, but no definite parasitic has been discovered. It is possible that it may be of the nature of an acute exanthem, as it occasionally has a sudden onset with slight fever and malaise. One attack is said to confer immunity, though this is not absolute, and it seems to occur at times almost in epidemic form. It has been thought by others to be a toxic eruption. The disease occurs chiefly in children and young persons, but no age is exempt.

Symptoms.—The eruption usually comes out suddenly, and the onset may, though this is not usual, be accompanied by slight fever, malaise and sore throat. In some cases the general outbreak is preceded by the appearance of a single patch, the "herald" patch, which may appear a week or 10 days before the general eruption. The lesions are usually most profuse

on the trunk and central portions of the limbs, the distal parts of the latter escaping. The face, neck and scalp may be affected, but this is not common.

The lesions are pinkish macules or papules, mainly macules, which vary in size from a lentil to patches a couple of inches across. They tend to be arranged in lines parallel to the ribs, and the larger patches are oval. When the lesions reach the size of a pea, central scaling commences, and as the lesions grow the scales tend to form a collarette, with the free edge directed towards the centre. The colour of the portion within the scales changes to fawn and eventually returns to the normal skin tint. Sometimes pinhead-sized follicular papules of skin colour are seen among the lesions in considerable numbers. In rare cases vesicles and bullæ form.

Subjective symptoms are usually absent, but sometimes itching is a prominent feature. The rash usually lasts about 4 weeks and then disappears, but it may persist for several months.

Diagnosis.—Seborrhœic dermatitis is distinguished by its greasy scales and by its distribution; secondary syphilis, by the presence of other syphilitic lesions; and ringworm, by the small number of lesions, their asymmetry, and the presence of fungus in the scales.

Treatment—No internal treatment is known to affect the disease. In the early stages a simple coal tar and lead lotion can be used, as this allays itching if present. Once the rash is fully developed a daily warm bath, followed by the application of 3 per cent. salicylic acid in linimentum calcis, will usually cause the lesion to disappear. If there is fever the patient should be put to bed.

LICHEN PLANUS

An intensely itchy eruption characterised by the presence of angula papules of pinkish or lilac colour tending to be localised in special areas.

Ætiology.—The disease occurs chiefly in adult life and is very rare in young children. Two views are held as to its ætiology. By some it is thought to be of nervous origin, following shock, mental anxiety, worry, etc. It is true such a history is often obtainable, but Graham-Little has pointed out that it did not appreciably increase during the War of 1914–1918, which might have been expected if such was the main factor in its production. The other view is that it is of toxic origin, but there is no direct evidence on this score.

Pathology.—The microscopic anatomy of the papules is very characteristic. There is a circumscribed, dense, round-cell infiltration in the upper part of the dermis beneath the papule, and the epidermis is much thickened. The papillæ are flattened out. The granular layer is irregularly thickened and there is a hyperkeratosis, most marked at the orifices of the hair follicles and sweat ducts.

Symptoms.—The most common variety is the *localised type*. The lesions are chiefly found on the flexor aspects of the forearms and wrists, the inner aspects of the thighs near the knees, and on the front of the shins. The trunk, especially the lower part, the palms and soles, and the penis are also occasionally involved. The mucous membrane of the mouth is frequently attacked. The lesions on the skin are discrete, lentil-sized papules, raised

sharply from the skin, with polished shiny surface and usually of lilac colour. They have a curious and typical angular outline, due to the fact that they are bounded by the fine lines of the skin, and sometimes they are definitely umbilicated. Frequently the papules are arranged in lines along scratch marks. Occasionally patches are formed by the aggregation of papules and resemble rather closely patches of psoriasis. On the palms the lesions are generally circular, vary in size from a pea to a threepenny-bit, and the horny layer over them is much thickened.

In the mouth irregular dead white patches are found, usually on the tongue or inner aspect of the cheeks.

Several other types of lichen planus are seen. In one variety the lesions form rings and gyrate figures (*lichen planus annularis*), while in another atrophy occurs (*lichen planus atrophicus*). In a large number of cases the horny layer is much thickened (*lichen planus hypertrophicus*), and this is especially seen on the legs, where warty patches occur (*lichen planus verrucosus*). Occasionally the lesions are arranged in a single line, sometimes following the course of a nerve (*lichen planus linearis*), and very occasionally bullæ and vesicles may occur. Rarely cases are met with in which the papules are few in number, and very much hypertrophied, forming dome-shaped tumours, which itch intensely (*lichen obtusus*).

Another variety is an *acute generalised* type in which a large number of lesions are scattered diffusely over the trunk and limbs. In this type the papules are pale pink in colour and not so raised as in the chronic forms. Even in these cases the face and scalp almost invariably escape.

In association with the above-mentioned lesions small groups of follicular papules with horny spines may be found. The same condition is sometimes found independently of lichen planus and has been called *lichen pilaris* or *spinosus*. Whether these latter cases have the same origin is still unsettled. Graham-Little has reported the association of this type with atrophic alopecia.

Lichen planus lesions are almost invariably accompanied by intense itching and often by marked neurotic manifestation, but otherwise the health remains good, though slight fever may accompany the acute cases.

The disease runs a very chronic course and is sometimes very resistant to treatment.

Diagnosis.—This is usually easy, as the lesions are very characteristic. The diagnosis from psoriasis has already been dealt with. From lichenification (*lichen simplex chronicus* of Vidal), which is produced by friction on the skin, the diagnosis is made by the fact that the latter only occurs in circumscribed patches, and that the typical discrete papules of lichen planus are absent.

Treatment.—In the acute generalised cases rest in bed is essential, and it is of the greatest service in chronic cases, materially shortening their course. Arsenic is looked upon as a specific, but may require to be pushed; arsphenamine (salvarsan) has been recommended by some authors. As in psoriasis, arsenic is not advised in cases in which the eruption is coming out, these cases doing best on mercury; liq. hydrarg. perchlor., min. 60, t.d.s. If the irritation is very bad, bromides or some hypnotic at night may be required, and for this symptom lumbar puncture has been recommended.

For local treatment anti-pruritic lotions and ointments are required; of these ichthammol, oil of cade, coal tar, phenol and menthol are most useful.

For the hypertrophic patches, ac. salicylic plaster, followed by X-rays or CO₂ snow, is the most satisfactory method of treatment.

SCLERODERMA

This is a condition of hardness and rigidity of the skin, caused by a degeneration of fibrous tissue, which is probably of inflammatory origin. It is met with in two forms—(1) Generalised scleroderma, and (2) localised scleroderma or *morphœa*.

Ætiology.—It is chiefly a disease of young adult life and is more common in women than in men. It has been variously attributed to a tropho-neurosis, to alterations in endocrine secretion, to an endarteritis, and to a primary hyperplasia of the fibrous tissue of the skin. Recent work by Dowling and Griffiths suggests that a relationship exists between this disease and thyrotoxicosis.

Pathology.—There is a degeneration of the fibrous tissue bundles in the dermis and subcutaneous tissue, with replacement of fat by fibrous tissue in the latter. There is also an exudation of cells around the vessels of the dermis, with some endothelial proliferation. The epithelium may be flattened by pressure and excess of pigment may be present. Degenerative changes may also be seen in the muscles in some cases.

Symptoms.—1. *Generalised scleroderma.*—This condition may appear rapidly or slowly. In both cases a disturbance of the general health, such as fever, joint pains, neuralgia or itching, may precede or accompany the attack. Stiffness of the parts involved is often the first symptom, and this may spread rapidly or slowly till it produces fixity of the joints, followed by progressive wasting of the muscles. Breathing may become difficult, owing to fixation of the skin of the chest, and taking of solid food may be prevented by the involvement of the cheeks and mouth. The skin appears swollen and glossy and is very hard; the deeper structures are fixed and the furrows of the skin disappear. The colour of the skin may be normal or waxy in appearance. The lesions are usually symmetrical and the mucous membranes may be affected. These cases may occasionally clear up spontaneously, but often end fatally.

Occasionally the disease begins in the hands. The skin is drawn tightly over the fingers, fixation of the joints occurs and atrophy supervenes, so that the fingers become pointed. This type is called *sclerodactylia*.

2. *Localised scleroderma or morphœa.*—In this type the lesions vary from the size of a pea to large patches involving almost all the back or front of the trunk. Patches may be pinkish in colour and raised, with a smooth polished surface and with a sensation of rigidity; or slightly depressed below the surface, very rigid and fixed, and often surrounded by a lilac border, and occasionally occurring in bands; or again they may be of dead white colour, with more or less irregular edges, and of normal consistence. This latter type is one of the forms of so-called *white-spot disease*.

These cases run a very chronic course, often of many years, and are resistant to treatment.

Diagnosis.—From sclerema neonatorum, by the age of the patient and the fact that this condition is confined to the subcutaneous fatty layer.

Treatment.—In generalised cases, the patient should be kept warm, and massage and hot air baths given. Cod-liver oil internally is of value. Thyroid is largely given in all varieties, but its action is very uncertain. In morphœa, local fibrolysin injections have been given with success. X-rays are claimed to be beneficial. Treatment, however, is unsatisfactory.

SCLEREMA NEONATORUM

This condition has no relationship to the foregoing. It occurs in newly-born infants, and is characterised by hardening of the subcutaneous fatty layer in certain parts of the body.

Ætiology.—It has been attributed to hardening of the fat, owing to lowering of body temperature, but this is certainly not an essential factor. There is evidence of a deposit of crystals in the tissues, and of a well-marked proliferation of the reticulo-endothelial cells in the neighbourhood of these deposits. This is probably due to a reduction in the olein in the fat, but how this comes about is not yet known.

Symptoms.—The affection usually begins within a day or two of birth. It occurs symmetrically and chiefly affects the calves, thighs, buttocks and back. The subcutaneous fat becomes very hard, and does not pit on pressure. The edges are well-defined. A certain number of cases die, but in the less severe cases the patches disappear in a month or two. A generalised hardening of the fat occurs all over the body in infants suffering from severe diarrhœa, but this condition appears to have no relation to that just described.

Treatment.—The child should be kept warm and given plenty of nourishment. Cod-liver oil is said to be very beneficial.

D—THE LYMPO-GRANULOMATA

These cases form a bridge between the inflammatory dermatoses on the one hand and the new-growths on the other. The lesions in many ways resemble the granulomata produced by bacteria, and in other ways resemble sarcomata. The following conditions are included: (1) Sarcoidosis, (2) leukæmia cutis, (3) lymphadenoma cutis, and (4) mycosis fungoides.

SARCOIDOSIS

Synonyms.—Besnier-Boeck-Schaumann's Disease; Lympho-granuloma benigna (Schaumann).

Dermatologists have been familiar for many years with two conditions of the skin which from their histological characters have been thought to be tuberculous. The first, which consists of a symmetrical granulomatous condition affecting chiefly the nose, cheeks, ears and fingers, was described by Besnier in 1889 under the name "lupus pernio," and differentiated by him from lupus vulgaris and lupus erythematosus, with both of which conditions

it has some features in common. The second condition, described by Boeck in 1897, was characterised by the presence, generally on the face, of granulomatous swellings, single or multiple, of varying size and of a translucent appearance, often showing small yellowish points closely resembling the "apple jelly nodules" seen in lupus vulgaris. To these lesions Boeck gave the name of "cutaneous sarcoids." In 1917 Schaumann showed that these two conditions were only clinical types of the same disease, and he further showed that they were merely cutaneous manifestations of a pathological process which affected many other organs in the body, the lymphatic system, bone-marrow, lungs and other viscera. It is now recognised as a diffuse reticulo-endotheliosis, somewhat resembling, though clearly distinct from, Hodgkin's disease. Schaumann has suggested that the condition should be called "lympho-granulomatosis benigna," but at the present time the name "Sarcoidosis" has been tacitly adopted in this country.

Ætiology.—The condition is not very common in the British Isles but more frequent on the European continent, especially in the Scandinavian countries. It affects both sexes equally, and is most frequent between the age of 20 and 40. It is very rare before puberty but isolated cases have been described even in infancy.

There is much controversy as to the cause of the condition. The histological architecture, described below, has long suggested a tuberculous ætiology, but certain features have raised doubts on this point. The absence of caseation in the lesions; the fact that the vast majority of the cases give negative tuberculin reactions; that acid-fast bacilli have not been found in the lesions, except in one or two cases in which the clinical diagnosis was by no means certain; and the very indecisive results of animal inoculations experiments—these are points which make the acceptance of the tuberculous hypothesis difficult. Schaumann has obtained the bovine tubercle bacillus from the sputum of some patients suffering from this disease, and has pointed out that active tuberculosis sometimes develops in cases of sarcoidosis and that when this happens the characteristic lesions disappear. He holds the view that sarcoidosis is an indication of a particular phase of immunity to the bovine bacillus, and supports the view of Jadassohn that the high percentage of negative tuberculin reactions is not due to passive anergy, indicative of an absence of tuberculous infection, but is due to an active anergy occurring in a certain phase of this infection.

Certain South American dermatologists have noted the close resemblance of sarcoidosis to tuberculoid leprosy, and have suggested that it may be a form of leprosy. The geographical distribution of the two diseases, however, does not correspond, and this seems to rule out this hypothesis. Lastly other observers think that the disease is an entity due to specific organism not yet discovered. This theory is at present largely based on negative evidence. It is only possible at the present time to say that the ætiology of sarcoidosis is unknown, but that it is difficult to ignore certain similarities to tuberculous infections.

Pathology.—Although the pathological changes were first only described in skin lesions, it is now known that similar changes may occur in many organs of the body. They have been found in the mucous membranes, the lymphatic system, the lungs and other viscera, the bone-marrow and certain other structures. The lesions consist essentially of nodules of epithelioid cells,

often though not always showing a few giant cells in the centre, surrounded by dense bands of normal connective tissue. Sometimes a zone of lymphocytes is found around the epithelioid cells, but these are often very sparse. Fibrous and elastic tissue is entirely destroyed by these granulomatous deposits, and bone is absorbed when invaded. The nodules show no signs of undergoing necrosis or caseation, but in older nodules fibrous tissue infiltration may occur; in healing nodules the cellular structure is gradually replaced by fibrous tissue.

As is mentioned above a very high percentage of cases show a negative tuberculin reaction, a percentage higher than is found in a series of apparently healthy individuals, and this has suggested the possibility of an active anergy and is thought by Jadassohn to be due to the presence of excess of anti-cutins in the blood and indicative of a tuberculous infection.

The blood picture usually shows little abnormal, but a monocytosis has been described in some cases.

Symptoms.—*Skin.*—Skin lesions are of several types and can roughly be classified into (1) the small nodular sarcoid, (2) the large nodular sarcoid, and (3) lupus pernio. In the small nodular sarcoid the lesions come out more or less symmetrically, and are most common on the face but may be found, often in considerable numbers, on other parts of the body, especially the shoulders and upper limbs. The nodules vary in size from a pinhead to a pea, are smooth dome-shaped papules of yellowish-brown colour, translucent in appearance.

The large nodular sarcoids are often present singly or in small numbers, and are not always symmetrical in arrangement. Again, the face is the site of predilection, the forehead, nose and prominences of the cheeks being the usual sites, but they are found also on other parts of the body. The lesions vary in size from a pea to plaques an inch or so across. They are raised above the skin, smooth and shiny in appearance, reddish to purplish-brown in colour, and soft to the touch. On glass pressure they exhibit translucency, and often "apple jelly" nodules are visible. These differ from those of lupus vulgaris in not breaking down under pressure from a pointed match.

Both the small and large sarcoids can disappear spontaneously or as a result of treatment, and in this case some atrophy of the skin results. In the large variety annular lesions, with a pale atrophic centre and a flattened purplish margin, are sometimes met with.

In lupus pernio the lesions form less well-defined infiltrations arranged more or less symmetrically, the face again being most affected. The nose and cheeks show marked swelling with ill-defined outline and are of a dull purple colour, similar to that seen in chilblains, though the skin is warm and not cold. The ears are also swollen and purplish in tint, especially the lobules. In this condition the skin of the hands and feet are also frequently affected, similar ill-defined purplish swelling involve the fingers and toes, and dorsum of hands and feet; this infiltration may extend up the forearms and legs. Lesions of the large and small sarcoids may be present at the same time. In these cases infiltrations of the subcutaneous tissue are sometimes present, especially on the forearms and arms.

Rare cases of generalised erythrodermia have been described in sarcoidosis. The skin becomes red and scaly over considerable areas of the body, or the eruption may be universal. A similar type of eruption is some

times seen in lymphadenoma or leukæmia. Nodules and diffuse infiltration of the mucous membranes, chiefly in the nose, but also in the pharynx, larynx and mouth have also been described.

Lymphatic system.—Palpable enlargement of accessible lymphatic glands is often present, though this enlargement may be limited to certain glands, of which the epitrochlear are the most common. When examined microscopically these glands reveal the same changes as are seen in the skin. Enlargement of the peribronchial glands is even more common, and can usually be seen in a radiogram of the chest. Of other lymphatic tissue the tonsil is frequently affected, and biopsies of the tonsil are sometimes useful for diagnostic purposes.

Bones.—In many cases of lupus pernio, swellings of the fingers and toes suggestive of tuberculous dactylitis have been observed. These, however, never showed any signs of breaking down. Radioscopic examination has shown that these swellings are mainly due to deposits in the connective tissue surrounding the small bones of the fingers and toes, but it has also demonstrated that in the bones themselves areas of rarefaction and cystic spaces can be seen, sometimes so extensive that the surrounding compact bone of the phalanx may collapse. This change, which may occur in cases where the fingers look quite normal, affects mainly the phalanges and heads of the metacarpal and metatarsal bones, but may occasionally occur in the long bones and even the spine is known to have been affected. These changes are of considerable diagnostic importance.

Lungs.—Patients suffering from sarcoidosis rarely show any symptoms of pulmonary involvement, such as cough or expectoration. Since, however, radioscopic examination of the chest has become a routine, changes have been demonstrated which are more or less diagnostic of the condition. In addition to enlargement of the hilar glands, mentioned above, most cases reveal evidence of deposits of granulomatous tissue in the peribronchial regions. These changes take the form either of a diffuse mottling due to the presence of small nodules scattered along the bronchi, or a reticulation or marbling, showing a more diffuse infiltration along the bronchi. In no case is there evidence of cavity formation.

Other viscera.—Lesions in most of the viscera have been described. Of these the spleen is most frequently affected and may become much enlarged, reaching down to the iliac crest. The liver may also be enlarged but usually to a less degree. Lesions have also been found in the heart, stomach and kidneys. In the case of the last, albuminuria and hæmaturia may be present.

Nervous system.—Involvement of the peripheral nerves, giving rise to localised anæsthesia, paresis and muscular atrophy, has been recorded. Lesions have also been described in the brain and in the pituitary body.

Ocular lesions.—Small yellow nodules have been found on the conjunctiva but special interest attaches to lesions of the uveal tract. These may take the form of an iritis; of nodules on the iris; of exudate with the anterior chamber; and opacities in the vitreous. White patches have also been observed in the choroid. These eye changes may be found in association with infiltration in the salivary glands, especially the parotid, and the condition known as uveo-parotitis or Heerfordt's disease is now known to be a manifestation of sarcoidosis (see p. 532).

General symptoms.—Patients suffering from sarcoidosis generally appear

to be in remarkably good health. Fever is generally absent but in rare cases persistent fever is present. It is not usually high, about 100° F., but may continue without intermission for months. The pulse in these cases is scarcely raised and the patient's general condition is good.

It should be remembered that in most cases only a few of the lesions mentioned above may be present, and that although the skin lesions are the most prominent numerous cases are now on record in which skin lesions are absent.

Diagnosis.—In cases where skin lesions are present they are usually sufficiently typical to enable a diagnosis to be made, but a biopsy can be done to confirm this. The presence of characteristic lesions seen in radiographs of the chest and hands mentioned above are of further assistance, as is a negative tuberculin test. The absence of necrosis and caseation is a useful sign in differentiating from classical tuberculous lesions, while the absence of follicular plugging distinguishes these cases from lupus erythematosus. The diagnosis from syphilis can be made by a careful consideration of the characteristic symptoms of that disease, assisted by a positive Wassermann reaction.

Prognosis.—It is generally held that the milder types of the disease eventually clear up spontaneously. According to Schaumann, however, some cases eventually develop frank tuberculosis, and in these the symptoms of sarcoidosis disappear. Severe cases may die from involvement of vital organs.

Treatment.—The best results have so far been obtained by the administration of chaulmoogra oil preparations or sodium morrhuate. The latter drug is best given either intravenously or intramuscularly in a 3 per cent. solution: 1 to 3 c.c. being administered weekly. It may be necessary to give the drug over a considerable period of time. There can be no doubt that lesions gradually disappear under this treatment but there is some tendency to recurrence, just as there is in the treatment of leprosy by the drug. Milder cases seem to be cured, but it is not easy to say the same of the more advanced cases. Chaulmoogra oil preparations are administered in the same doses as for leprosy. Arsenic also appears to have some influence on the lesions, and is best given in the form of sulpharsphenamine intramuscularly, doses of 0.3 to 0.45 gm. being administered weekly in courses of 10 injections.

No local treatment has much influence on the lesions, but general arc-light baths appear to be of value.

LEUKÆMIA AND LYMPHADENOMA CUTIS

Symptoms.—In both these conditions itching may be a marked symptom; sometimes it occurs without any cutaneous lesions, while at other times very persistent urticarial or prurigo-like lesions are present. Hæmorrhages may occur into the skin, and in some cases exfoliative dermatitis is present.

The more characteristic lesions are, however, granulomatous infiltrations of the skin, which form tumours, either in certain localities or more or less all over the skin. In the former case the face is most affected, the tumours forming chiefly on the forehead, about the nose and on the cheeks, producing a leonine appearance. The lesions vary from a pea to an orange in size, and

are usually of a dull purplish-red colour. Most of the cases recorded occur in lymphatic leukaemia, but a few have been reported in the myeloid cases. They also occur in Hodgkin's disease, but there is great difficulty in distinguishing aleukæmic leukaemia from Hodgkin's disease, unless the glands have been examined microscopically.

Treatment.—This has been dealt with in the articles on Leukæmia and Hodgkin's disease (pp. 802 and 841).

MYCOSIS FUNGOIDES

A chronic inflammatory dermatosis with a tendency to form granulomatous tumours, which usually ends fatally.

Ætiology.—It is a disease of late middle life, and more common in men. Its cause is quite unknown, but it is probably an infective process and closely related to leukaemia, though no characteristic blood changes have been observed.

Symptoms.—In the early or premycotic stage the most frequent lesions are patches of redness and scaling, associated with intense itching. After a time these lesions become infiltrated and raised above the surface of the skin. Later, tumours appear in these patches, usually about the size of an orange, but not infrequently much larger. The epidermis over them gives way and a fungating mass is produced. These tumours are usually multiple.

Sometimes the initial lesion takes the form of an eczema, an urticaria or a dermatitis exfoliativa, but in all these cases itching is a prominent symptom. In other cases the tumours appear without any pre-existing dermatosis.

The course is slow, and the general health is affected first by loss of sleep and then by septic absorption. Practically all cases eventually terminate in death.

Diagnosis.—This may be very difficult in the premycotic stage. The itching and the persistence of the symptoms in spite of treatment, together with the age of the patient, will help in coming to a diagnosis.

Treatment.—The only treatment known to benefit these cases is X-rays or radium. Either of these will keep the lesions quiescent for a considerable time, but recurrence generally takes place sooner or later. Arsenic and antimony may also be given.

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V. TUMOURS OF THE SKIN

Tumours can be divided into epithelial and connective-tissue tumours, and each of these varieties into benign and malignant.

BENIGN EPITHELIAL TUMOURS

1. WARTS

These are benign epithelial tumours, characterised by an overgrowth of the prickle-cell layer, with or without hyperkeratosis, and produced by an

infective agent, which appears to be a filter-passing virus. The following types are recognised :

1. *Verruca vulgaris*.—This is the common wart which is so frequently met with on the back of the hands, but may occur on the face and other parts of the body. The lesions are raised tumours, varying in size from a pinhead to a filbert, and are usually discrete, but may group to form larger swellings. They have a rough surface, rise sharply from the surrounding skin, and are skin-coloured. Histologically they show great hypertrophy of all the epithelial layers, with downward growth of the interpapillary areas, and a corresponding papillary elongation. They occur chiefly in children. They are inoculable from one spot to another, and from one individual to another.

Treatment.—Isolated lesions are best removed by the application of CO₂ snow for from 15 to 40 seconds, according to their size. They may be burnt away with glacial acetic, trichloracetic, or nitric acid, silver nitrate or the actual cautery. When very numerous, evulsion with a sharp spoon or X-rays are most satisfactory.

2. *Verruca plana juvenilis* are pinhead-sized warts, seen chiefly on the face and hands of children, though they are met with in adults. They have smooth, flat tops, and are usually very numerous.

Treatment.—They are best treated by touching with the galvanocautery, or by magnesium ionisation.

3. *Verruca plantaris*.—This is a wart which occurs on the sole of the foot, and has the appearance of a corn, because it is surrounded by a hyperkeratotic ring, and on account of the pressure on the foot does not stand up above the level of this ring. It is, therefore, often mistaken for a corn. It is usually extremely painful.

Treatment.—The most satisfactory treatment is by X-rays; 1½ to 2 skin units are usually required, the surrounding zone being carefully screened. These warts may also be removed by salicylic acid plaster, followed by touching with acetic acid, by CO₂ snow, or by evulsion.

4. *Verruca filiformis*.—These minute warts are usually about 1 mm. in diameter at their base, with long filamentous processes. They are sometimes found on the face; but chiefly occur on the genitals, and around the anus. They frequently occur in very large numbers, forming cauliflower-like growths. They are often seen in cases of gonorrhœa, and are sometimes known as *gonorrhœal warts*; but they may be present apart from this disease, and are probably due to some other infecting agent. In these warts there is very little overgrowth of the horny layer.

Treatment.—Locally antiseptic applications, such as 1 in 1000 perchloride of mercury in spirit painted on frequently, or silver nitrate 3 per cent. in sp. æth. nitros., will cause them to dry up, or they may be removed with the galvanocautery, or painted with trichloracetic acid.

5. *Keratoma senile*.—These co-called senile or seborrhœic warts are commonly seen on the face, back and chest of old people; but may occur in younger persons. They vary in size from a pea to a filbert, and are only slightly raised from the skin. They are soft to the touch, and have a slightly warty surface. Their colour varies from yellow to a deep black, and they may itch a good deal. The distribution is much the same as for seborrhœic dermatitis.

Treatment.—Washing with soap and water, followed by the application

of 10 per cent. ac. salicyl. ointment will often remove them. Failing this, painting with trichloroacetic acid, freezing with CO₂ snow, or a skin unit of X-rays should be employed.

2. ACANTHOSIS NIGRICANS

This is a rare condition in which warty pigmented growths appear on the neck, axillæ, groins, umbilicus, and flexures of the limbs and on the face. These growths often fungate and suppurate, especially in moist areas. In addition areas of pigmentation and scattered warty growths may occur. The mucous membrane of the lips, cheeks and tongue may be affected. In a large proportion of cases abdominal malignant growths have been found; but in other cases no such complication exists. The nature of the condition is not understood.

Treatment.—No treatment is known to affect the condition.

THE DYSKERATOSES

1. MOLLUSCUM CONTAGIOSUM

In this condition small tumours appear on the skin which, like the warts, are infective and produced by a filter-passing virus. They differ from warts not only in clinical characters, but in the peculiar degenerative changes which occur in the process of horny cell formation.

Pathology.—The most striking feature in a section is the presence between the Malpighian and horny layer of large cells containing large transparent oval bodies. These are known as "molluscum bodies," and at one time were thought to be coccidial bodies, but are now considered to be degenerations of the cell protoplasm.

Symptoms.—The lesions consist of small lentil- to pea-sized bodies of a white or pinkish colour, with a smooth glistening surface. They may appear anywhere on the skin. They are dome-shaped, and have a central pit, in the floor of which the thickened horny layer can be seen. Not infrequently they become inflamed.

Treatment.—By taking a sharpened match, and introducing it into the central depression, the whole horny mass can be forced out, and if the cavity be painted over with pure carbolic acid a cure will result.

2. DARIER'S DISEASE

This is a very rare condition of the skin usually seen in young adults in which an eruption of follicular papules develops on the face, scalp, abdomen, back, and the flexor aspects of limbs. The lesions run together and form warty-looking masses. The disease is slowly progressive, and the lesions are resistant to treatment, but the general health is not affected.

Microscopic examination shows similar changes to those seen in molluscum contagiosum, namely, an irregular hyperkeratosis with formation of "psorosperms" in the region of the granular and Malpighian layers of the epidermis.

Treatment.—This consists of baths and the application of keratolytic agents, such as salicylic acid.

3. PAGET'S DISEASE OF THE NIPPLE

This is a chronic affection which usually develops around the nipple in middle-aged women, but has been described in other parts of the body and in men. It is seen as a sharply defined, red, oozing area involving the nipple, the areola, and the skin around for a short distance, and is almost always unilateral. The whole area has a distinct parchment-like induration. The nipple becomes retracted, and eventually disappears. The condition is associated with carcinoma of the breast, and whether it is primary or secondary is still a matter of dispute.

In this condition, as in the two diseases just referred to, "psorosperms" are seen under the microscope. The surface horny layer is mostly lost, and the deeper layers of the epidermis are much hypertrophied and cedematous, but show no obvious epitheliomatous proliferation.

Treatment.—Amputation of the breast is the only treatment that can be advised.

MALIGNANT EPITHELIAL TUMOURS

1. RODENT ULCER

A slowly growing epithelioma usually single, but sometimes multiple, which may cause considerable local destruction of tissue but does not form metastases.

Pathology.—This variety of epithelioma is usually described as a basal-cell epithelioma. In section epithelial processes are seen penetrating into the underlying dermis and subcutaneous tissue; but the processes are bounded by a regular basal layer of cubical cells, and although degeneration cysts may form in these processes no cell-nests are formed.

Symptoms.—The lesions chiefly occur in old people; but this is not always so. They also rarely begin anywhere but on the face, and then chiefly in the neighbourhood of the eye, or on the nose or cheek. At first a small raised white nodule appears, with small vessels coursing over it. Then, as it spreads, an ulcer forms in the centre, but the raised intensely hard white border persists. If not treated a great deal of tissue destruction occurs; the nose may be destroyed, or the antrum perforated, and the whole of the nasal cavities opened up. In advanced cases practically the whole face is destroyed. Some cases, however, remain superficial, spreading slowly, the older parts healing as the lesion spreads. Multiple lesions are not very rare.

Benign forms are also recognised. In one the lesions are pea-sized or slightly larger nodules, scattered over the face, and of the same type as the early lesions referred to above. Sometimes they undergo cystic change. They were described by Brooke under the name of *epithelioma adenoides cysticum*. Another benign type is seen in which multiple walnut-sized tumours form on the scalp (*Spiegler's tumours*). A third type occurs in the form of multiple psoriasiform patches on the trunk, and has been named by Graham-Little *erythematoid benign epithelioma*.

Diagnosis.—This can always be made in cases of doubt by microscopic examination.

Treatment.—Excision is the best treatment when possible ; but excellent results are obtained by radium, or X-rays. CO₂ snow has been advocated for early cases, and the results obtained are excellent. In advanced cases, which are unsuitable for surgical treatment, diathermy is useful.

2. SQUAMOUS EPITHELIOMA

In this condition rapidly growing tumours form, which ulcerate and cause local destruction of tissue, and also cause secondary glandular involvement. It is chiefly a disease of old age.

Pathology.—Sections show an irregular proliferation of the Malpighian layer, with the formation of cell-nests, and the limiting basal layer is absent.

Symptoms.—The lesions begin as nodules, much like those of rodent ulcer, but they spread much more rapidly and either form irregular deep cut ulcers, without the characteristic edge seen in rodent ulcer, or else they become raised and form mushroom or cauliflower-like growths. The glands may be involved, and general dissemination may occur. The condition may sometimes supervene on pre-existing non-malignant conditions. It may commence in a keratoma senile, in the warty conditions which occur in cases of atrophy of the skin due to exposure to tropical sun, on an old lupus scar, on X-ray dermatitis, in xeroderma pigmentosa, in arsenical keratoses and in tar molluscum.

Treatment.—This is purely surgical, and consists of erasion of the local growth and of the glands draining the area concerned. Radium is now extensively used in treating these growths. In the case of epithelioma complicating lupus vulgaris, however, secondary glandular involvement does not appear to occur, and local destruction with arsenic paste, or by diathermy, gives even better results than excision.

TUMOURS OF THE APPENDAGES OF THE SKIN

1. MILIUM

In this disease pinhead-sized yellowish-white bodies are seen in the skin of the face, chiefly on the cheeks, eyelids and forehead. They are often very numerous. They can be shelled out, and are found to consist of a whorl of epithelial cells. Their origin is unknown, but they are probably derived from the lanugo hair follicles.

Treatment.—These tumours can be destroyed by electrolysis.

2. SEBACEOUS CYSTS

These are painless cystic swellings chiefly found on the scalp, face, ears, back and scrotum. They vary in size from a pea to an orange. When incised they are found to be filled with cheesy matter. They are either due to blocking of the sebaceous duct, or according to some authorities they are of embryonic origin. True *dermoid cysts* of the skin are also found.

Treatment.—Excision is the most satisfactory method of treatment.

3. ADENOMA SEBACEUM

A symmetrical eruption of pinhead-sized, bright red papules, of congenital origin, on the face. It commences very early in life, and is often associated with mental defect—in fact, cases are most often seen in asylums. The lesions are distributed chiefly over the nose and cheeks, and consist of hypertrophied sebaceous glands and numerous capillary vessels.

Treatment.—The lesions can be destroyed with the galvano-cautery, by electrolysis or by diathermy.

Tumours of the sweat glands and ducts are so rare as to need no description here.

CONNECTIVE-TISSUE TUMOURS

1. KELOID

This is a fibrous tumour developing in a scar. The mere overgrowth of a scar is sometimes referred to as a *hypertrophic scar*, while the term keloid is limited to those cases in which the tumour extends beyond the original limits of the scar. In this latter condition processes often grow out in all directions like tentacles, and also in some cases the condition appears to start spontaneously from the normal skin; but there can be little doubt that some small abrasion was present. Small keloids often appear after acne vulgaris, varicella and other dermatoses which lead to scarring. There can be little doubt that this fibrous overgrowth is due to some chronic bacterial infection of the wound—probably a staphylococcal infection.

Treatment.—The best results are obtained by radium and X-rays.

2. FIBROMA—MOLLUSCUM FIBROSUM

Hard fibromata of the skin are rare, and usually occur in pear-sized nodules scattered about the skin. Soft fibromata are common, and are met with as small pedunculated tumours, chiefly on the trunk. They may occur in large numbers, and are then described as molluscum fibrosum. In this condition the tumours vary in size from a small pea up to several inches in diameter. Not only the skin but the mucous membranes may be the seat of these tumours. At times they form huge dependent unshapely masses, which completely disfigure the part from which they arise; this condition is called *dermatolysis*. Not all the tumours are pedunculated—as some are sessile—but all have the same softness. Some definitely surround nerve trunks, and it has been thought that they all develop in connection with the nerve fibres; hence they are often called *neuro-fibromata* or *plexiform neuromata*.

In some cases true neuro-fibromata and molluscum fibrosum lesions occur in combination with pigmented spots about the body. This syndrome is called *Recklinghausen's disease*, and is sometimes associated with mental disturbance.

Treatment.—Nothing can be done except surgical removal of the tumours, and this is only occasionally necessary.

3. LIPOMATA

These are soft freely movable lobulated tumours in the subcutaneous tissue, and may be single or multiple. One variety is very painful and associated with general adiposis, and is referred to as *Dercum's disease* (p. 1480).

Treatment.—Excision is the only treatment.

4. MYOMATA

Small multiple tumours of the size of a pea are sometimes found which have the structure of leiomyomata, and arise from the arrectores pilorum muscles. The lesions are often numerous, grouped and painful.

Treatment.—The cautery, or excision, is the only treatment.

5. MULTIPLE IDIOPATHIC SARCOMA OF KAPOSI

This is a curious condition chiefly seen in old people, and generally in the natives of Eastern European countries; but cases have arisen *de novo* in this country. The lesions occur chiefly in the region of the ankles, but have also been found on the hands and on the trunk. They are irregularly shaped red plaques raised from the skin, and of firm consistence. Histologically they consist of an overgrowth of fibrous tissue with dilated blood spaces. Whether this condition is of inflammatory origin, or is a species of *nævus* is as yet undecided. The condition does not affect the general health.

Treatment.—No treatment is known to affect the condition.

6. SARCOMATA

Both round- and spindle-celled sarcomata have been found arising in the skin, but are rare. They may be single or multiple, of any size, sessile or pedunculated, and are usually of a purplish-red colour. They tend to break down and produce fungating ulcers, and run a rapid course ending in death unless removed in the early stages.

Treatment.—This is purely surgical.

7. NÆVI

This term should be applied only to certain new formations of congenital origin; but in practice certain other conditions have been included. They fall into four classes: (1) Vascular *nævi*; (2) lymphatic *nævi*; (3) pigmented *nævi*; and (4) hyperkeratotic *nævi*.

VASCULAR NÆVI.—There are two chief varieties, the capillary and the cavernous *nævi*.

Capillary *nævi*.—These *nævi* are essentially dilatations of the capillary vessels of the papillary and subpapillary layers of the dermis. They form flat red patches of varying size. They may be small pea-sized lesions, or they may practically cover the whole body, including the mucous membranes. When they occur in large patches they are called *port-wine stains*.

The lesions are usually not raised and not infiltrated, the only change being in the colour of the skin, which is red or purple in the affected areas.

Sometimes, however, thickenings occur irregularly throughout the patches. On examination with a lens the capillaries can often be seen. These *nævi* are either present at birth or appear shortly afterwards. They tend to get paler as age advances, but rarely disappear.

Treatment.—These cases are very difficult to treat, especially the more extensive ones. CO_2 is useless, unless the application is sufficiently long to destroy the skin. Radium, though sometimes successful in removing the *nævus*, is inclined to produce atrophy and telangiectases in its place. The best hope in disfiguring cases rests with excision and plastic surgery, though some fair results have been obtained with diathermy.

Cavernous *nævi*.—These are soft or hard tumours, which appear as bright red sharply defined swellings raised above the level of the skin or as purplish indurations in the skin and subcutaneous tissue. The blood can usually be squeezed out of them by pressure. They consist of a fibrous stroma surrounding irregular blood spaces, the whole being more or less encapsuled. The tumours vary much in size and extent, some being as large as a cricket-ball or larger. Any part of the body may be affected, but they are frequent on the face and scalp, the lips and eyelids often being attacked.

Treatment.—Small tumours are best treated with CO_2 snow, one exposure of 15 to 30 seconds being sufficient to remove them. Larger tumours may be dealt with by repeated applications of snow, but they do better with multiple punctures with a fine galvano-cautery at dull red heat. In some situations excision is the best treatment, while radium can often be used with success. Electrolysis was formerly much employed, but is slow and has been superseded by the methods mentioned above. It is often advisable to abstain for a time from active treatment, as there is a tendency for the tumours to disappear spontaneously.

Stellate *nævi*.—These are not strictly *nævi* at all—that is, they are not congenital growths. The cause is not clear, but they may be degenerations or possibly traumatic dilatation of venules. The lesions consist of a central pinhead-sized dilatation of a venule, with a stellate arrangement of dilated vessels running into it. They are seen chiefly in children on the face, but also occur in adults. It has been thought that insect bites may be a determining cause.

Treatment.—If the central vessel is destroyed by a fine galvano-cautery or by electrolysis the lesion will disappear.

LYMPHATIC NÆVI.—Synonym.—Lymphangioma Circumscriptum.

This occurs as a raised circumscribed patch of skin colour, which on close examination is seen to consist of closely grouped vesicles, varying in size from a pin's head to a lentil. There may be a few discrete vesicles surrounding the main patch. In some cases, too, the surface is warty. The patches appear at or soon after birth, but may come out later. Microscopic examination shows dilatation of the lymphatic vessels of the dermis, with or without epidermal hypertrophy.

Treatment.—Excision, cauterisation or treatment by radium are the three methods applicable.

PIGMENTARY NÆVI OR MOLES.—*Nævi* of this class are very numerous and vary considerably in type. They may consist of pigmented patches of varying size and various depth of colour from a pale yellow to a deep black. These may be associated with hairy growths. In other cases smooth lobulated

pigmented tumours may occur on any part of the skin. Some cases have a rough, warty surface, while others are hairy. They may be quite small, no larger than a pea, or may cover large areas of the body, and have a distinct tendency to occupy segmental areas. They may appear at or soon after birth, or may occasionally develop later in life. The histological picture is characterised in all types by the presence of masses or columns of round embryonic cells in the dermis and also in the deeper layers of the epidermis. There is excess of pigment in the cells of the basal layer, in the adjoining Malpighian layer, and also pigmented wandering cells in the upper part of the dermis. The epidermal changes vary with the type of nævus.

There is a slight tendency for these pigmented moles to undergo malignant transformation into *nævo-carcinoma*, which has a high degree of malignancy, giving rise to rapidly generalised metastases.

Treatment.—It is best to leave pigment moles alone unless some definite indication for treatment is present. Free excision with grafting or plastic procedures is indicated in some disfiguring lesions, or in those liable to irritation from friction. CO₂ snow, diathermy or electrolysis may be used in the case of the smaller tumours.

HYPERKERATOTIC NÆVI.—**Synonyms.**—Linear Nævi; Ichthyosis Hystrix.

In this type of nævus the lesions are arranged in lines or bands, usually on the limbs, and often appear to follow the course of certain nerves. They are frequently unilateral, though in the ichthyosis hystrix type they are frequently symmetrical. The lesions consist of thick horny plugs, which can be pulled out from depressions in the skin, and are closely packed together; sometimes great horns protrude from the skin. On microscopical examination an irregular hyperkeratosis is found, with alternating depressions and elevations.

Treatment.—This is very unsatisfactory. Salicylic acid plasters may be used to soften and remove the horny masses, and small areas can be excised or cauterised.

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VI. OTHER MORBID CONDITIONS OF THE SKIN

ANOMALIES OF PIGMENTATION

Pigmentation may be produced by the deposit of blood pigment in the skin, by the excessive production of melanin—the normal pigment of the skin, or by the deposition of such substances as silver, seen in poisoning by that substance.

Blood pigment is found after hæmorrhages have subsided and in congestive conditions.

Increase in melanin occurs in the pigmentary nævi already referred to; in certain internal diseases, such as Addison's disease, diabetes and exophthalmic goitre; and in pregnancy; after the ingestion of arsenic; and locally, after certain inflammatory conditions of the skin, such as sunburn, erythema ab igne, lichen planus and syphilis. It also occurs in a curious condition named vitiligo, the ætiology of which is obscure.

1. LENTIGO

This is the name given to *freckles*, which occur on parts of the body exposed to the sun in certain individuals. The lesions are so well known as to require no description.

Treatment.—This is purely preventive. The application of a greasy preparation, such as lanolin, to the skin before exposure to the sun will protect the face from an erythema; sunshades and veils, especially red or brown, are also useful.

2. CHLOASMA UTERINUM

This is a peculiar yellowish-brown pigmentation which occurs chiefly about the face in women who are pregnant, or suffering from some uterine disturbance. It occurs in ill-defined patches, chiefly on the forehead and on the abdomen. It disappears after the termination of pregnancy, or when the pelvic condition is rectified.

3. VITILIGO

This condition, also known as *leucoderma* and *melanoderma*, may occur at any age or in either sex. The ætiology is quite unknown, both toxic and tropho-neuritic theories having been invoked to explain the phenomenon, but very little evidence is at present forthcoming in favour of either view. It consists of patches of a dead white colour appearing in various parts of the body; they may be quite small, or may in rare cases completely cover the body. The edge of the patches is sharp, and the surrounding skin is hyperpigmented; the texture remains normal. In the white areas pigment is entirely absent, but no other histological changes can be observed. The patient's health is in no way affected, nor can any derangement of any of the organs of the body be made out in the majority of cases.

Treatment.—The only treatment which appears to have any effect is the repeated application of ultra-violet rays, either by the Kromayer lamp or by arc-light baths.

4. ALBINISM

This is a congenital condition in which there is complete absence of pigment in the skin and other epidermal structures. The hair is white, the eyes pink from absence of pigment in the iris, and the skin fails to pigment, even when exposed to the strongest sun.

ATROPHIES OF THE SKIN

Various conditions may cause atrophy of skin, particularly local inflammations, but under this heading certain atrophic conditions are dealt with that have not been considered elsewhere.

1. SENILE ATROPHY

Generalised atrophy of the skin occurs in old age. The skin becomes thin and loses its elasticity; irregular pigmented spots, small telangiectases

and vascular cysts (de Morgan's spots) appear, especially on the face and trunk; wrinkles are very numerous; and the skin develops a yellowish colour. A generalised pruritis may occur. Senile warts are frequently found, and these may be the seat of a localised pruritus. They occasionally become transformed into squamous epitheliomata.

Treatment.—This is purely symptomatic.

2. STRIÆ ATROPHICÆ

These are bands of atrophic skin which develop in areas where the skin has been much stretched. They are seen best on the abdomen, breast and hips of women who have borne children. The lines when first formed are red in colour and about $\frac{1}{2}$ inch in diameter, but as they get older they become greyish-white. It is thought that they are produced by damage to the elastic fibres of the skin by stretching.

No treatment is required.

3. XERODERMA PIGMENTOSA

This is a rare condition of the skin which is hereditary. The ætiology is quite unknown, but there is no doubt that light-rays play a part in the production of the lesions. The affection begins in infancy, and is characterised by the appearance on the face and backs of the hands of macules of yellow and brown pigmentation. The disease is slowly progressive, and in addition to pigmentation other signs of skin degeneration appear, namely, atrophic patches, telangiectases and warty growths. Later, ulceration occurs and epitheliomatous tumours appear on the warty growths.

Treatment.—The patient should be protected from the sun's rays as much as possible. The warty growths can be removed and the ulcer treated antiseptically. Epitheliomatous growths can be checked by radium, but the cases always end fatally.

CONGENITAL CONDITIONS OF THE SKIN

Most of these, such as ichthyosis, the nævi and xeroderma pigmentosa, have been already considered. There still remains one condition which has not been alluded to, namely, epidermolysis bullosa.

EPIDERMOLYSIS BULLOSA

This is a congenital defect of the skin which renders it extremely sensitive to the slightest injury. In those affected, the slightest knock is sufficient to produce a blister. The disease is hereditary, and can often be traced to a considerable number of members of a family. The lesions usually appear first in early infancy, but occasionally they have occurred for the first time later in life. They vary much in degree. In some cases the lesions are slight and cause very little inconvenience, and no disturbance to the general health. In other cases the lesions are numerous, almost all parts of the body being affected at one time or another; teeth and nails develop badly, septic com-

plications are often severe, and these cases usually do not live to adult age.

Treatment—Nothing can be done except by prevention of sepsis and the antiseptic treatment of the lesions when once formed.

DISEASES OF THE HAIR

1. ALOPECIA

Loss of hair occurs in many diseases. It may fall out after acute illnesses, such as influenza and typhoid fever, in inflammatory conditions of the scalp, in secondary syphilis, and in dermatitis exfoliativa. It is also lost locally in scarring conditions, traumatic or inflammatory, as seen in lupus erythematosus or tertiary syphilis. A progressive loss also occurs in senile atrophy of the skin; this, however, may occur prematurely.

ALOPECIA PREMATURA.—**Ætiology.**—The disease is essentially one of the male sex, and usually begins at about the age of 20. The ætiology of the disease is not clear. Two factors appear to be present, heredity and seborrhœa of the scalp. It is quite clear that the latter condition by itself does not always produce baldness, but it appears to accelerate the loss of hair, as might be expected. Heredity seems to be important, especially in those cases where complete baldness occurs at an early age, and there is no doubt that fine hair is more liable to fall out early than is stouter hair.

Symptoms.—This gives rise to a very characteristic type of hair loss which is familiar to every one. The hair gradually gets thin on both temples and on the vertex, and by slow progression these thinned areas eventually meet, leaving the top of the head entirely bald or only covered by a fine down, while the sides and back of the scalp are covered normally. The progress varies very considerably in different individuals, some becoming completely bald in a year or two, while others still have a good crop of hair at 50.

Treatment.—This has mainly to be directed to curing the seborrhœa, and the methods for doing this have been dealt with under that heading. Apart from this, avoidance of tightly fitting hats, and gentle massage with the fingers are the most appropriate remedies. Certain drugs such as pilocarpine, have been thought to have a stimulating effect on hair growths, and rubefacients, such as cantharides, are also much employed. The general health should receive attention.

ALOPECIA AREATA.—In this condition the hair falls out in patches, leaving smooth, shiny, bald areas. There is a general tendency for the hair to grow again.

Ætiology.—The malady affects both sexes and generally occurs in early adult life. It is probable that the disease is an inflammatory condition, but the nature of the irritant is unknown. A somewhat similar loss of hair can be produced by the administration of thallium salts, which lends support to the toxic theory. It was at one time thought to be due to an external parasite, but there is no evidence to support this view. It has also been thought to be of nervous origin, as damage to nerves has produced bald patches over the areas supplied.

Symptoms.—The disease may start suddenly or slowly. In some cases a large circular patch of baldness may occur in a single night, and in these cases the skin may be tender and reddened. Generally, however, a small bald patch appears, which spreads slowly, and other patches may subsequently arise, causing considerable loss of hair and a curious patchy condition of the scalp. Not only the scalp hairs but those of the beard, the eyebrows, axillæ and pubes may be affected, and in severe cases all the hairs of the body, including the eyelashes, may fall out. In one type a band of hair may be lost extending from ear to ear around the margin of the scalp, either in front or behind the head or even in a complete circle.

In the patchy form, the individual patches are characteristic. The centre is usually completely bald and shiny, though new downy hairs may be seen. Around this a row of stumps may be observed. These are club-shaped, like a note of exclamation, being very thin as they enter the scalp and thicker above. When pulled out, a shrunk hair bulb comes out and the hair does not break as in ringworm. The zone outside the zone of stumps looks normal, but if the hairs are pulled upon many loose hairs may be detached.

Course.—This varies in different cases. Usually new hair grows fairly rapidly, and the patches cease to spread. The new hair is usually white when it first appears, but pigments later. In some cases, however, the patches progress as new hairs grow and this may continue for many months. In the band-form hair growth is usually much slower. In the generalised cases the prognosis is not so good, a large proportion losing the hair permanently.

Diagnosis.—This has to be made from ringworm and is generally easy. In ringworm the patch is scaly and covered by stumps, which break easily and have an irregular fractured end. Under the microscope the fungus can be seen.

Impetigo contagiosa of the scalp sometimes gives rise to bald patches. They are numerous, small and usually red in colour, and no stumps are seen.

Treatment.—The general health must be looked to and all possible sources of irritation removed. The teeth must be attended to, tonsillar sepsis treated, and errors of vision corrected. General tonic treatment should be prescribed, and rest from overwork and worry ordered. Thyroid extract has been recommended, but in the writer's experience has sometimes made the condition worse.

Local applications which cause hyperæmia are of most value. Painting the patches with pure phenol, iodine or blistering fluid, or rubbing in oil of turpentine is useful. Various antiseptic lotions, such as perchloride of mercury and resorcinol, have been used with success. High frequency current and ultra-violet rays have given good results when other means have failed. If seborrhœic dermatitis is present it should be treated.

CICATRICAL ALOPECIA.—In this condition, also known as *pseudo-pelade* or *folliculitis decalvans*, progressive loss of hair takes place, and the scalp shows signs of atrophy or scarring. There is a progressive patchy loss of hair occurring over considerable areas of the scalp, and on examination inflammatory lesions are often present round the hair follicles. The denuded areas show obvious scarring, and hair does not regrow on the patches. The disease occurs chiefly in young adults, and though it does not usually lead to complete baldness, very disfiguring patches remain.

Treatment.—This consists in the application of antiseptic ointments and lotions, perchloride of mercury, resorcinol and sulphur being the most useful, but no treatment is very efficacious.

2. HYPERTRICHOSIS

This is the term applied to an excessive growth of hair. It is usually confined to those cases in which a growth of stout hairs occurs in sites usually covered with lanugo hairs, such as the face in women. This may sometimes be very excessive, and the “bearded woman” and the “dog-faced man” are extreme examples, though the latter are often cases of hairy moles. The only conditions that the medical practitioner is likely to have to deal with are those in which stout and dark hairs occur on the chin and upper lip in women. The treatment consists in removal of the hairs by electrolysis, but considerable judgment is often required to decide whether a case is suitable for treatment. Electrolysis consists in passing a current of about 1 milliampere for a quarter to half a minute into the hair bulb by means of a fine needle attached to the negative pole of a galvanic battery. The hair then loosens and can be removed. It is important not to remove hairs too close to one another at the same sitting, or troublesome scarring will supervene.

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VII. TROPICAL SKIN DISEASES

In the tropics many skin diseases occur which are met with in Europe; in addition, there are certain conditions peculiar to hot climates, and it is with these that the present section deals. At the same time it must be realised that skin diseases may be modified by racial immunity, social custom and skin pigmentation. Leucoderma has, for instance, a peculiar and sinister significance, not only on account of the great disfigurement it produces in dark-skinned people, but also because of its superficial similarity to the depigmented patches of nerve leprosy. Again, native custom may modify such conditions as keloid, which may assume a magnitude never experienced in Europe, owing to the fact that primitive people often purposely irritate wounds to produce tribal marks of a keloid nature.

CRAW-CRAW

A West African native name (Kra-kra) applied to any itchy, papular or pustular eruption of the skin. It originates as a papular dermatitis.

Ætiology.—O'Neill found filarial embryos in an eruption resembling scabies, but they were probably *Microfilaria streptocerca*, which Scott Macfie has since commonly found in the skin of West African negroes.

Symptoms.—The papules are hard and horny, occur chiefly in the limbs, and are very itchy: scratching and secondary infection lead to a pustular dermatitis with enlargement of adjacent lymph glands.

Diagnosis.—The condition must not be confused with scabies or coolie itch: no acari are obtained and no burrows seen.

Treatment.—Pustules are opened, ulcers scraped and crusts removed, then disinfected with 1 in 1000 sublimate solution and subsequently dressed with boric acid ointment. Carbolic lotion improves some cases.

PRICKLY HEAT

A form of miliaria associated with excessive sweating in hot climates.

Ætiology.—The condition quickly disappears in cold weather, and is possibly purely a mechanical process due to blocking of the sweat glands with sodden, inadequately cornified cells of the stratum corneum. Bacteria and yeast-like fungi have been incriminated, but these are probably secondary invaders.

Symptoms.—The red eruption consists of small, watery vesicles and inflamed, red papules which feel like grains of sand and may involve the trunk, limbs, forehead or almost any part of the body. The pricking sensations and great itching may be sufficient to prevent sleep.

Treatment.—The underclothes should be frequently changed (twice daily), and antiseptic soaps used in bathing. After a warm bath the application of corrosive sublimate solution (1 in 1000) containing eau-de-cologne is helpful. McLeod recommends the following lotion: R acid. salicyl., grs. xxx., hyd. perchlor. grs. ii., sp. vini rect. 3 ii., aq. dest. ad 3 vi. After this has dried a dusting powder such as zinc oxide, boracic acid and starch in equal parts, or boracic acid and menthol should be employed.

VELDT SORE

This is a chronic, septic, ulcerated sore met with in the tropics and sub-tropics, generally involving the exposed hairy parts of the body.

Ætiology.—The disease has a widespread geographical distribution in hot, dry, sandy or desert country, being known as barcoo rot in Northern Australia, and veldt sore in South Africa: during the war it became known as desert sore, and affected troops in the Near East, especially in Sinai and Mesopotamia. Vitamin deficiency may be a predisposing factor, for there is often a history of living on tinned foods and an absence of fresh fruit and vegetables. Organisms isolated from the lesions include streptococci and diphtheria bacilli; staphylococci are also present, but these are probably surface contaminants. Horse manure may constitute a source of origin for the infecting organism.

Symptoms.—The lesion commences as a painful vesicle, containing yellowish fluid, on exposed parts of the skin, especially the dorsum of the hand, forearm, elbows, knees and occasionally the face. Rupture follows, and the condition ultimately results in a punched-out, circular or oval ulcer with a tough, dirty grey base and thick, bluish indurated edges; it may take many months or even years to heal, leaving a thin scar. Typical diphtheritic paralysis of the limbs and palate was noted in some of the Sinai cases associated with Klebs-Loeffler bacillus (Craig) in the sores.

Diagnosis.—The condition must be distinguished from ulcus tropicum and Leishmanial sores; cultures may reveal the causative organism, which, in some cases at least, is the diphtheria bacillus.

Treatment.—**PROPHYLACTIC.**—Adequate dietary, protection of exposed parts and antiseptic treatment of abrasions should help. *Curative.*—The only specific treatment is anti-diphtheric serum (4000 units); dramatic cure may follow in certain cases. The lesions themselves must have sterile protective dressings: ammoniated or nitrate of mercury ointment often does good. Iron and arsenic tonics and a high vitamin diet should be given, and autogenous streptococcal vaccines are worth a trial in intractable cases.

PEMPHIGUS CONTAGIOSUS

A contagious skin eruption, known also as *Pyosis mansonii*, due to coccal infection, characterised by inflammatory vesicles and bullæ which ulcerate and scab.

Ætiology.—The disease is common in the humid tropics such as Ceylon and Malaya, also in parts of Africa. European children are specially affected. Culture generally shows *Staphylococcus aureus* or *albus*; streptococci may be isolated.

Symptoms.—The condition begins as a minute red speck which is transformed first into a vesicle, then a bulla and later a pemphigus-like blister. The fluid content, which is at first clear, later becomes purulent and, after bursting, the lesions generally dry up, desquamate and heal, sometimes leaving pinkish, slightly glazed spots on the skin. The eruption is mainly confined to the axilla and crutch, but in children may be spread more widely by auto-infection, the whole body, except the face, being sometimes involved (Smith). Constitutional disturbances are minimal.

Diagnosis.—The condition is allied to impetigo contagiosa and may need to be distinguished from early small-pox, chicken-pox and ringworm.

Treatment.—Cleanliness is all-important and auto-infection must be avoided. The parts should be washed with perchloride of mercury (1 to 1000) followed by a dusting powder of zinc oxide, boracic acid and starch (equal parts). Ammoniated mercury ointment is often useful. Sulphapyridine preparations (M. & B. 693) in full dosage is worthy of trial.

TROPICAL ULCER

Ulcus tropicum or tropical sloughing phagedæna is a gangrenous ulceration of the skin and subcutaneous tissues of unknown ætiology, resulting in the formation of sloughing ulcers of great chronicity.

Ætiology.—In contradistinction to veldt sore, this disease is met with in damp, steamy jungle in the tropics. The lower limbs are generally involved, and a history of preceding trauma is the rule. It is common in debilitated and diseased populations, may affect people of any age and either sex, and has occasionally assumed epidemic proportions, as amongst coolies in the tea plantations of Assam. Some regard it as a dietetic deficiency. Fusiform bacilli and a spirochæte named by Prowazek, *Treponema schaudinni*, are commonly present in the ulcer: various cocci, fungi and diphtheroids have also been found. The condition is directly transmissible by inoculation of ulcer material from man to man (Smith).

Symptoms.—Phagedænic ulcers generally affect the dorsum of the foot and the front of the legs, and more rarely the hands and forearms. The disease originates as a serosanguinous bleb which soon ruptures, leaving a dirty grey slough. This process rapidly extends, forming a foul sloughing ulcer, which may attain several inches in diameter, giving rise to pain, and sometimes fever, and occasionally involving deeper structures like muscles, tendons, blood vessels, nerves, periosteum, and even joints. Three stages are recognisable: (1) spreading sloughing ulceration; (2) a stage of tissue equilibrium when destruction and growth of granulation tissue are equalised; (3) healing. Generally these ulcers persist for months, a factor delaying healing being inadequate epithelial proliferation, even after a healthy granulation tissue base has formed. Many cases show a decrease in blood calcium.

Diagnosis.—In the humid tropics diagnosis is generally easy, though varicose ulcers, yaws, syphilitic and blastomycotic ulcers and oriental sore may need differentiation.

Treatment.—Protection of the legs with puttees is very advisable. Curative treatment varies with the stage of the ulcer. Rest, a nutritious diet, calcium, cod-liver oil and general vitamin reinforcement by multivite pellets (B.D.H.) are advised. In the rapidly ulcerating stage sloughs should be removed and ensol dressings or lotions of carbolic or permanganate applied. Good results have been reported following curettage of the ulcers and daily dressing with B.I.P.P. Cod liver oil dressings have also been favourably reported on, and local treatment with tar spread on lint and changed every three days has been found suitable for mass treatment of natives. The application of iodoform powder may be followed by firmly bandaging with elastoplast which is left undisturbed for a week; septic dermatitis sometimes complicates this treatment. Probably the most effective procedure in the chronic stage is complete excision, followed by skin grafting. Rarely in the acute stage of fulminating cases, with rapid sloughing and gangrene, amputation is necessary to save life; even more rarely has it to be done in chronic cases.

TINEA

Ringworm infections abound in the tropics, some being confined to special regions, while others are much the same as in temperate climates. The chief ones are: (1) *Tinea cruris* or dhobie's itch; (2) Hong-Kong foot or ringworm of the foot; (3) *Tinea unguium*; (4) *Tinea imbricata*. The first two are due to the *Trichophyton*, *Epidermophyton inguinale*: they are not peculiar to warm climates and are described elsewhere (p. 1436).

TINEA UNGUIUM.—A mycotic infection of the nails affecting Europeans from the Far East: it may last for years and be associated with ringworm elsewhere. The nail-bed is involved, leading to brittleness, ridging and opaqueness of the nail. Diagnosis is made by demonstrating *Epidermophyton inguinale* in scrapings mounted in liquor potassæ. In severe cases the nails may have to be removed before cure is effected.

TINEA IMBRICATA (Tokelau).—A form of ring-worm mainly indigenous in the Eastern Archipelago and South Pacific, and characterised by non-inflammatory raised brown spots, giving rise to flaky tissue-paper scales which are free centrally, but attached at their peripheral bases, producing

a rosette-like appearance. These circles are about $\frac{1}{4}$ inch in diameter and as adjacent ones form they cause a characteristic festooned appearance. The fungus, *Endodermophyton concentricum*, is readily demonstrable in the scales: it affects the face, trunk and limbs, but the palms, soles, scalp, axillæ and crutch generally escape.

PITYRIASIS VERSICOLOR or *Tinea flava* is common in the tropics, producing pale, yellowish-brown, scurfy patches on the pigmented negroid skin, especially on the face, neck, arms and chest. Castellani holds that the yellow patches met with in his Ceylon cases differed from the brownish patches long recognised as being caused by *Microsporon furfur* in the European disease, and has named the tropical variety *Tinea flava* and the causal fungus *Malassezia tropica*; the black variety, which is caused by *Cladosporium mansonii*, Castellani calls *Tinea nigra*.

PINTA

This is a group of dermatomycoses associated with coloured patches of pigmentation in the skin.

Ætiology.—The disease, also called caraate or mal de los pintos, is found in tropical America, is contagious and attacks either sex at any age. A variety of fungi are implicated, including *Penicillium*, *Aspergillus* and *Monilia*.

Symptoms.—Patches of pigmentation are first noted on the back of the hands or face, from which they spread elsewhere: they are somewhat rough, dry and raised, and vary in colour with the fungus, red, violet, white and black types all being encountered. The skin may be offensive and itchiness marked. When the scalp is involved the hair may become white.

Diagnosis.—Microscopic examination of material scraped from the pigmented areas reveals the fungi. The patches are not anæsthetic like leprosy, while leucoderma, which the white variety may resemble, fails to show fungi.

Treatment.—As for ordinary ringworm.

PIEDRA

Trichosporosis or Piedra is a disease common in Colombia and British Guinea in which hard, gritty nodosities form around the hair of the scalp; it is caused by the *Trichosporon giganteum* and may be confused with ordinary Trichomycosis nodosa.

CREEPING ERUPTION

Synonyms.—Larva migrans, Myiasis linearis, Hautmaulwurf.

Definition.—A peculiar linear, slightly raised red eruption, gradually creeping forward in a sinuous or straight line, the posterior end fading away.

Ætiology.—The condition may be produced by *Gastrophilus* or other fly larvæ wandering under the skin, but more commonly it is due to nematode larvæ of animals which have accidentally invaded man. The following species have been implicated: *Ancylostoma braziliense*, *A. caninum*, *Uncinaria stenocephala* and *Gnathostoma hispidum*.

Symptoms.—The symptoms vary in different individuals and include smarting pain and intense itching along the raised line which first shows red spots, and later hard round red papules 2 to 5 mm. in diameter ; pustulation may occur. Unless treated the condition persists for a long time.

Treatment.—Freezing the anterior end of the line where the larva is located, with an ethyl chloride spray for 2 minutes, is suitable for the type due to canine ancylostomes. Multiple lesions may be treated with collodion ethyl acetate or salicylic acid, and blisters and pustules with mercurochrome solution. An injection of pure carbolic an eighth of an inch in front of the spreading spot may kill the larva, or if the condition is due to the larva of *Gastrophilus*, this may be cut down on and removed. Recently oleum chenopodii applied locally either pure or diluted with three parts of castor oil has been favourably reported on.

CERCARIAL DERMATITIS

Definition.—An inflammatory condition of the skin due to the passage through it of different species of cercariæ.

Ætiology.—In 1928 Cort in Michigan described a form of dermatitis due to the passage of *Cercaria elva* through the skin and Taylor and Baylis have also found this in England.

Symptoms.—The skin at the site of entry of the cercariæ becomes intensely itchy and smart, then red spots or urticarial wheals appear, these being followed by papules which sometimes go on to pustulation.

Treatment.—No specific treatment is known. The part should be kept clean and dusted with boracic and zinc powder. Calamine lotion combined with lead acetate may reduce the itching.

ULCERATING GRANULOMA

Synonyms.—Granuloma venereum ; Granuloma inguinale ; Granuloma inguinale tropicum ; Ulcerating Granuloma of the Pudenda ; Serpiginous Ulceration of the Genitals.

Definition.—A very chronic ulcerating condition of uncertain ætiology occurring in the tropics, involving the genitals, perineum and groins.

Ætiology.—The disease occurs in the West Indies, Guiana, Brazil, Porto Rico, parts of India and Africa, the Pacific Islands and Northern Australia. Both sexes are affected, but not before puberty, and all races are susceptible. Spirochætes have been reported, and Donovan and many other observers have found a short, oval bacillus specially located within the mononuclear cells ; it is a non-motile, capsulated bacterium of the rhinoscleroma group, but though found with frequency in the lesions there is still doubt as to its real ætiological significance. The disease itself is probably contracted during coitus.

Pathology.—The condition resembles an infective granuloma, and microscopic section of the nodules situated at the edge of the sore shows infiltration with plasma and round cells containing poorly staining nuclei in which phagocytosed bacilli may occur in clumps. The granulomatous tissue is very vascular, while in the older areas fibrosis and scarring are marked. Spread is by direct continuity and the lymphatic system is never involved.

Symptoms.—The disease begins on the genitals as a flat papule which desquamates, leaving a red granulation-tissue surface which bleeds easily: this superficial ulceration extends serpigginously producing offensive pus. As the process advances the older areas cicatrise, but this scar tissue readily breaks down again. The disease is auto-inoculable so that adjacent parts such as the scrotum and thighs, or the surfaces of the labia become infected. Ultimately the whole of the penis, scrotum and groins in the male, and the clitoris, vulva, labia, vagina, perineal and perianal region in women become involved, and, if unchecked, the urethra and rectum as well. Though skin ulceration extends slowly over a period of many years, the process accelerates once the mucuous membranes are involved, and here there is little tendency to heal. Until the terminal phase the general health remains good and the local lesions give rise to a minimum of pain and discomfort.

Complications.—These include recto-vaginal fistula, urethral stricture, septic cystitis and pyelitis.

Diagnosis.—Ulcerations due to syphilis, tubercle or lupus vulgaris may be confused, and where the glans penis is involved with fungating granuloma, epithelioma may be suspected.

Prognosis.—This has greatly improved by modern treatment; formerly the condition was hopeless, lasting for life.

Treatment.—Illicit intercourse, especially with native women, should be avoided. Surgical excision of the early lesions is curative, especially as the ulceration does not extend deeply, but in more advanced cases is not feasible. The modern treatment consists of intravenous injections of tartar emetic which is a specific. This drug is given as in schistosomiasis (p. 311) only a longer course of injections and a greater total dosage, *i.e.* 50 to 60 grains, is generally necessary; in extreme instances as much as 150 grains have been given. Certain pentavalent preparations of antimony, such as stibosan and stibenyl, are reported to be more efficacious, a total dosage of 3–4 g. being necessary to obtain complete cure. Protein shock produced by the intravenous injection of T.A.B. vaccine starting with 50,000,000 per c.c. and gradually increasing to 300,000,000 per c.c. may be employed in addition to antimony: sometimes it stimulates healing in a remarkable fashion.

In certain chronic cases unaffected by antimony, gauze soaked in an aqueous solution of zinc oxide (40 per cent.) is an effective dressing, especially if non-hæmolytic anerobic streptococci be present in the ulcer. Sulphapyridine therapy (M. & B. 693) is still under trial.

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SECTION XX

DISEASES OF THE NERVOUS SYSTEM

AFFECTIONS OF THE CRANIAL NERVES

INTRODUCTION

AFFECTIONS of the cranial nerves may be due to their involvement: (1) by purely local disease at some point between their central origin and their peripheral distribution; (2) by some general toxic or infective disease of the whole nervous system; as, for example, by alcoholic or diphtheritic poisoning; (3) by multiple local lesions of the central nervous system, as, for example, by syphilis or disseminated sclerosis; and (4) in some systemic neurone degeneration, as in progressive muscular atrophy.

In the case of local lesions within the brain stem, the presence of one or more cranial nerve palsies may have considerable localising importance in diagnosis. In their peripheral course, both inside and outside the skull, these nerves may be involved by a great variety of inflammatory processes, by hæmorrhage, or by compressing tumours.

THE OLFACTORY NERVE AND TRACT

The sense of smell includes the appreciation of all flavours, and in its absence taste is reduced to the appreciation of bitter, sweet, salt and sour. The view that only readily oxidisable substances are appreciated by the sense of smell, formerly expressed in these pages, cannot be sustained. Benzene, for example, has a powerful smell yet is chemically very stable. The same is true of chloroform and carbon tetrachloride, both substances with a strong odour. The gas phosgene is a final example. Most conditions of anosmia are due to affections of the nasal mucosa, or occur in such general diseases as syphilis or influenza. More important is unilateral anosmia, which indicates the involvement of one olfactory tract by the pressure of a tumour. Bilateral anosmia is a not uncommon sequel of head injuries in which the olfactory filaments are torn as they traverse the cribriform plate.

OPTIC NERVE

The optic nerve, from its origin at the back of the eyeball to its termination in the optic chiasma, is liable to injury from the pressure of tumours within the skull, or at the back of the orbit, and from inflammatory conditions of

the bone and periosteum as it passes through the optic foramen. It may be affected by a primary thrombosis of vessels, or from thrombosis of the ophthalmic artery in the condition known as carotid hemiplegia. These conditions result in blindness of one eye, which may be partial or complete, and with a varying visual field, according to the degree of the lesion and the manner in which the pressure falls upon the optic nerve. On account of the cutting off of the light-reflex path, the pupil will be moderately dilated and insensitive to light. Notwithstanding the fact that the nerve cells which control the nutrition of the optic nerve fibres are situated in the retina, lesions of the optic nerve of any long duration produce atrophy of the optic disk.

RETROBULBAR NEURITIS

Inflammatory and other local lesions in the substance of the optic nerve between the globe and the chiasma are of very common occurrence. According to their severity they give rise to partial or complete blindness, which often recovers wholly or in part. The central part of the optic nerve is the seat of election for these lesions and, therefore, the visual defect appears commonly in the form of a central scotoma. According to the degree of the visual defect, the pupil tends to be dilated, and to react poorly to light, and dilate badly to shade. These conditions of retrobulbar neuritis are very often followed by optic atrophy of varying degree. When the inflammatory lesion occurs far forwards and impinges upon the optic papilla, papilloedema occurs, and, as this region is sensitive, whereas the optic nerve is not, there is usually pain on movement of the eyeball. The prognosis in this condition varies with the causes, which are as follows :

1. *Syphilis*.—The lesion is a diffuse gumma of the nerve, and sometimes there is thrombosis. It is commonly unilateral, and often recovers, if treated early and energetically, but in severe untreated cases, and when thrombosis occurs, it not uncommonly ends in complete blindness. It is the common cause of complete blindness confined to one eye in tabes, and in other syphilitic conditions.

2. *Disseminated sclerosis*.—Plaques in the optic nerve tracts and chiasma are very common in this disease. When they first form they are swollen oedematous pink patches and cause pressure, and on their subsequent shrinking, this pressure is removed, and the nerve fibres recover to a great extent, and never become completely destroyed. This condition is the cause of the very common transient amblyopia or blindness, which may come on very suddenly. Though the plaques often occur in the tracts and chiasma, yet the visual defect always indicates a lesion of the optic nerve, and therefore it is possible that it is determined by the swelling of the optic nerve as it passes through the optic foramen, and is compressed against the bony canal. It may be unilateral or bilateral, and may recur several times. It always causes some degree of optic atrophy ; but is never followed by complete blindness.

3. *Local septic conditions*.—Before the general recognition of disseminated sclerosis as the common cause of retrobulbar neuritis in young adults, the chief rôle in its production was attributed to local infection, or suppuration in the sphenoidal or other accessory sinuses in the neighbourhood. The natural restoration of vision that marks the subsidence of the retrobulbar neuritis was accordingly often ascribed to the operative procedures designed

to deal with the local infection. It is probable that such local infections are but a rare and exceptional cause of retrobulbar neuritis.

4. *Diabetes*.—In this malady a form of retrobulbar neuritis occurs. It commences with a central scotoma for colour, and the failure of vision may progress to blindness, with optic atrophy.

5. *Other causes*.—Tobacco, if over indulged in, may cause a curable form of retrobulbar neuritis, commonly called "tobacco amblyopia," of which the chief sign is central scotoma, and the symptom mistiness of central vision. It recovers rapidly on the removal of the cause. Alcoholic indulgence, and especially taking of wood spirit and many other poisons such as arsenic, lead, bismuth and quinine, may cause retrobulbar neuritis. A large proportion of cases of retrobulbar neuritis are without discoverable cause; there is evidence that some of these are dependent upon local oedematous reactions within the optic nerve of an allergic nature.

The expansion of the optic nerve within the globe of the eye is visible on ophthalmoscopic examination, and the pathological changes therein occurring afford important indications which cover the whole realm of medicine. Among these changes the most important in connection with diseases of the nervous system are: syphilitic choroiditis, as an indication of the presence of syphilis, papilloedema, which is fully described in the section on intracranial tumours, and optic atrophy.

OPTIC ATROPHY

Optic atrophy is recognised, on ophthalmoscopic examination, by a peculiar opaque whiteness and flatness of the disk, with a very high contrast at the edge of the disk between disk and surrounding retina, both as regards colour and limitation. The lamina cribrosa—the sieve-like cross-latticing of the strands of the sclerotic through which the bundles of optic nerve fibres pass—becomes visible as a stippling of the temporal region of the disk. The vessels of the retina become atrophied, and are seen to be unduly small. In many atrophies the edge of the disk is sharply cut; but when atrophy follows papilloedema the edge is apt to be fluffy, like that of torn cotton-wool. Optic atrophy may be of three kinds—(1) Primary optic atrophy results from an original devitalisation and death of the nerve cells of the retina with their processes, which constitute the fibres of the optic nerve. This is a primary neuronc degeneration, analogous to that of the anterior-horn cells in progressive muscular atrophy. (2) Secondary or retrograde optic atrophy results from lesions of the optic chiasma and optic nerve, and is the constant result of long-continued pressure upon these structures. (3) Consecutive optic atrophy follows the more severe grades of papilloedema and papillitis, in proportion as these are of long standing, and proportional to the amount of exudate, and is due to strangling of the optic nerve fibres by the oedema in the first place, and by the cicatrization subsequently. Severe degrees of papilloedema may, if pressure be relieved, recover perfectly without atrophy or impairment of sight. It may be that there is another factor in the atrophy following papilloedema, and that is the long-continued pressure of a distended infundibulum upon the optic chiasma.

Ætiology.—1. It is of frequent occurrence in familial, hereditary or

congenitally installed diseases in which primary degeneration of neurones occurs, as in cerebral diplegia, amaurotic family idiocy, and hereditary cerebellar atrophy where it is characteristic of Marie's type, and sometimes occurs in Friedreich's type. It is the chief feature of the familial optic atrophies, of which Leber's type, appearing about the twentieth year, is one. It occurred in several members of a family with peroneal atrophy under our observation.

2. It is one of the common manifestations of syphilis of the nervous system, and may occur alone, but much more usually as part of the syndrome of tabes and general paralysis. It is not rarely met with in congenital syphilis. It is commonly associated with local lesions of the chiasmal region, pituitary neoplasms being the most often met with. The optic atrophy of disseminated sclerosis is not a primary optic atrophy, but is secondary to retrobulbar neuritis.

3. It may follow the exhibition of certain drugs, and noticeably the injection of the earlier used arsenic preparations, such as atoxyl, soamin and orsudan. In rare cases it has followed the use of quinine.

4. Optic atrophy also occurs in connection with diabetes, malaria and arterial disease. It is common as a primary condition in later life. Its occurrence in glaucoma from increase of the ocular pressure requires no explanation.

Prognosis.—This is uniformly bad in primary atrophy. When once the degenerative process is installed, the atrophy proceeds to complete blindness, sometimes slowly, sometimes quickly, and seems entirely uninfluenced by any form of treatment.

Secondary optic atrophy is frequently arrested, with the recovery or removal of the cause; but some defect of vision usually remains. Consecutive atrophy from neuritis may be of any degree of severity, from the slightest, which allows of $\frac{5}{8}$ ths vision, to the most complete with utter blindness and loss of light reflex.

OCULO-MOTOR NERVES

The third nerve supplies the two internal muscles of the eye, and all the external muscles of the eyeball, except the superior oblique, which is supplied by the fourth nerve, and the external rectus, which is supplied by the sixth nerve. Complete paralysis of this nerve produces a dilated and inactive pupil with complete ptosis, a downward and outward strabismus and complete loss of upward, downward and inward movements. There is often no diplopia complained of by the patient, because of dropping of the lid. When diplopia is present it is a crossed diplopia, because the strabismus is divergent. There is secondary deviation of the sound eye, and false projection in the visual field.

The fourth nerve supplies the superior oblique muscle. Paralysis produces no obvious strabismus, but in looking outwards or downwards there is a wheel movement of the globe which can be detected by observing the conjunctival vessels when the eye moves. The diplopia is most discomforting, and occurs in every position of the eyes, except on looking up. The diplopia is uncrossed, and the false image is lower than, and with its top tilted toward, the true image.

The sixth nerve supplies the external rectus muscle. Paralysis produces a convergent squint and an uncrossed diplopia. In the case of the skeletal musculature, we are accustomed to distinguish between upper and lower motor neurone paralyses; the essential difference being that in the former case we are dealing with loss of co-ordinated movements, in the latter with paralysis of individual muscles. Disorders of ocular movements may usefully be considered on the same basis of classification. Thus we may contrast the loss of conjugate deviation of the eyes to the right—an upper motor lesion—with paralysis of the left internal rectus the result of a lesion of the lower motor neurone. In the former case, the internal rectus muscle acts normally on convergence, but does not act on attempted conjugate deviation to the right. In the latter case—a lesion of the peripheral nerve supply—the muscle is paralysed in all movements of the eye.

We may thus classify paralyses of ocular movement under the following headings: (1) paralyses of ocular movements, conjugate and supranuclear; and (2) paralyses of ocular muscles, nuclear, stem and root palsies.

1. PARALYSES OF OCULAR MOVEMENTS.—Conjugate palsies consist in the loss or impairment of co-ordinated ocular movements in one or more directions. Since both eyes are equally and simultaneously involved, there is neither squint nor diplopia. The commonest form is that in which there is loss of conjugate movement to one or other side. The lesion in such a case is so situated as to interrupt the association path between the sixth and third nerve nuclei, probably where this path lies in the dorsal longitudinal bundle. If a lesion in this situation be a coarse one (*i.e.* not a simple nerve cell degeneration), there will probably be an associated nuclear or stem palsy, in which case there may be diplopia and squint. Possibly a lesion lying just oral and ventral to the sixth nerve nucleus is capable of causing a pure paralysis of conjugate deviation to the side.

Lesions involving the corpora quadrigemina cause loss of vertical (upward) movement of the eyes, sometimes with dilatation and immobility of the pupils. This combination of signs may be seen in pineal tumour.

Supranuclear ocular palsies differ from conjugate palsies in that with the latter a given movement is totally lost, while in the former, whether or not the movement occurs will depend upon the particular stimulus in action. Thus, in a supranuclear palsy the patient may be unable to deviate his eyes to order, but deviation may be elicitable by labyrinthine stimulation. Or again, the gaze may be unable to follow a moving object, but if the object be still and the head slowly rotated passively, the gaze may remain fixed on the object, and the eyes thus come to take up a position of deviation.

2. PARALYSES OF OCULAR MUSCLES.—The lesion in this case may be in the brain stem (nuclear or stem palsies), or in the peripheral course of the nerve (root palsies). Further, the lesion of the peripheral part of the nerve may involve the nerve between its point of origin from the brain and the point of entry into the orbit; that is, in its intracranial course; or in the region of the sphenoidal fissure; or, finally, within the orbit.

Within the orbit lesions of any of these nerves or of their branches may occur from perforating wounds, blows upon the eyeball or from local syphilitic lesions of a gummatous nature. Injury to the lenticular ganglion, with resulting complete internal ophthalmoplegia, not uncommonly results from a blow on the eyeball. A lesion, confined to the nerve to the inferior oblique,

occurs in rare cases from syphilis, and produces a peculiar attitude of the head, for, since the resulting diplopia occurs only above the horizontal level of the eyes, the patient constantly throws back his head, and looks down his nose to avoid the diplopia.

Lesions at the back of the orbit may involve one or more of the oculomotor nerves, and since the first division of the fifth nerve passes through the sphenoidal fissure with these nerves, and the second division of the fifth nerve is entering the infra-orbital canal at the apex of the orbit, both these nerves are commonly involved in the same lesion. New-growths, including those of the bone, periosteal inflammation and subperiosteal hæmorrhages arising from fracture of the skull are the common lesions. A not infrequent clinical picture may be described. It has been attributed to a periostitis of the orbital bones in the region of the sphenoidal fissure. There is, however, no pathological confirmation of this view, which remains speculative. It has been thought, too, that the condition is closely allied to the common facial palsy from exposure to cold, but here also proof is lacking. It may occur at any age from puberty onwards. The serum reactions for syphilis have always shown that this infection is absent. The condition may arise from exposure to cold or from septic conditions of the nose and its accessory sinuses, and sometimes without obvious cause. The malady commences with pain in the orbit, which is often severe and long lasting. Soon after some proptosis is evident, and there is tenderness on pressing the globe backwards. This is soon followed by signs of involvement of the nerves which pass through the sphenoidal fissure. The sixth nerve is the first and sometimes the only nerve involved, but usually the paralysis of this nerve is followed by that of the fourth, the first division of the fifth, the third, and the second division of the fifth nerve in that order. The final result usually is that of a total ophthalmoplegia with anæsthesia of the upper two divisions of the fifth nerve, unilateral proptosis and tenderness of the eyeball, and often excruciating and lasting pain. When the sixth nerve is involved alone, and there is no spread to the divisions of the fifth nerve, there may be little pain, and the proptosis and tenderness may be little marked. Under treatment with mercury by inunction, salicylates in full doses, warm applications to the eye and counter irritation, the condition commonly recovers in a few weeks in the milder cases, to a few months in the more stubborn ones. It is in their intracranial course that the oculomotor nerves are most frequently involved by disease-processes, or by injury; thus, by acute or chronic meningitis, by toxic or infective neuritis, by compression from tumour, aneurysm, hæmorrhage or fracture of the skull.

Fractures of the skull, may involve the orbit, or the middle fossa of the skull, thus producing ocular palsies. Sometimes a blow on the skull without fracture is followed by an ocular palsy. It is possible that in this instance small hæmorrhages in the brain-stem involving the nuclei may be responsible.

In subarachnoid hæmorrhage, the effused blood in the region of the interpeduncular space may compress the cranial nerves, thus producing ocular palsies. These may also occur in the course of an acute lepto-meningitis. In association with middle ear disease, there is occasionally a localised meningitis at the tip of the petrous bone, which gives rise to a unilateral sixth nerve palsy and to pain on the side of the head and face from fifth nerve involvement. This is the condition known as *Gradenigo's syndrome*. It is

usually seen in children ; and a mastoid operation may be necessary before it clears up.

Formerly, one of the commonest causes of ocular palsies was a gummatous infiltration of the nerves of the base of the brain in neurosyphilis. In some cases of *tabes dorsalis*, however, it is probable that the lesion underlying the defects of ocular movement (squint, diplopia, ptosis) may be due to primary degeneration of nerve cells in the nuclei.

In some cases of alcoholic and diphtheritic multiple neuritis, paralysis of one or more ocular muscles may occur, and also an isolated sixth nerve palsy has been known to follow the spinal administration of stovaine.

In some elderly subjects with atheroma and high blood pressure a sixth or third nerve palsy may develop, and recover after some two or three months. It is probable that in such not infrequent cases one or other of these nerves has been compressed by a tortuous and rigid artery. Thus, during its passage forwards the sixth nerve lies in close contact with the middle cerebellar, the basilar and the internal carotid arteries ; the fourth nerve crosses the posterior cerebral artery ; and the third nerve lies between the last-named vessel and the superior cerebellar artery.

Stem palsies, in which the lesion is in the brain-stem, are usually recognisable from the presence of associated signs due to involvement of other structures. These may be the long projection paths, sensory or motor, association nuclei and reflex centres, or other cranial nerve nuclei. Thus, a lesion in the crus will produce a homolateral third nerve palsy, with a crossed hemiplegia (Weber's syndrome). A lesion in the region of the red nucleus will cause a homolateral third nerve palsy, with tremor of the crossed limbs (Benedikt's syndrome). A pontine lesion will produce associated sixth and seventh nerve lesions on the side of the lesion.

Nuclear palsies may result from a number of inflammatory processes within the brain stem : e.g. epidemic encephalitis, disseminated sclerosis, acute poliomyelitis, botulism, chronic alcoholism (Wernicke's encephalitis hæmorrhagica superior acuta). Barbiturate poisoning, diphtheria and diabetes may also be associated with ocular palsies of nuclear origin. Finally, there is the rare condition known as *chronic progressive nuclear ophthalmoplegia*, in which there is a primary degeneration of the third, fourth and sixth nuclei. The course of this malady, which is related to progressive muscular atrophy, is slowly progressive, and finally all the extrinsic ocular muscles are paralysed.

PATHOLOGICAL CONDITIONS OF THE PUPIL AND OF ACCOMMODATION.—Myosis or unusual smallness of the pupil is a common sign of syphilis of the nervous system. It is an important sign of paralysis of the cervical sympathetic. It occurs in lesions of the pons below the third nerve nucleus, is often met with in advanced age without pathological associations, and is also a symptom of the morphine habit.

Eccentricity of the pupil and deviations of its form from the circular are important signs of nervous syphilis, and these signs occur also in lesions of the foremost part of the third nucleus.

Inequality of the pupils occurs in connection with all nuclear and peripheral ocular paralyses, and with cervical sympathetic paralysis. It accompanies all defects of vision from lesions of the visual path between the eye and the external geniculate bodies, provided the appreciation of light be unequal in the two eyes. It may be congenital or associated with inequalities

of the refraction of the two eyes, and then has no pathological significance. It is commonly a sign of nervous syphilis.

The Argyll Robertson pupil (reflex iridoplegia) as originally described includes loss of the light reflex, myosis, inequality and irregularity of the pupils. Atrophic changes in the stroma of the iris have also been described. These are best seen in blue-eyed persons in whom the stroma is not concealed by pigment, and they impart a fineness of texture and a pale, washed out tint that, in association with myosis, gives the eye of the tabetic so characteristic an appearance.

The iridoplegia may be due to a lesion in the region of the posterior commissure and the aqueduct of Sylvius, and it is here that some believe the essential lesion of the Argyll Robertson pupil to lie. But the Argyll Robertson pupil of neurosyphilis is almost constantly associated with the other pupillary phenomena noted above, and it is difficult to see how a central lesion could produce any of these. In short, the pathogenesis of this familiar phenomenon still remains obscure.

When reflex iridoplegia is met with apart from neurosyphilis—which is but rarely—it is not accompanied by myosis, or irregularity of the pupils, and the absence of these signs is of diagnostic importance.

It has been said to occur in disseminated sclerosis and in epidemic encephalitis, but the statement is one to be accepted with reserve.

THE MYOTONIC PUPIL.—During the past fifteen years a number of writers (Foster Moore, Holmes, Adie) in this and other countries have described a pupillary phenomenon which has the following components: There is no reflex contraction to light, and there is a very slow contraction on convergence, followed by an extremely slow relaxation and dilatation. In some patients this condition is associated with a total loss of all tendon jerks. The patient is more commonly a young woman in normal health, the phenomenon being discovered usually in the course of a routine examination of the eyes. It constitutes no disability. Serological examination of the blood and cerebro-spinal fluid is always negative, and the condition is not related to neurosyphilis or tabes, with which, however, it may be confused if a complete examination of the nervous system be not made.

Total internal ophthalmoplegia is met with in lesions of the anterior part of the third nucleus, and in lesions of the lenticular ganglion in the orbit.

Wernicke's hemianopic pupil phenomenon is a test for the position of a lesion causing hemianopia. If the lesion is situated upon the visual path where that path contains the light reflex path, the pupil does not react when light is thrown on the blind side of the retina. In other words, this sign is present if the lesion is involving the visual path between the eye and the external geniculate body. When the lesion is between the geniculate body and the visual cortex in the occipital lobe, the pupil reacts equally well from the blind and from the seeing field.

PARALYSIS OF THE CERVICAL SYMPATHETIC

Synonym.—Horner's syndrome.

So far as the eye and orbit are concerned, the sympathetic is the tonic retractor of the lid, the tonic protruder of the eyeball, and the tonic dilator of the pupil, and stimulation of this mechanism results in retraction of the

lid or widening of the palpebral fissure, exophthalmos and wide pupil, while paralysis of the cervical sympathetic produces narrowing of the palpebral fissure (cervical sympathetic ptosis), and a small pupil. It is customary to include enophthalmos amongst the components of cervical sympathetic palsy, but it is extremely doubtful that this is ever present. The excitation condition is seen in Graves's disease; the paralytic condition is of common occurrence in nervous diseases. The cervical sympathetic is also the tonic vaso-constrictor and secreto-motor nerve of the head generally, but disturbance of the mechanism does not often give rise to characteristic or important clinical phenomena. A curious lack of expression is, however, sometimes observable in the face on the side of the lesion. Cervical sympathetic paralysis occurs in the following clinical associations: (1) In all lesions of the cervical cord, especially when the last cervical and first dorsal segments or roots are damaged. It is common in syringomyelia. (2) In lesions of the cervical sympathetic trunk by trauma, pressure, growths, etc. (3) It is very common in tabes and nervous syphilis generally, where it appears as partial bilateral ptosis with small pupils. It appears to be a primary neuronie degeneration in this condition and never improves.

THE FIFTH OR TRIGEMINAL NERVE

Symptoms of Lesions of the Fifth Nerve.—Pain over the sensory distribution of this nerve occurs from irritating lesions and reflexly, if its periphery is irritated. With organic lesions in any part of its course, the pain is followed by sensory loss, corresponding with the part involved. The initial expression of this sensory loss in a progressive lesion of the fifth nerve: *e.g.* compression by an eighth nerve tumour, is loss of the corneal reflex. The peculiar disease, neuralgia, of which the pathological basis has not been as yet discovered, is practically confined to the distribution of this nerve. Herpes zoster over the distribution of this nerve is common, and results from a lesion by a virus infection in the Gasserian ganglion, and is in every respect comparable with that occurring in the distribution of the spinal nerves from similar lesions in the posterior root ganglia. It produces bad scarring, and when affecting the cornea is apt to produce ulceration, very destructive to the eye. It is accompanied by severe pain, which may be persistent for months. It should be borne in mind that the sensory supply to the cornea is entirely from the naso-ciliary branch, via the long ciliary nerves, and that herpes zoster of the cornea is usually accompanied by a small group of vesicles only at the tip of the nose on the same side.

Taste.—It has frequently been argued that loss of taste over the anterior two-thirds of the tongue follows destruction of the Gasserian ganglion and proximal portions of the fifth nerve. Cushing has, however, investigated this subject upon a series of cases of complete Gasserectomy, and has found that in every case the sense of taste was preserved. The path of taste thus seems proved. It is from the glossopharyngeal nucleus via the fasciculus solitarius, portio intermedia, facial nerve, chorda tympani and lingual nerve to the tongue.

Trophic changes.—Lesions of the first division of the fifth nerve are often productive of serious corneal deterioration and ulceration, which may be followed by septic panophthalmitis. These changes, however, have been

proved to be the result of mechanical damage upon the insentient surface. If, for example, after destruction of the fifth nerve for neuralgia, the eye be carefully protected either by covering or sewing it up, these changes do not occur. After a little while, the anæsthetic cornea becomes much less vulnerable, and will stand the wear and tear of ordinary life without disturbance.

Paralysis of the motor function of the fifth nerve occurs in lesions of the nucleus in the pons, or of any part of the peripheral course of the motor division. The signs of such paralysis are not apparent to the patient, who experiences no difficulty in mastication, provided the lesion be unilateral. To the observer, the jaw deviates to the side of the paralysis on opening the mouth, on account of the action of the unopposed external pterygoid of the sound side. The masseter, as felt by the finger on its anterior edge, does not harden on biting, nor do the temporal muscles harden. The floor of the mouth does not stiffen on the paralysed side on forcibly opening the mouth.

Bilateral involvement of all the muscles supplied by the fifth nerve is the rule in all cases of progressive muscular atrophy where the bulbar nuclei are affected.

THE SEVENTH OR FACIAL NERVE

This nerve supplies all the facial muscles of expression. In the petrous bone, it gives off a branch to supply the stapedius muscle. One quarter of an inch above the stylomastoid foramen, it gives off the chorda tympani, which enters a small foramen, the iter chordæ posterius, which leads it to the tympanum, where it crosses the long process of the malleus and enters the temporal fossa by a canal, the iter chordæ antierius, and subsequently joins the lingual, by which it is conveyed to the anterior two-thirds of the tongue and to the submaxillary and sublingual glands.

1. BELL'S PARALYSIS

Synonym.—Common facial palsy.

Definition.—A common variety of peripheral paralysis of the facial nerve, sometimes of uncertain ætiology and sometimes a sequel of herpes of the geniculate ganglion. The paralysis almost invariably recovers, but if the recovery is slow, a very peculiar spasm or facial contracture may accompany or follow the recovery.

Ætiology.—Facial paralysis is rare at the extremes of age and it is most common in early adult life. The sexes are equally affected. Many different views have been held as to the pathogenesis of Bell's palsy, as, for example, that it is the result of a local inflammation of the fibrous tissue forming the deep part of the sheath of the parotid gland, from which a process in the form of a sheath accompanies the facial nerve into the Fallopian aqueduct, and along which the inflammation extends and compresses the facial nerve in that canal. The proofs adduced in favour of this view are, that facial paralysis is so often accompanied at its onset by pain in the stylomastoid region and behind the mastoid process and by tenderness on pressure, and that in some cases there is very considerable swelling of the deep part of the parotid gland. Moreover, it might be thought that the pathological process begins outside, and subsequently spreads up the facial canal, since the loss of taste in the anterior two-thirds of the tongue, from involvement

of the chorda tympani, is so often not present when the palsy first appears, and develops in the course of a few days, as the inflammatory process spreads up the facial canal, and reaches the region where the chorda tympani leaves the facial trunk. However, it is apparent that this can be no more than speculation, lacking pathological confirmation. On the other hand, it is clear that a proportion of cases of Bell's palsy are a sequel of herpes of the geniculate ganglion. There is also, in the experience of the present writer, a curious periodicity in the appearance of cases of Bell's palsy in the late autumn and early spring. While this may perhaps be attributed to cold weather, it is perhaps more strongly indicative of an infective origin and suggests that geniculate herpes may be responsible for a greater proportion of cases of Bell's palsy than is commonly believed.

Symptoms.—The onset is usually rapid and sometimes even sudden. Pain of a neuralgic character below the ear, behind the mastoid process, or referred to the occipital region, is common, but it does not last more than a few days, and sometimes pain is entirely absent. On deep pressure upon the styloid region behind the ramus of the jaw on both sides, one can almost always elicit the fact that there is tenderness on the paralysed side, and sometimes obvious swelling of this region may be felt. The first sign of the facial paralysis is that the patient feels the face to be stiff when he attempts to move it. Subsequently, the paralysis appears rapidly, and the face is drawn over to the opposite side. The paralysed side is motionless, according to the degree and distribution of the paralysis, if incomplete, and, if complete, is expressionless. The eye cannot be closed, and there is epiphora from paralysis of the tensor tarsi. The paralysis at the corner of the mouth causes difficulty in articulation and escape of fluids on drinking, but the patient soon learns to dodge these disabilities. When the paralysis is partial it is nearly always the lower part of the face which is the most affected. The facial muscles soon become hyperexcitable to mechanical stimuli. In nearly all the severe cases, there is loss of taste over the anterior part of the tongue. It should be remembered that the sense of taste is confined to a very small area on the lateral edge of the tongue, some half an inch behind the tip.

There is never any pain in the distribution of the facial nerve. After a time, which may vary from a few days to two years, the paralysis begins to recover, and invariably this recovery appears in the upper facial region first, and in almost every case becomes complete. We have seen perfect recovery follow complete paralysis lasting 21 months. Bilateral Bell's palsy is not so rare as is supposed. When seen, there is usually a lapse of 4 or 5 days before the second nerve shows signs of paralysis. The paralysis may become complete on both sides, or upon one only. Perhaps less common is recurrent Bell's palsy. The writer has seen several cases of the kind, including one in which the patient has three separate Bell's palsies, two on the right and one on the left side of the face. All recovered.

Treatment.—Those who believe in a fibrositic aetiology of Bell's palsy will base their treatment on this hypothesis. Salicylates and iodides are given internally and mercurial inunction (3 grs. rubbed in over the mastoid region daily for 2 weeks). The local application of warmth and of such a counter-irritant as tincture of iodine may also be used. The patient is probably best kept in the house for the first week and instructed to massage the face gently using olive oil for 5 to 10 minutes daily. In cases which show

early signs of recovery this is adequate, but when after a month or 6 weeks no evidence of recovery is seen, undue stretching of the paralysed muscles may be minimised by "splinting" the face. For this purpose a silver wire, rubber covered where it turns round the lip, may be bent so as to hook round the lip at one end and over the ear at the other, so that the mouth is kept symmetrical during facial movements. Gentle massage may also be continued. In such cases it has been customary to give galvanic stimulation to the paralysed muscles. Since the present writer abandoned this practice many years ago, he has satisfied himself that recovery proceeds as quickly and completely without it, and the distressing facial contractures that are sometimes seen do not occur. In other words, electrical stimulation has no place in the treatment of Bell's palsy. When geniculate herpes is in question, the vesicles and the swollen pinna require the local treatment suitable for this condition.

Facial paralysis from caries of the temporal bone rarely makes any recovery, and it is almost always complete and permanent. To remedy the unsightly and permanent distortion of the face, union of the peripheral trunk of the facial to the central end of the divided spinal accessory or preferably the hypoglossal nerve, has been performed, and with considerable success. Section of the hypoglossal with consequent hemiatrophy and hemiparalysis of the tongue produces no disability with speech, mastication or swallowing. It is not so much that reunion of this nerve restores volitional power to the face, but associated movement does return and also some after-contraction, which restores to some degree the symmetry of the face.

Facial Paralysis from herpes of the geniculate ganglion.—Among the not infrequent causes of facial palsy must be numbered geniculate herpes. Attention was first drawn to this cause by Ramsay Hunt. The herpetic vesicles, preceded by local pain, appear in the external auditory meatus and adjacent parts of the pinna, and sometimes also just behind the pinna and on the soft palate and anterior pillar of the fauces. When the innervation of the last named derives fibres from the geniculate ganglion, the clinical picture of geniculate herpes is apt to be a misleading one if it be not thought of. The patient complains of pain in the ear, and in the throat on the same side. As the eruption develops the fauces on the affected side are red and injected, and several small ulcers (ruptured vesicles) may be seen. At the same time, the vesicles appear in the ear, rupture, and give off a watery discharge which may be mistaken for otorrhœa. The pinna may then swell very considerably. After some days, during which the patient may feel ill and be feverish (temperature of 100° to 102° F.), a facial paralysis almost invariably develops and becomes complete within 12 hours. In milder cases there may be only initial pain in the pinna and the appearance of herpetic vesicles on the pinna without much swelling. It is in the severe cases that an erroneous diagnosis of middle ear disease with otorrhœa may be made and hazardous and unnecessary steps be taken to deal with this. According to Ramsay Hunt facial palsy always follows geniculate herpes, and undoubtedly many cases of this kind, where the herpetic eruption is minimal, escape accurate diagnosis.

Diagnosis.—Care in diagnosis is necessary lest peripheral facial palsy of very unfavourable prognosis should be mistaken for it. The facial palsies which result from lesions of the nerve in the temporal bone, from caries and

from tumour, those due to lesion of the nerve within the skull and from pontine lesions, rarely make any recovery. To this rule the following exceptions must be made: In the peripheral facial paralysis of poliomyelitis, lethargic encephalitis, tetanus and diphtheria recovery always occurs, if the patient survives.

Facial contracture.—In cases of long duration when recovery commences, the face goes into a condition of persistent spasm which causes often a very unsightly distortion of the face, which is very disappointing to the patient, who after waiting many months for improvement, now finds the place distorted in the opposite direction and to a more severe degree than at the onset. No adequate explanation of facial after-contracture has ever been put forward, and no similar condition occurs after the lesion of any other peripheral motor nerve, so far as we are aware. It recovers slowly in the majority of cases. Patients should be warned from the first about the occurrence of after-contracture so that disappointment may be obviated, and at the same time encouraged as to the probability of complete recovery.

The diagnosis is not difficult, and mistaken diagnosis means faulty examination. In disease of the temporal bone, the facial palsy is accompanied by signs of such disease, which should be carefully sought, namely, deafness, perforation of the drum, discharge from the ear, and signs of long-standing otitis.

Lesions within the skull are apt to co-involve the auditory nerve, the fifth nerve of the cerebellum, and the characteristic signs of tumour of the lateral recess are common. In the pons, hemiplegia, hemiataxy and hemi-anæsthesia are likely to coexist.

Course and Prognosis.—Recovery is so usual that it should be promised in every case. The date of recovery is often difficult to forecast. If at the end of a week after the onset there is the slightest trace of any voluntary power in the orbicularis palpebrarum, which is the “ultimum moriens” of the facial muscles, or if any trace of faradic excitability to bearable stimuli remains, then it may be confidently said that recovery will be complete and rapid within 3 months, and that there will be no contracture. Cases in which no complete paralysis occurs in any region of the face usually recover in a fortnight. In complete cases, with complete reaction of degeneration in the muscles, it is difficult to say when recovery will occur or when the effect of contracture will be at an end. Cases which show no loss of taste and, therefore, in which there is no great extension of the inflammatory process up the facial canal, usually recover rapidly. Traumatic facial paralysis from blows upon the side of the face, and obstetrical facial paralysis from the pressure of forceps during delivery, always recover and leave no sequelæ.

2. PERIPHERAL FACIAL SPASM

Synonym.—Facial hemispasm.

Definition.—A unilateral malady of the facial nerve, in which inter-mitting spasm of the facial muscles occurs, exactly like that caused by faradism of the facial trunk. Rarely it is associated with a slowly oncoming facial paralysis, and may follow a facial paralysis due to injury.

Ætiology.—This malady occurs in adults, and the onset is usually in-

sidious and without known cause. It is most often seen in middle-aged women. It is certainly due to a lesion of the peripheral facial nerve trunk, and this lesion seems to be of such a nature as to irritate, and not in most cases to destroy; but in rare cases partial destruction, with the appearance of partial facial paralysis, does occur.

Symptoms.—It commences with twitching of some part of the facial musculature, which occurs at first at rare intervals, and subsequently becomes more and more frequent, so as in some cases to be almost continuous. Commencing locally, it tends to spread so as to involve the whole face in a sudden and hideous contortion. We have seen cases in which the attacks of peripheral facial spasm at first glance almost exactly resembled a Jacksonian fit of the face. The spasms may be so severe and continuous as to keep the eye closed for long periods together, and to interfere greatly with the work and enjoyment of life. The malady is associated with no other symptoms. Cases exist in all degrees of severity, from the mildest, in which an occasional flicker of the face occurs, to the most severe and incapacitating and unsightly malady.

Course and Prognosis.—Some of the cases recover spontaneously, and others under treatment; but when the malady becomes severe and the spasm hardly remitting, it is practically intractable, except by operative interference.

Treatment.—In the milder cases, measures calculated to subdue chronic inflammation, such as mercury, iodides and salicylates, are said to be of benefit. In severe cases, the only remedy which affords relief is the injection of alcohol into the facial nerve either at the stylomastoid foramen, or as it crosses the ramus of the jaw half an inch below the external auditory meatus, or when one division of the nerve only is affected, in any part of the *pes anserinus*.

THE AUDITORY AND VESTIBULAR NERVES

Lesions of the cochlear nerve produce deafness, and in addition pathological changes in its peripheral termination are productive of tinnitus. Except from direct involvement of this nerve or of its terminations in the cochlea, deafness is practically unknown as a symptom of disease of the nervous system. In other words, lesions of the central auditory paths are not as yet recognisable by any known symptoms.

Nerve deafness may be produced by any lesions of the cochlea and cochlear nerve, and is confined to diseases of the temporal bone and labyrinth, lesions of the internervine part of the eighth nerve by tumours, meningitis or pressure, and lesions of the lateral side of the medulla. The deafness is the same wherever the lesion may be, and the position of the lesion is to be deduced from the associated involvement of contiguous structures. Nerve deafness, which characterises lesions of the cochlea and its nerve, is distinguished from deafness due to middle-ear disease by the facts that hearing both by air conduction and by bone conduction is diminished or lost, while in middle-ear deafness the hearing by bone conduction is increased. If a tuning-fork in vibration be applied to the forehead until it is no longer audible, and then presented to the ear, it will not be heard aerially in middle-ear disease, since the aerial conduction is impaired in that condition. But in nerve deafness it is either not heard through the bone when the tuning-fork

is applied, or if heard, when it has ceased to be audible to bone conduction, will still be audible when presented to the ear. This is known as Rinne's test, and it is a reliable one. Weber's test for nerve deafness consists in the application of a tuning-fork to the forehead in the middle line, the patient being asked which ear the sound comes to most. In middle-ear deafness the sound is heard best on the deaf side, and in nerve deafness it is best heard on the sound side. As a symptom of nervous disease, nerve deafness is met with in disease of the lateral region of the medulla, in tumours of the cerebello-pontal angle growing from the eighth nerve, following epidemic meningitis, and in syphilis of the nervous system, especially congenital syphilis.

1. TINNITUS

Ætiology.—Tinnitus or the occurrence of persistent recurring noise referred to the ears may be produced by wax in the ear, by otitis media, or by any other condition of vascular congestion, or by inflammation in the region of the auditory mechanism. It occurs in those who work exposed to deafening noise, as in boiler-makers and riveters, and may be produced by the administration of quinine and salicylates. It is much more frequently indicative of intractable disease of the cochlea, which often ends in complete deafness. Persistent tinnitus is a malady of adult life, the earliest cases occurring after puberty. It is rare for the malady to commence in old age. It begins insidiously, and as a rule without cause, but debilitating influences may precede its onset.

Diagnosis.—The diagnosis of tinnitus presents no difficulty. A careful examination of the ears will discover and cause to be removed any local trouble in the external auditory meatus and tympanum. Moreover, these conditions do not give rise to persistent tinnitus with nerve deafness.

Symptoms and Course.—The sounds commence faintly and often intermittently, and at first may be only perceived in stillness and silence at night, and later become louder and more persistent, and are often absolutely continuous. The slight sounds may be low pitched, a low rumble like a distant wagon, or a faint murmur such as may be heard when a shell is held to the ear. The loud sounds are never low in tone. They may be humming, hissing, rushing or bell-like noises. The common simile used by the patient is that of a hissing kettle, of a gas jet, of a threshing machine, of a steam-engine, or of a room full of machinery. The same patient may have several sounds, sometimes successive and sometimes heard all at once. When the sounds are rhythmical they are usually synchronous with the pulse. In some of the cases labyrinthine vertigo occurs, and the attack may be heralded by an increasing intensity of the sound. The condition of hearing in patients suffering with tinnitus may vary in each case, and from time to time in any one case. In many cases hearing is perfectly normal, and may remain so for years, in spite of increasing tinnitus. One of my patients retained perfect hearing for over twenty years, with increasing tinnitus; but the hearing rapidly declined afterwards. In many cases, however, there is some degree of nerve deafness on one or both sides. In the course of time the deafness increases even to absolute deafness, and in a few of them the noises persist in spite of absolute deafness. As a rule, the noises decrease as deafness becomes severe.

Prognosis.—The prognosis is very uncertain, and in most cases unfavourable. In many cases the noises persist in spite of all treatment, sometimes treatment secures considerable relief, and not infrequently the symptom is removed by treatment.

Treatment.—In the early stages the disease may be much benefited by the exhibition of salicylates and iodides. As a symptom, tinnitus is more affected by bromides than by any other drug, and these should be given in doses of from 10 to 20 grains twice or three times daily. The effect of the bromide is sometimes increased by the addition of from 5 to 10 minims of tincture of belladonna.

2. VERTIGO

Definition.—The word “vertigo,” which by derivation means a “turning,” is used to designate any movement or sense of movement or unsteadiness either in the individual himself (subjective vertigo) or in external objects (objective vertigo) that involves a defect, real or seeming, in the equilibrium of the body. It is a sensation of involuntary movement, either of subject or of external objects. It always involves a slight interference with consciousness, which, in severe vertigo, is often momentarily lost.

Ætiology.—Vertigo is always the result, direct or indirect, of disturbance of the labyrinth, vestibular nerves or cerebellum. It is commonly associated with vomiting and with vasomotor and secretory phenomena, such as “cold perspiration.” The disturbance of the vestibular mechanism which results in vertigo may be set up by multitudinous causes, among which may be mentioned toxic states as in specific fevers, and from the administration of alcohol, anæsthetics and morphine, irregularities of blood supply as in fainting, loss of blood, cardiac feebleness, Stokes-Adams’ disease, and sudden alterations of position and in arterial disease; from visual or bodily disorientation as in diplopia, dancing, swinging, sea-sickness and train-sickness; in anæmic states; in migraine, and as an aura in epilepsy; in diseases of the tympanum, labyrinth and semicircular canals; in diseases of the vestibular nerve and cerebellum, and in conditions of raised general intracranial pressure.

Diagnosis.—The vestibular mechanism is closely connected functionally with the cerebellum, and the symptoms which result from its disturbance are almost identical with those resulting from lesions of the lateral lobe of the cerebellum, and comprise nystagmus to the side of the lesion, vertigo, forced movements, hemiataxy and hypotonus on the side of the lesion. There are two points which serve to separate the two conditions. In the first place, vestibular lesions are usually associated with nerve deafness, which is absent in cerebellar lesions, and secondly, the cerebellar symptoms are only marked in vestibular lesions when the condition is acute, or during acute exacerbations.

Tests for vestibular lesions.—1. Barany’s calorice test is made by irrigating the external auditory meatus with either hot or cold water or air. With an intact vestibular mechanism this causes irritation of the vestibular apparatus with the appearance of nystagmus or lateral deviation of the eyes to the side of the irrigation. When the vestibular mechanism is impaired this test fails relatively or completely.

2. If the patient be rotated either by placing him in a special rotating chair, or by turning him round several times in the standing position, lateral

conjugate deviation of the eyes immediately after the rotation will show nystagmus in the opposite direction to the rotation, if the labyrinth on that side is intact. It will not appear if the functional activity of the vestibular mechanism is deficient.

3. MÉNIÈRE'S DISEASE

Synonym.—Labyrinthine vertigo.

Definition.—A malady in which paroxysmal attacks of severe labyrinthine vertigo occur at irregular intervals, associated with tinnitus and progressive deafness, and due to disease of the labyrinth of a chronic nature.

Ætiology.—The cause of this symptom-complex has always been obscure, and this is not surprising since until a recent careful pathological examination of the labyrinth in two cases of typical Ménière's syndrome by Hallpike nothing was known of its underlying morbid anatomy. Ménière's original hypothesis was that hamorrhage into the labyrinth as the responsible factor, but is inherently improbable and lacks pathological support. According to Hallpike the essential lesion is a gross distension of the endolymph system together with degenerative changes in Corti's organ and the presence of albuminoid coagula throughout the endolymph spaces. He regards these changes as incompatible with an infective origin, and as probably primarily degenerative in nature.

The correlation of these changes with the paroxysmal character of the vertigo can at present only be surmised, but Hallpike believes that the attacks are probably due to rapidly induced bouts of asphyxia of the labyrinthine end-organs caused by rapid rises of fluid pressure in response to small volume increases in the endolymph.

Symptoms.—The attacks set in suddenly with a buzzing noise in the ears, followed immediately with intense vertigo, both subjective and objective. The vertigo may be so intense that the patient feels he is hurled to the ground. He often falls as if shot; sometimes he has time to assume the sitting or lying position, before the vertigo reaches its height. Consciousness is often lost, or seriously impaired, for a few moments only. Spontaneous nystagmus occurs to the side of the lesion, and unilateral cerebellar signs on the side of the lesion. The patient becomes nauseated, and often vomits repeatedly. The skin is pale and covered with a clammy sweat. The patient lies perfectly still, and in terror lest the least movement should bring on more vertigo. The duration of the attack and the time taken in the recovery from an attack vary from a few minutes to 24 hours. Sometimes the attacks are excited by some sudden movement, such as coughing or sneezing, but they are usually without any such antecedent. They may occur during sleep, and wake the patient. The recovery from the attack is usually perfect, the vertigo disappearing; but in some cases slight persistent vertigo remains between the attacks. When Ménière's disease is persistent a slow onset of nerve deafness and signs of slow vestibular destruction follow, and as these signs deepen the attacks become less and less severe, and finally cease when the functions of the labyrinth become destroyed.

The **Diagnosis** of Ménière's disease presents no peculiar difficulty, for the symptoms are highly characteristic, and although the attacks vary in the degree of their severity, from a slight momentary giddiness to a sudden falling, with the most acute cerebellar symptoms, yet the first attack is usually

severe. The rapid disappearance of the symptoms is striking. Vertiginous attacks from all other causes must be excluded. In epilepsy consciousness is usually lost. In Ménière's disease it is momentarily impaired, and there is no convulsion. In acute cerebellar lesions the symptoms are very like those of labyrinthine vertigo, but they are not transitory in a few hours. A careful search of the nervous system for signs of organic nervous disease should in every case prevent any mistake.

Prognosis.—The outlook in Ménière's disease is uncertain. Some cases go from bad to worse in spite of treatment, and progressive deafness ensues with disappearance of the attacks. Many cases, however, recover perfectly with little or no impairment of hearing.

Treatment.—The salicylates seem to have a definite specific effect upon the morbid process, and should be given in doses of 20 grains thrice daily. In the form of aspirin they may be even more beneficial from the sedative effect of the latter drug. The bromides have a wonderful effect in relieving the symptoms, and in averting the attacks, to the extent that it may be said that labyrinthine vertigo may be almost diagnosed by the beneficial effect of bromides upon it. They should be given in doses of from 10 to 20 grains three times a day. Syphilis must be excluded, and if present, treated. Counter-irritation of the mastoid region has been recommended, and can certainly do no harm.

THE NINTH OR GLOSSOPHARYNGEAL NERVE

Lesions of this nerve involve loss of taste over the posterior one-third of the tongue with some unilateral paresis of the pharynx. It is rarely involved alone; but, with the other nerves taking origin in the neighbourhood, by tumours of the lateral region of the medulla.

THE TENTH OR VAGUS NERVE

This nerve is a mixed nerve. The motor fibres supply the voluntary muscles of the soft palate (except the tensor palati), pharynx and larynx in conjunction with the accessory fibres, and the non-striated muscles of the respiratory and alimentary tracts.

The sensory fibres of the vagus supply the respiratory tract, the pharynx and œsophagus. Its visceral fibres supply the lungs, heart and abdominal viscera. No sensibility seems to be supplied to the abdominal viscera by this nerve, since with division of the spinal cord above the offshoot of the splanchnic nerves all sensibility in the abdomen is lost.

LESIONS OF THE VAGUS.—The important signs of lesion of this nerve and its nuclei are pharyngeal and laryngeal paralysis and loss of sensibility. Symptoms indicative of lesions of its complicated and mysterious visceral supply are neither well marked nor well understood, and in unilateral lesions seem to be entirely absent; they are therefore not considered.

Lesions of the vagus in the medulla are common. Syringomyelia, when affecting that region, usually involves the nucleus ambiguus, causing unilateral palsy of palate, pharynx and larynx. Thrombosis of the posterior inferior cerebellar artery which supplies that region of the medulla containing the nucleus ambiguus is likely to produce vagus paralysis of the same side. Pro-

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gressive muscular atrophy, in the form of progressive bulbar paralysis, may affect its cells, as do often polyneuritis and lethargic encephalitis and rabies. Lesions of the nerve roots often occur from tumours of the lateral region of the medulla, and growths outside the medulla from nerve roots and meninges, and here the lesion of the vagus roots is associated usually with those of the glossopharyngeal, spinal accessory and hypoglossal. In the neck perforating wounds and growths may implicate the nerve, and in the thorax tumours, particularly aneurysms and new-growths, are apt to cause paralysis of the muscles supplied by its recurrent branches.

Unilateral pharyngeal paralysis.—This is characteristic of all unilateral lesions of the vagus high up. It is recognised by the low-lying motionless palate and the loss of sensibility of one side of the pharynx, with loss of the pharyngeal reflex on that side. There is no impairment whatever of deglutition.

Bilateral pharyngeal paralysis.—This results from nuclear lesions of the nucleus ambiguus on either side, and is common in diphtheria, polyneuritis, myasthenia gravis and progressive muscular atrophy. The whole palate is low and paretic or paralysed, the voice is nasal, there is nasal regurgitation of liquids, the cheeks cannot be forcibly blown out, and there is difficulty in pronouncing final “b” and “g,” the words “rub” and “egg” becoming “rum” and “enck.”

Total unilateral laryngeal paralysis.—Since the superior laryngeal nerve which supplies the cricothyroid muscle, which is the chief tensor and adductor of the vocal cords, is given off high in the neck from the ganglion of the trunk of the vagus, it follows that total paralysis of the larynx on one side can only result from a lesion of the vagus, between the ganglion of the trunk and the nucleus ambiguus in the medulla. The vocal cord on the paralysed side is motionless in the cadaveric position—that is, midway between abduction and adduction. The larynx is insensitive on the same side. There is some loss of tone of voice but no stridor.

Unilateral abductor paralysis or recurrent laryngeal paralysis.—This occurs from all lesions of the trunk of the vagus below the ganglion of the trunk, and from lesions of the recurrent laryngeal branch. The vocal cord on the side of paralysis lies close to the mid-line. It fails to abduct on taking a deep breath. There is no change of voice; but there may be slight stridor on inspiration—the sensibility of the larynx is not affected.

Bilateral abductor paralysis.—This condition is most commonly seen in the earlier stages of nuclear laryngoplegia, and is most often met with in tabes, sometimes in bulbar paralysis, and we have seen it in disseminated sclerosis. It occurs also in bilateral lesions of the recurrent laryngeal nerves in the thorax, which may occur from aneurysm and new-growths. It is the most dangerous form of laryngeal palsy, as the vocal cords cannot be abducted from close to the middle line, and they tend to open during expiration, but to suck together during inspiration, and for this reason may cause death from asphyxia, or necessitate laryngotomy.

THE ELEVENTH OR SPINAL ACCESSORY NERVE

This nerve may be caught with the vagus by lateral lesions outside the medulla, or by lesions in the region of the jugular foramen; but it is more

often damaged by injuries to the neck, and by operations for the removal of cervical glands. The spinal accessory nerve, as it crosses the posterior triangle of the neck, is very liable to injury, either from blows or from sudden strains, and most of the isolated trapezius palsies are due to local neuritis of the nerve trunk, so arising. Paralysis and wasting of the sternomastoids is conspicuous in most cases of myotonia atrophica. That of the trapezius is often conspicuous in the facio-scapulo-humeral type of myopathy. Both muscles are commonly affected in progressive muscular atrophy.

When the sternomastoid is paralysed there is neither weakness complained of, nor deformity, nor peculiar attitude of the neck, other muscles compensating for its paralysis. The muscle does not harden when turning the head to the side opposite the paralysis, and its reaction to faradism is diminished or lost.

Paralysis of the trapezius, on the other hand, causes great disability in raising the arm above the horizontal level of the shoulder and also difficulty in shrugging the shoulder or approximating the scapula to the middle line behind and therefore also in carrying the extended arm backwards. It produces a very ugly deformity, for the scapula unsupported by the trapezius rotates so that the superior internal angle appears as a hump in the slope of the neck above the clavicle, and there is also winging of the angle of the scapula with the axillary border of that bone horizontal. This paralysis of the trapezius may be confused with that of the serratus magnus, for in both winging of the angle of the scapula is marked. In trapezius palsy, however, the deformity is much more marked, the scapula is farther away from the spine, and is much more rotated. Tests for the movements of these two muscles and the faradic excitability should prevent any confusion.

COMBINED LESIONS OF THE NINTH, TENTH, AND ELEVENTH NERVES

No account of the glossopharyngeal, vagus and accessorius nerves is complete which does not consider their clinical interrelationships. Not only are they closely associated in part of their peripheral course, but their central origins are very intimately connected. It is therefore not surprising that in both central and peripheral nervous lesions two or more of them may be involved. A number of characteristic syndromes have thus been observed and described. We may classify these according to whether the causative lesion is intramedullary, or extramedullary (at the base of the skull, or in the neck).

Intramedullary lesions include thrombosis, nerve cell degeneration (chronic bulbar palsy), and syringobulbia; and its component symptoms (*syndrome of Avellis*) are unilateral paralysis of the palatal, pharyngeal and laryngeal muscles, with a crossed hemianæsthesia of syringomyelic type (for pain and temperatures).

At the base of the skull injuries or new growths involving the jugular foramen may give rise to *Schmidt's syndrome*: unilateral paralysis of pharyngeal, laryngeal, palatal, sternomastoid and trapezius muscles; or to *Jackson's syndrome*, in which a hypoglossal palsy may be added to those of the above syndrome.

Injuries high in the neck may produce *Tapia's syndrome* (first described

in bull fighters from penetrating wounds caused by the bull's horn), which consists in unilateral paralysis of the vocal cord and tongue, the palate being intact.

THE TWELFTH OR HYPOGLOSSAL NERVE

The nerve supplies all the muscles of the tongue, both intrinsic and extrinsic.

Unilateral lesions of the hypoglossal nerve are usually the result of tumours in the lateral region of the medulla, or local lesions just lateral to the medulla, and catching the nerve roots. Hemiatrophy of the tongue is perhaps more commonly seen in tabes than in any other condition. A hemiatrophy also occurs in cases of facial hemiatrophy, where the lower distribution of the fifth nerve is the region affected; but this variety does not involve paralysis of the tongue. Spastic paralysis of the tongue, with well-marked dysarthria and dysphagia, occurs in double hemiplegia and amyotrophic lateral sclerosis. Atrophic paralysis of the whole tongue, with exactly similar defects of articulation and swallowing, occurs when the hypoglossal nuclei are affected, and is commonly seen in progressive bulbar paralysis and sometimes in polyneuritis and myasthenia gravis. The sole physical sign of a lesion of one hypoglossal nerve is atrophic paralysis of one side of the tongue with loss of faradic excitability. The affected side of the tongue shrinks and comes in the end to consist solely of mucous membrane, fibrous tissue and glands. The tongue becomes sickle-shaped, with the concavity on the paralysed side. There is little impairment of movement, and no defect of articulation from a unilateral lesion.

The treatment is that of the condition causing the paralysis.

TRIGEMINAL NEURALGIA

Synonym.—Tic Douloureux.

Definition.—A disease of the fifth cranial nerve, in which no definite morbid changes in the nerve have been discovered, and in which no loss of function, either motor or sensory, occurs in the distribution of the nerve. The chief feature of the malady is the occurrence of pain of varied intensity which tends to be paroxysmal, and is often excruciating. Tenderness over the branches of the fifth nerve is always present during the bouts of pain, and when the third division of the trigeminal nerve is affected there is conspicuous unilateral furring of the tongue when pain is present.

Ætiology.—The malady is first met with at the age of puberty; it is not seen in childhood. In the earlier years of adult life it is often a mild and curable condition, though notable exception to this rule may occur; but as age advances, and especially after the age of 50 years, it tends to be increasingly severe and intractable by any measures save those for the destruction of the affected branch of the nerve, or of the Gasserian ganglion. No causal factors can be adduced. The sexes are equally affected. It is much more common in cold and damp climates than in southern and dry countries. Any debilitating influences, such as overwork, general ill-health and specific fevers, especially influenza, may precede the onset of the malady.

Symptoms.—The chief feature of the malady is pain, which may be general throughout the area of distribution of the nerve, but which is more commonly confined to one of the three divisions of the nerve and often to one branch of a division. It is characteristic for the pain of neuralgia to commence locally, and subsequently to spread in each attack and gradually, in the course of the disease, permanently to invade a larger area. Two different kinds of pain occur, the sharp and paroxysmal, and the dull and continuous pain. The paroxysmal pains are sudden in onset and in cessation. They have a lightning-like character, and are described as piercing, knife-like, or as if the affected region were penetrated by red-hot wires. Often quite spontaneous, these pains may be brought on by touching the surface, by a cold draught, by movement of the face and jaw, or by the act of swallowing, and in this last condition mastication and deglutition may become so difficult as to render feeding the patient a matter of great anxiety. When the paroxysms are occurring in a severe case the patient remains for a period, which may be from a few minutes to several hours, paralysed under the fear of the pain, unable to move a muscle lest a spasm more dreadful than the last should occur. The paroxysmal pains are usually followed, if severe, by a more lasting dull continuous pain often of a boring character, and sometimes such pain becomes absolutely continuous. The skin over the affected region is sore and tender after the paroxysm, and the patient may be unable to bear brushing the hair or shaving the face. The pain may be of every degree of severity, from mild momentary starts to continuous incapacitating pain, interrupted only by excruciating attacks of agony which render life a piteous burden. The distribution of the pain may be anywhere or everywhere in the distribution of the trigeminal nerve. The lightning-like onset of the agony often causes convulsive spasm of the face and of the body and limbs, and from this feature the names "tic douloureux," "spasmodic neuralgia," and "epileptiform neuralgia" arose. The tender points of Valleix are constantly present during the attack, and for some little time after. When the first division is affected, the tender points are found above the supra-orbital notch, over the external angular process, on the upper outer aspect of the nose, and on the globe of the eye. When the superior maxillary division is affected the chief tender point is over the infra-orbital foramen, while other points may be found over the points of exit of the temporo-malar nerves and in the roof of the mouth. When the third division of the nerve is involved, the chief tender points are over the mental foramen, the side of the tongue, and just in front of the external auditory meatus. When the third division is affected, unilateral furring of the tongue, which always occurs when the pain is present and which does not seem to occur with organic lesions of the fifth nerve, nor constantly in any other malady except neuralgia, is seen. Vasomotor and secretory disturbances are common. During the paroxysms, tears and saliva may flow in abundance. The trophic changes which have been described in the skin are usually the result of rubbing during attacks of pain, or of the application of heat or liniments. Local greying of the hair, however, does undoubtedly occur. The clinical picture of trigeminal neuralgia is completed with varying degrees of general physical ill-health, mental apathy and depression, which occur in proportion to the frequency and severity of the attacks, the presence of continuous pain, the ability to

take food and to sleep, and the possibility of taking any interest in life. It is surprising in England how few of the sufferers from severe neuralgia become habitual drug-takers.

Course.—In patients under the age of 40 years the malady is often transient and is completely and permanently recovered from, though even at this age cases occur which are only amenable to surgical interference. But when the malady commences after the age of 40 years, it is the rule for it to become progressively worse. The paroxysms become more severe, and occur at shorter and shorter intervals, continuous pain sets in, sleep and the taking of nourishment become difficult, and useful life becomes more and more restricted.

Diagnosis.—There should be no difficulty in making a correct diagnosis if proper care be taken. In the first place, all local cause for pain in the peripheral distribution of the trigeminal nerve should be excluded. The teeth should be most carefully examined and the jaws skiagraphed for any concealed disease, which should be put right if present. Organic disease of the fifth nerve can be excluded by the facts that such disease cannot long exist without signs of loss of function, which never occur in neuralgia. Diminution of sensibility, which is first marked perhaps by increased tolerance of the conjunctiva and cornea to touch, and weakness of the musculature with deviation of the chin on opening the jaw, and diminution of taste are certain signs of a local organic lesion. Moreover the pain of neuralgia, with its lightning onset and cessation, is hardly imitated by any pain of organic origin. Ocular conditions, such as glaucoma, which may give rise to agonising pain, can hardly be mistaken for neuralgia.

Treatment.—Having in the first place seen that all possible causes of local irritation in the region of distribution of the fifth nerve are absent, or, if present, adequately dealt with, it is essential to improve the nutrition and general physical health with tonic, dietetic and hygienic treatment, and such remedies alone will often cure slight cases. It is important to remember that in its early stages, the malady shows complete remissions of long duration. These remissions do indeed tend to become shorter after some years, but their occurrence suggests that in planning treatment it is essential to consider the circumstances of each individual case. Thus, if a patient who may be expected to enjoy a long period of freedom from pain can be tided over the present attack by medical means, it is clearly not wise to give an alcohol injection. This confers a long period of "cover" from pain which the patient will probably not require, and the premature recourse to injection means that in the end more injections may be called for than would otherwise have been needed.

With this qualification, treatment may be undertaken on the following lines: *Tr. gelsemii* in doses of from 10 to 20 minims thrice daily is an admirable remedy, and arsenic is a useful adjuvant. All the analgesic antipyretics of the coal-tar series are of great value, not only as immediate relievers of pain, but also as curative agents, and among these aspirin is most important. In cases where malaria has been recently present, quinine should never be omitted. In very severe cases, and when operation is to follow, morphine is an invariable temporary relief to the pain. But if persisted in, the beneficial effects of moderate doses soon disappear. In every case except in old subjects, a thorough trial of the above treatment should be made over a

sufficient period to make a competent judgment of its efficiency or inefficacy, as the case may be. When failure is met with, and in old subjects, who will be found to respond little if at all to such treatment, operative relief should be sought. In the first place, the injection of alcohol should be performed, and if this fail, as it sometimes does, on account of anatomical peculiarities of the individual, recourse should be had to the operation for dividing the fifth nerve proximal to the Gasserian ganglion. The permanence of the effect of alcohol injection varies, sometimes lasting relief is obtained; more often, after a period which varies from months to years, some return of the pain occurs. It is, however, a most difficult procedure for the operator, and requires great skill and experience. While absolutely devoid of risk in skilled hands, alcohol injection should never be undertaken by one who has not special training in its performance. The radical operation produces final cure.

GLOSSOPHARYNGEAL NEURALGIA

Definition.—A comparatively rare form of neuralgia within the distribution of the glossopharyngeal nerve. It is strictly comparable with trigeminal neuralgia in the quality and severity of the pain, its paroxysmal incidence, the remissions in its course, its provocation by special stimuli, and finally by the absence of any discoverable lesion in, or loss of function of, the nerve.

Ætiology.—Nothing is known of its ætiology. It is most frequently seen in middle-aged or elderly males. A symptomatic neuralgia of the same distribution is occasionally found in cases of carcinoma of the tongue in which the growth invades the faucial region.

Symptoms.—When fully developed, the malady consists in paroxysms of shooting pain of great severity in the region of the throat and ear. The exciting stimulus is commonly the act of swallowing. But just as in trigeminal neuralgia the pain may at first be confined to a single branch of this nerve, so in glossopharyngeal neuralgia, the pain may for long be confined to the tympanic branch, the pain being felt deep in the ear. This pain does not spread to the pinna. In other cases, pain in the faucial region predominates, the pharyngeal branches being affected. As in trigeminal neuralgia, the patient may enjoy long intervals of freedom from pain. During a paroxysm the patient screws up his face and may hold his head in his hand as does the subject of trigeminal neuralgia.

Diagnosis.—The presence of neuralgic pain of great severity, provoked by the act of swallowing, and in its general characters and behaviour resembling the very familiar and characteristic paroxysms of trigeminal neuralgia, but differing from these in its restriction to the ear and throat, occurring also in the absence of objective signs of a lesion of the cranial nerves: these together are the features which make a diagnosis of glossopharyngeal possible and easy.

Treatment.—In the early attacks, the same forms of medication employed in trigeminal neuralgia may be employed. If the pain does not respond to these, then surgical measures are called for. The tympanic branch of the nerve leaves the main trunk within the skull, so that when pain in the ear is present an intracranial section of the glossopharyngeal

nerve is necessary. On the other hand, when pain is confined to the distribution of the pharyngeal branches, division of the nerve high in the neck is adequate. But intracranial section appears to be the operation of choice.

FACIAL HEMIATROPHY

Synonym.—Parry-Romberg syndrome.

Definition.—A peculiar malady confined to some part of the distribution of the trigeminal nerve, or rarely extending from thence on to the area of sensory distribution of the upper four cervical nerves. It is characterised by a progressive atrophy of all the tissues, skin, subcutaneous tissue, muscle and bone, without sensory loss or paralysis. It comes to an arrest after a few years. No pathological condition has been discovered to account for the atrophy.

The disease may commence in childhood even as early as the second year, but it is most commonly started in early adult life. Females are much more often affected than males.

Symptoms.—The atrophy may be distributed over the whole area of the supply of the trigeminal nerve, or, as is more usual, may be confined to one or more of its branches. In general atrophy, a gradual diminution in the bulk of the whole side of the face is the first indication of the disease. When the disease is confined to one of the three great divisions of the nerve, the atrophy usually commences in one spot, commonly on the cheek just below the malar bone, where the skin becomes thin and pale from loss of pigment, and the down falls out. The submalar fat disappears, leaving an unsightly hollow. The atrophy spreads to the side of the nose, where the cartilages and bones become gradually smaller. The jaws gradually decrease in size upon the affected side, until they are too small to hold the teeth, which are actually pushed out by the decreasing size of the tooth sockets. The half of the tongue upon the affected side decreases in size, and thereby is rendered sickle-shaped. Even the eye may be remarkably lessened in size. The upper part of the first division of the fifth does not seem so liable to involvement, for it is rare to see any diminution of the size of the forehead, or dropping out of the hair of the scalp. The atrophy not uncommonly affects the ear. The skin in the end becomes very thin and parchment-like.

Treatment.—The only treatment is cosmetic, to improve somewhat the appearance of the face, by the injection of semisolid paraffin, to replace the fat and fill the unsightly submalar hollow.

THE SIGNS OF LOCAL LESIONS WITHIN THE SKULL AND BRAIN

Owing to the complete inaccessibility of the central nervous system to direct examination by any method comparable with those in use in the case of the viscera, the clinical localisation of disease within that system must necessarily depend upon the study and interpretation of disorders of function in tissues innervated by the nervous system. Many bodily functions have a localised representation in the brain and in the spinal cord. It must be remembered, however, that in clinical diagnosis we are concerned not directly with the localisation of functions within the nervous system, but with some-

thing rather different, namely, the localisation of symptoms of lesions. A simple example will serve to make this distinction clear. In a case of unilateral ataxy of movement, our object is to locate the lesion which by damaging some part of the nervous mechanism has allowed ataxy to develop. We may decide from our examination that this lesion is within the cerebellum, and we conclude that a destructive lesion of this organ is followed by ataxy. This ataxy is clearly produced by the activity of the intact remaining parts of the brain, working without the co-operation of the cerebellum. In this instance we have not localised any "function" of the cerebellum; we have simply localised the symptom following a lesion of this organ. Nor can we conclude that one of the functions of the cerebellum is to prevent ataxy, the fact being that the functions of this organ are still very imperfectly understood. Nevertheless, the localisation of symptoms of cerebellar lesions can be performed with reasonable accuracy.

This brings us to a brief consideration of the ways in which lesions within the nervous system may disturb its functions. The functions of a region of the brain that is directly involved in a disease-process may be deranged in either of two ways. They may be stimulated to overaction, or they may be impaired or destroyed. We may thus speak of "*irritative*" or *excitatory symptoms* on the one hand, and of *paralytic symptoms* on the other. A Jacksonian fit is an example of the first; a hemiplegia of the second. Further, although there is some measure of localisation of topographical and of functional representation within the brain, normally this organ works as a whole and derangement of the functions of one region may derange the functions of the whole, as we have already seen illustrated in the case of cerebellar ataxy. There is another way in which such general disturbance may follow a local lesion, and that is by what is known as diaschisis or shock. We see this mode of disorder in the coma which accompanies a cerebral hæmorrhage. In this state the cerebral hemispheres are for the time being out of action, even those parts that are not actually damaged by the lesion. Such *shock symptoms* are necessarily transient. A final group of symptoms are those we speak of as "*release symptoms*." When the coma of the hemiplegic subject has passed off, he is left with paralytic symptoms, namely, the hemiplegia. In a few weeks the paralysed limbs become spastic, their tendon jerks increase, and clonus makes its appearance. These symptoms of persistent overaction of nervous mechanisms freed by the lesion from the normal control of higher mechanisms, are what we refer to when we speak of release symptoms. Such symptoms may persist for years and may in some instances entirely dominate the clinical picture. But the practical task of localising lesions is sometimes even more complicated than this analysis of disorders indicates. The degree of disturbance of function produced by any lesion depends also on temporal factors. A suddenly arising lesion, such as an arterial occlusion or a hæmorrhage, or a direct injury is apt to produce a much more severe and widespread disorder of brain function than a slowly developing lesion. Thus, the intracranial cavity may come to accommodate a large new growth which compresses and markedly deforms the brain without giving rise to any subjective discomforts or disabilities, or to any abnormal physical signs discoverable on examination. Again, it is known that a chronic cerebral or cerebellar abscess is commonly present for some weeks before it reveals its presence by signs or symptoms. This is its period of clinical latency. Finally,

a tumour within the brain, while it may give rise to symptoms of a general rise of intracranial tension, such as headache, papilloedema and sickness, may yield on examination no localising signs, and this not necessarily because it is in what is known as a "silent area" of the brain. Or, it may cause but a minimal disturbance of local function even when large regions of known and specific function are directly involved. Finally, we have to take into consideration that space-occupying lesion within the skull or brain may come ultimately to produce indications of local disorder of function in parts of the brain remote from the lesion. These may be spoken of as false localising signs.

From what has been said it will be apparent that at least two factors determine the symptoms associated with disease within the brain, namely, (i) the localisation of the lesion, and (ii) the nature of the lesion. The latter determines its rate of development, its stimulating or paralysing effects upon the nervous tissue, and its capacity for producing remote effects.

Hence it is that the localisation of a lesion within the brain (the topographical diagnosis) and the determination of its nature (the pathological diagnosis) are frequently something more than a simple essay in applied anatomy and physiology, and that complete diagnosis calls also for a knowledge of the natural history of the different disease-processes, that is, for clinical experience. In this chapter we must be content with a brief consideration of the signs upon which we depend for the localisation of symptoms. We may take first the various regions of the brain, and secondly, since we have to deal not only with lesions within the brain, but also with all lesions within the skull, that may be outside the brain, we will consider the symptomatology peculiar to lesions in the three cranial fossæ.

THE CEREBRAL HEMISPHERES

General lateralising signs.—A lesion within or involving one hemisphere may reveal by the signs it produces whether it is right- or left-sided without affording further localising information. Such signs are unilateral diminution or absence of the abdominal reflexes, a unilateral extensor type of plantar response, and a just perceptible unilateral paresis of movements of the lower part of the face.

The frontal lobes.—These include that part of the hemisphere anterior to the ascending frontal convolution. The lesions to be met with in this region include tumour, abscess and thrombosis of the anterior cerebral artery, the last named being comparatively rare.

The syndrome of the anterior cerebral artery consists of spastic weakness of the crossed lower limb with the appropriate changes in the reflexes, sometimes a slight degree of weakness of the crossed face and arm, sometimes forced grasping and groping in the arm of one or both sides, and apraxia of the left arm. There may also be some mental obfuscation.

The syndromes of frontal lobe tumour vary according to the rapidity of development of the tumour and under other factors not fully understood. As a rule an early, if not the initial, symptom is a change in the patient's mental state. He becomes apathetic and lacking in initiative. The association and flow of ideas tends to fail. He sits about idly. He is apt to permit the unhindered passage of urine and even of feces, and to be totally indifferent to and unaware of the social embarrassments such conduct involves. This

form of "incontinence" is in fact a diagnostic symptom of great value in frontal lobe lesions. Rarely, the patient develops an abnormal facetiousness and euphoria—the so-called "Witzelsucht." Movement is disordered by the development of apraxia, and sometimes by that of forced grasping and groping, another useful sign of frontal lobe involvement, though it must be admitted one occasionally seen in lesions elsewhere in the cerebral hemispheres, as in a case under the care of the present writer in which bilateral grasping and groping and forced sucking were prominent features, the lesion being found at necropsy to be a bilateral degeneration of the thalamus. Nevertheless, this sign is an important one. It has been analysed by Walshe and Robertson into two components: (1) Volitional grasping movements made by the conscious patient when some object is felt by him in his palm or is seen by him to approach his hand. These movements wane and cease when consciousness is failing, or when attention is defective. (2) A true tonic reflex grasp of any object held in the hand, if this object be so pulled away by the observer (or by the patient with his other hand) so as to put the flexors of the fingers on the stretch. The flexors tighten as the pull is maintained and may become of great force, so strong indeed that sometimes the patient can be pulled out of the bed by this involuntary grasp which he is unable voluntarily to relax. This reflex may persist even though consciousness be lost. If the orbital lobule be involved, there may be bilateral anösmia and even direct pressure upon one optic nerve. These two symptoms will be further considered in connection with the syndrome of the anterior fossa of the skull. The tumour being an expanding and space-occupying lesion may when in this situation lead to the appearance of slight crossed hemiparesis, and when left-sided may be accompanied by motor or "expressive" aphasia. When, in the case of tumour, the corpus callosum is involved, the patient becomes completely apathetic and silent and immobile, displaying no initiative of any kind.

Syndromes of the central region (region of the "motor cortex.")—Hemiplegia is the characteristic paralytic manifestation of a lesion, Jacksonian fits of an "irritative" lesion. Such a fit may be followed by transient hemiparesis, and in the case of tumour by a slowly progressive and permanent hemiplegia. Local involvement of some part of this region will affect face, arm or leg predominantly according to its situation.

Conjugate deviation of the head and eyes may be met with, away from the side of the lesion, if this be irritative, as at the commencement of an apoplexy, or of a local fit, or towards the side of the lesion if the lesion be paralyzing. The two forms of conjugate deviation often follow the one after the other, from the same lesion which is at first exciting and afterwards paralyzing. Conjugate deviation of the head and eyes is seen with acute lesions rather than with those of slow development. The more deeply the lesion extends into the sublying white matter, the more does it tend to produce an extensive hemiplegia, since the pyramidal fibres converge from the cortex towards the capsule. Loss of localisation to sensory stimuli is not infrequent from simultaneous involvement of the neighbouring post-central convolutions.

Parietal lobe.—A characteristic series of sensory disorders may mark the presence of a lesion in this region. These include defective localisation of tactile stimuli, defective appreciation of two simultaneous contacts (Weber's compass test), defective appreciation of three dimensional space

(i.e. of size and form). There is, in addition, defective power of differentiating varying intensities of stimulus (painful or thermal), and a ready fatigue of sensory functions. The simple recognition of painful and thermal stimuli may be relatively intact. It will be seen that the defects in spatial discrimination which result from these modes of sensory loss lead to that inability to recognise and identify objects held in the hand, or to describe their size, shape or texture, which is known by the name of astereognosis. The appreciation of active movement and passive position is apt to be faulty, and some ataxy may result therefrom. Trophic changes may be observed in the periphery of the limbs, and lesions in this situation seem to be responsible for the arrest of growth which is seen in cases of infantile hemiplegia. Jacksonian attacks, consisting of a peripheral sensory aura, sometimes followed by convulsions, occur. These localising signs are confined to the opposite side of the body.

Occipital lobes.—Lesions of the cuneus and region of the calcarine fissure on the mesial aspect of the occipital lobe result in hemianopia of the opposite field, but central vision escapes. Gordon Holmes has found that if the lesion is limited above the calcarine fissure a quadrantic hemianopia of the lower field results, and if the lesion is below the calcarine fissure the quadrantic hemianopia resulting is of the upper field. Since central vision is represented at the posterior pole of the hemisphere, a lesion of the posterior pole causes central hemianopic scotoma, vision in the periphery of the field remaining intact. Consequently a bilateral lesion of both posterior poles will result in bilateral central scotoma, and a bilateral lesion of the calcarine region will produce blindness of both peripheral fields, central vision remaining intact. If the lesion extends deeply into the occipital lobe so as completely to sever the optic radiation to the occipital cortex, complete hemianopia, affecting both the central and peripheral part of the visual field, will occur. The hemianopias resulting from lesion of the occipital lobe are distinguished from those due to lesion of the optic tract by the fact that in the former the pupil reacts to light thrown on to the blind part of the field (Wernicke's hemianopic pupil phenomenon). On the outer surface of this lobe, a lesion extending deeply on the left side may sever the connection of the visual centres with the speech centres, and so produce word-blindness. Such a lesion is usually situated at the junction of the left occipital and temporal lobes. Jacksonian attacks are often of great value in occipital localisation, and take the form of visual hallucinations, often accompanied by transient hemianopia.

Temporal lobe.—The uncinate and hippocampal regions of this lobe are the cortical seats for taste and smell, and the localising symptoms which are rarely absent when lesions in this region exist are Jacksonian attacks in the form of hallucinations of taste and smell, nearly always of an unpleasant nature. The hallucination is often immediately followed by a "dreamy stare," during which smacking movements of the lips, or champing movements of the jaw, or spitting may occur. Experience seems to prove that all highly organised hallucinations, whether auditory, visual or psychic, when occurring from organic lesion of the brain, point to a lesion in the uncinate region. The senses of taste and smell are not lost from a unilateral lesion of this region, since they are bilaterally represented in the cerebral hemispheres. The outer surface of the temporal lobe is concerned with hearing, but from

the complete semi-decussation of the auditory path, unilateral lesions never produce detectable deafness. On the left side, however, the temporal lobe is concerned with speech, and destruction results in serious disorder of speech functions. Inasmuch as lesions of this region are situated far forward toward the insula, they result in "verbal aphasia," or towards the centre of the convexity of the temporal lobe, in amnesia, or lack of recall of words and "word-deafness," while if towards the posterior limits of the lobe they produce "word-blindness" from severance of the visual path to the speech region. Deeply seated lesions isolating the speech region from the incoming auditory path produce jargon aphasia. Jacksonian attacks, consisting of auditory hallucinations, which may or may not be followed by aphasia or by convulsions, may occur. Extensive lesions of the left temporal lobe cause much mental impairment. On account of the wide excursion which the optic radiation makes into the deep part of the temporal lobe in its course from the thalamus to the cuneus, homonymous hemianopia, especially of the upper quadrants, is very common in deep-seated lesions of the temporal lobes. The occurrence of incontinence of sphincters of a mental type is occasionally seen in some cases. When lesions extend deeply there may be a paresis of the opposite face for emotional movements, out of all proportion to the loss of volitional movements.

Internal capsule.—In this region, the chief motor tract is condensed into a small space, and is situated immediately in front of a narrowly localised sensory tract, while not much farther, posteriorly, the visual path enters the thalamus. Lesions of this region therefore produce severe and widely-spread hemiplegia of the opposite side, often associated with hemianæsthesia and not infrequently with hemianopia of the opposite side. From the proximity of the thalamus and corpus striatum, there is often involvement of these structures in a capsular lesion, with appearance of the characteristic, spontaneous involuntary movements and sensory loss.

BASAL GANGLIA

Optic thalamus.—A very characteristic clinical picture results from destruction by thrombosis of this structure which is termed the "thalamic syndrome" of Dejerine and Roussy; there is hemiparesis with spontaneous involuntary movements of the opposite side, which may be of the nature of tremor, intention-tremor, choreic, athetotic, dancing or irregular movements. Most post-hemiplegic involuntary movements are due to a lesion of the thalamus. In addition, there is hemianæsthesia, often with a characteristic hyper-sensitivity to aggressive stimuli, such as tickling, cold water, etc., which may produce agonising distress. Sometimes spontaneous, constant and unrelievable pain occurs on the opposite side. Emotional movement of the opposite face may be impaired much more than is volitional movement. The thalamic syndrome is not invariably, or even commonly, seen when the lesion is a tumour. In this case, as Smyth and Stern have pointed out, the symptom-complex varies according to whether the growth primarily arises in this structure or invades it from its lateral aspect. In the former case they arise in the sub-ependymal glia and spread laterally. They are characterised by early mental deterioration, with conjugate ocular palsies. Sensory changes are absent or only terminal in appearance. In

the case of tumours secondarily invading the thalamus from its lateral side, sensory changes of the order described under the "thalamic syndrome" of Dejerine and Roussy, Head and Holmes, are seen.

Corpus striatum.—Little is certainly known of the symptomatology of focal lesions of this structure, and the various syndromes (tremor-rigidity, athetosis) which have been described are associated with diffuse lesions involving other parts of the cerebral hemispheres in addition to the corpus striatum. However, a local lesion (thrombotic) in a neighbouring mass of grey matter, the corpus subthalamicum, or corpus Luysii, is followed by violent unilateral choreiform movements, the so-called *apoplectic chorea*.

Region of the falx cerebri.—Lesions of this structure are likely to affect both hemispheres equally. Tumours opposite the paracentral lobules cause bilateral crural monoplegia, and those in the posterior region of the falx, bilateral hemianopia. Thrombosis of the superior longitudinal sinus produces widely spread bilateral softening of the hemispheres, with double hemiplegia.

Corpora quadrigemina.—The oculo-motor nuclei lie on either side of the aqueduct of Sylvius, and lower down on either side of the middle line, in the floor of the upper part of the fourth ventricle, and lesions of this region cause nuclear ophthalmoplegia—that is, paralysis of both eyes in terms of the conjugate movements upwards, downwards or laterally. From before backwards, lesions of this column of oculo-motor nuclei will produce reflex iridoplegia, paralysis of accommodation, paralysis of upward, downward and lateral movements respectively. Immediately ventral to the oculo-motor nucleus and decussating beneath it, lie the superior peduncles of the cerebellum, involvement of which causes bilateral ataxy of limbs and trunk. Lesion of the dorsal part of the quadrigeminal layer produces a characteristic syndrome of nuclear ophthalmoplegia with bilateral ataxy, which is termed Nothnagel's syndrome. The pyramidal fibres for the face leave the pyramidal tract in this region of the tegmentum and may here be involved alone, causing bilateral spastic paralysis of the face. In the ventral portion of this region of the brain stem are the crura cerebri with the third nerve, perforating each crus to emerge upon its inner side, and the optic tract running round the crus from the geniculate bodies to the optic chiasma. A lesion of one crus will cause hemiplegia of the opposite side, and paralysis of the third nerve on the same side. This pathognomonic localising combination is known as Weber's syndrome. Situated a little more dorsally, a lesion of the crus will produce ophthalmoplegia of one eye with tremors and inco-ordination of the opposite limbs. This is known as Benedikt's syndrome. Extension of a lesion outwards from the crus will cause tract hemianopia, in which the half-fields are completely involved, with no light reaction from the blind fields. Interference with the fillet may cause hemianæsthesia.

PONS AND MEDULLA

In these regions the motor and sensory tracts, the cerebellar peduncles, the cranial nerve nuclei, and the outgoing cranial nerves are closely packed together, and the signs resulting from destruction of these will be varying combinations of spastic paralysis, ataxy and sensory loss—from interference with the long conducting tracts—in the body and limbs, with

nuclear and peripheral nerve palsies and anæsthesia in the region of the face. If the lesion is unilateral the body and the face will be affected on opposite sides, causing the "crossed paralyses" or "alternate paralyses" of lesions of the brain stem, of which facial palsy with contralateral hemiplegia, trigeminal palsy and sensory loss, and vagal palsy with contralateral hemiplegia are usual varieties. From the smallness of the brain stem lesions most often involve both lateral halves of this structure, and bilateral symptoms result. Lesions of the brain stem below the oculo-motor nuclei, cause small pupils (pontine myosis) from cutting off those nuclei from the spinal cord, whence the tonic dilator of the pupil—the cervical sympathetic system—emerges. Glycosuria may occur from interference with the vasomotor centre, and involvement of the respiratory centre is frequent. The common lesion involving the medulla is softening of the lateral region following thrombotic occlusion of the posterior inferior cerebellar artery, the so-called cerebellar apoplexy (see p. 1602).

CEREBELLUM

When lesions of this structure develop suddenly they are apt to produce more striking disturbances of movement than when they develop gradually, a point which it is important to remember when the presence of an abscess or a tumour within the cerebellum is suspected. Further, it is well to regard the several different components of cerebellar ataxy not so much as special disorders of different cerebellar functions, but as expressions of a single disorder, which owe their varying appearance to the varying nature of the clinical tests employed. The current nomenclature of cerebellar symptoms is redundant and complicates description, but it is universally employed and so is here adopted, but with no conviction of its scientific value.

Nystagmus.—In unilateral lesions there is a coarse nystagmus on deviation of the eyes to the side of the lesion, with a finer and more rapid movement on deviation away from the side of the lesion. In extensive lesions, there may even be considerable difficulty in deviation to the side of the lesion, and in this condition a true nystagmus may be present only on looking to the opposite side. In bilateral lesions the nystagmus may be symmetrical, but it may be entirely absent. Rarely—usually after acute lesions—such as gunshot wound or operative interference—the phenomenon of "skew deviation" may appear temporarily; the eye on the side of the lesion being displaced down and in, the opposite eye upwards and outwards.

Hypotonia.—This is usually seen in acute lesions rather than in chronic ones, and consists of a marked flaccidity and extensibility of the limb muscles, and leading to the "pendular" form of knee jerk.

Dysdiadochokinesis.—This is a clumsiness and slowness in the performance of rapidly alternating movements (pronation-supination), although the single movement can be normally performed. In carrying out this test, it is common to see adventitious movements of the limb occur, the whole limb being in movement.

Dysmetria.—When the patient is asked to extend the arm to pick up some object, such as a glass, the limb is shot forwards with undue haste and force and may overshoot the mark.

Tremor.—This is not a resting tremor, but an unsteadiness which develops

during movement, and in purposive movements tends to increase in range and severity as the climax of the movement is reached. It is an "intention tremor." Similarly, if the arms be horizontally extended, they may show a tendency to droop, which is corrected by a series of jerks which thus gives the form of a tremor.

Excessive rebound.—This diagnostically valuable sign may be present when more striking components of the cerebellar syndrome are absent. If the arms be horizontally extended by the patient, and the observer smartly strikes them downwards somewhere in the region of the hand, the arm on the normal side is quickly brought to rest in its original position with a minimum of recoil. On the side of the lesion, however, the hand and arm "bounce" freely, and may swing two or three times before being brought to rest.

Gait.—In bilateral lesion the gait has a reeling, tottering character, and in strictly unilateral lesions there may be a tendency to sway and deviate to the side of the lesion. The disorder may vary in severity from a slight unsteadiness to a complete inability to stand or walk unassisted. There is a tendency to walk with the legs abnormally separated, to raise the legs unduly, and to stamp them down heavily (manifestations of dysmetria).

Numerous other defects may be elicited by special tests, but one only calls for special mention, that is the defective movement of the articulatory musculature in speech. The defect is known as "scanning" or *staccato* speech. It consists in a slowness of articulation, and a tendency to say each syllable of a word as though it were a separate word. In polysyllabic words the syllables are not normally run together.

The cerebellum forms part of the non-sensory afferent nervous system. It is not a sensory organ and there is no disturbance of any form of sensibility in cerebellar lesions (Holmes).

THE ANTERIOR FOSSA OF THE SKULL

The lesion commonly found in this region is meningioma arising from the olfactory groove. The signs are unilateral anosmia from pressure upon the olfactory bulb and tract; primary optic atrophy and visual loss on the side on which the tumour develops; and crossed papilloedema as an expression of the general rise of intracranial tension. Aneurysm of the anterior cerebral artery may give rise to a similar syndrome, papilloedema in the crossed eye being absent.

THE MIDDLE FOSSA OF THE SKULL

A rich variety of lesions may arise in or invade this fossa, and the syndromes also vary according to the situation, mesial or lateral, of the lesion.

The lesions in the midline include pituitary adenomata, tumours of the pituitary stalk, and meningioma of the sellar diaphragm. In the lateral parts of the fossa, passing from the mesial to the lateral extremity, we have to consider cerebral aneurysm, meningioma arising from the sphenoidal ridge, and growths invading the base of the skull and either occluding its foramina and thus producing cranial nerve palsies, or actually invading the

cranial cavity. Epithelioma of the naso-pharynx is the common lesion of the last-named type.

(i) *Region of the optic chiasma and the pituitary body.*—The most common lesion in this region is pituitary tumour, which involves the optic chiasma, at first in the middle line posteriorly, and subsequently advancing forwards. Three sets of symptoms are likely to arise: (1) Those due to dyspituitarism, such as acromegaly or gigantism if there is hyperpituitarism, or Fröhlich's dystrophia adiposo-genitalis, or Lorain infantilism if there is hypopituitarism. Adenomata of the pituitary body produce hyperpituitarism if they contain eosinophil cells, and hypopituitarism if such cells are absent. (2) Those due to the pressure upon the optic chiasma, which commence as bitemporal paracentral scotomata, which enlarge as the compression extends until a complete bitemporal hemianopia results. It cannot be too strongly impressed upon the reader that the pattern of the visual field defect is determined by the position of local pressure upon the visual paths, and that any variety of defective field may occur. While bitemporal loss is the most usual, yet when the pressure is far forward, unocular hemianopia, blindness of one eye, and central scotoma are all of common occurrence, and when the pressure is farther back than usual homonymous hemianopia is frequently seen. And (3) those due to the general effect of the tumour, namely, headache and vomiting. Optic atrophy is the rule, as the result of the direct pressure, and not papilloedema. It is to be remembered, that all pituitary cases are prone to headaches and subject to fits. We have seen also (p. 1593) that cerebral aneurysm may be productive of this syndrome, or at least of the chiasmal component thereof.

(ii) *Lateral region of the middle fossa; syndromes of the sphenoidal ridge.*—The dural sinus which runs along the sphenoidal ridge (sinus sphenoparietalis) is one of the sites of election of the development of meningioma. From the point of view of localising diagnosis this ridge may be divided into three parts, namely, outer, middle and inner (or clinoidal).

A meningioma arising from the *outer end* of the ridge may produce as its localising syndrome unilateral exophthalmos without squint, some fullness of the temporal fossa with local tenderness on pressure, together with the general signs of raised intracranial tension.

Meningioma of the *middle part of the ridge* may remain for long without clear localising indications, and radiography (including ventriculography) may be necessary. Meningioma of the *inner or clinoidal part of the ridge* commonly gives rise to a striking and characteristic syndrome which in addition to the general signs of raised intracranial tension, unilateral failure of vision due to primary optic atrophy, unilateral exophthalmos, crossed papilloedema, more or less complete ophthalmoplegia on the side of the lesion and finally symptoms referable to pressure on the temporo-sphenoidal lobe (uncinate fits, hemiparesis, personality changes). It will be remembered that internal carotid aneurysms also arise in this neighbourhood and may produce a similar symptom-complex, papilloedema, however, being commonly absent. The syndrome of naso-pharyngeal tumour when the skull is invaded also closely resembles this. In other words, this syndrome, in partial or complete form, should arouse in the observer's mind the possibility of one or other of these three varieties of lesion—meningioma of the inner end of the ridge, cerebral aneurysm, and naso-pharyngeal tumour invading the skull.

THE POSTERIOR FOSSA OF THE SKULL: SYNDROME OF THE LATERAL RECESS

Lateral recess.—The angle formed by the posterior surface of the petrous bone and the tentorium is a common situation for neurofibromata which grow usually from the eighth nerve, but occasionally from the seventh and from the fifth nerve, and press into the lateral lobe of the cerebellum. A highly characteristic clinical picture results, of slowly oncoming nerve deafness, unilateral signs of cerebellar involvement and some peripheral facial spasm, to which are sometimes added facial weakness and tinnitus. Such tumours are not of great size, and therefore headache and papillœdema are often absent or occur late.

INTRACRANIAL TUMOURS

Under this heading are grouped all new formations which encroach upon the intracranial space, and which produce the familiar pressure symptoms and local symptoms of tumour, though some of these are not, strictly speaking, neoplasms.

Ætiology.—The brain is one of the commonest seats of new growth in the body. Further, new growth is one of the commonest forms of structural disease of the brain—vascular lesions naturally not being included under this heading. Thus, out of a total of 1309 patients admitted to the National Hospital in 1928, there were 163 cases of intracranial tumour, 132 of disseminated sclerosis, and 113 of neurosyphilis.

Age.—Cerebral tumour may occur at any age, but it is relatively uncommon in the very young and in the very old. It seems to be somewhat more common in the female sex. The relation between head injury and the first appearance of symptoms of cerebral tumour, is one which occurs much too often to be ignored, though it is likely, in some of the cases in which this relation exists, that the blow on the head has simply served to bring a pre-existing tumour into symptomatic prominence, either by causing œdema or hæmorrhage in its substance, or vicinity. It must be remembered in this connection that a cerebral tumour may exist for long periods without definite symptoms.

Pathology.—The pathological classification of intracranial tumours has a practical importance, for when the nature of a new growth can be determined clinically, some idea of its future behaviour can be formed, and the surgeon can make his plans to meet the special problems which each variety of tumour presents.

The chief varieties of intracranial tumour are as follows:

Tumour of the brain substance—Glioma.

Tumour arising in the meninges— $\left\{ \begin{array}{l} \text{Meningioma.} \\ \text{Auditory nerve tumour.} \end{array} \right.$

Tumour of the pituitary body and stalk.

Secondary carcinoma.

Infective granuloma—Tuberculoma, Syphiloma.

Blood vessel tumours.

Parasitic cysts.

It is not possible to indicate the relative incidence of all these different types of tumour, but it is possible to state that glioma constitutes 40 per cent. of all intracranial tumours, and meningiomas and pituitary tumours together from 20 to 30 per cent. Formerly, the incidence of secondary carcinoma was said to be about 6 per cent., but a recent estimate by Elkington places the figure at 20 per cent., and there can be no doubt that as greater precision in diagnosis is reached the frequency of this complication of visceral carcinoma will be more fully recognized.

As its name implies the *glioma* is a tumour arising in the glial or supporting tissue of the brain, but within the limits of this term are included growths of varied cytological type and modes of growth. Some are richly cellular, highly vascular, rapidly growing and fairly circumscribed tumours. To these the name *glioblastoma* is given. Others are diffuse infiltrating tumours, invisible to the naked eye except where degenerative processes have occurred in them, very extensive and often bilateral. To this type the name of *astrocytoma* is given. They are prone to cyst formation and grow more slowly than the glioblastoma. In childhood a variety of glioma known as the *medulloblastoma* is common. It is a richly cellular, highly malignant growth of the fourth ventricle.

Very many other types of glioma have been described, but these classifications are ephemeral and largely artificial, for each type may be represented within a single tumour, and changes from one type to another—from astrocytoma to glioblastoma—may occur in a given growth. Scherer has shown that none of these types is capable of extirpation, the astrocytoma because it is diffuse and largely invisible, the glioblastoma because death follows attempt to remove it. Thus, there is no such thing as a benign glioma. The best that can be said is that the astrocytoma recurs in some cases relatively slowly. Recurrence is invariable in all who survive partial extirpations. The glioma is not always a single tumour, contrary to what has been believed in the past.

Meningioma and auditory nerve tumours.—These tumours of the meningeal sheaths which insulate the ectodermal nervous system occur next in order of frequency to the gliomas. The meningioma, or endothelioma as it is sometimes called, grows from the endothelial cells of the arachnoid villi where these penetrate the walls of the dural venous sinuses. It is therefore found in the neighbourhood of the various sinuses, especially the superior longitudinal, the spheno-parietal and the petrosal sinuses. The meningioma does not invade the brain, but compresses and displaces it, and may become imbedded in it. It may also develop outwards and invade the skull, appearing externally as a rounded boss on the top of the head. The auditory nerve tumour may be single, or may appear as part of a generalised neurofibromatosis, in which case it may be bilateral on the eighth nerve. It grows in the lateral recess, where it compresses and stretches the fifth, seventh and eighth nerves, and also compresses and displaces the cerebellum and gives rise to internal hydrocephalus.

Secondary carcinoma is probably more common than is generally realised. It is a frequent event in pulmonary cancer, and the presence of a secondary involvement of the brain may first bring the patient to notice. Indeed, in all adult cases presenting the signs and symptoms of intracranial tumour the possibility of carcinomatous metastasis should be explored, especially

in a patient who is losing weight. Secondary carcinoma gives rise to numerous deposits in the brain, of various sizes, and may also produce a fine infiltration of the pia-arachnoid, with or without macroscopic masses in the brain. When this "meningitis carcinomatosa" occurs alone its diagnosis may be difficult. Secondary growths may follow primary carcinoma of the breast, stomach, uterus, or prostate, as well as of the lung.

Cholesteatomata.—Sometimes called "mother of pearl" tumours, on account of their glistening appearance, are found in connection with the basal meninges. Their origin is uncertain. They are either of slow growth, or run a symptomless course. They consist of a greasy, greyish, friable and more or less laminated mass, made up of layers of a closely packed mosaic of flat polygonal cells. The tissue is necrotic, and contains no blood.

Among the rarer tumours of the brain may be mentioned dermoid tumours, teratomata, chordomata, which arise from rests of the anterior end of the primitive notochord and are found below the base of the brain, lipomata, fibromata, neuromata, neuroblastomata, consisting actually of undifferentiated nerve cells, enchondromata, angiomas and psammomata.

Cysts.—Cysts of the following nature may occur—(1) Congenital interpeduncular or pituitary cysts, which arise from a pharyngeal rest in connection with the development of the pituitary gland; the resulting signs are those of pituitary insufficiency, together with those of pressure upon the optic chiasma. (2) Simple serous cysts, which are presumably the remains of soft tumours, which have become completely degenerated. (3) Tumours containing cysts, presumably on the way to the formation of the above. (4) Blood cysts, the rare results of hæmorrhage which has become arrested. (5) Cysts which result from softening after embolism and thrombosis.. When occurring in the young, these cysts may lose every trace of their original origin, and form thin-walled cavities, containing colourless fluid, often extending from the ependyma to the pia mater, and involving the whole thickness of the pallium. They are termed "porencephaly." (6) Cystic distension of the ventricles from obstruction, which forms local or general hydrocephaly. These are met with in connection with tumours in any situation and result from adhesive meningitis, particularly syphilitic meningitis. (7) Dermoid cysts. (8) Parasitic cysts, of which the more common is the bladder worm of the tapeworm, *Tænia solium*, which is called, on account of the thickness of its wall, *cysticercus cellulossæ*. They are usually multiple, and choose the region of the fourth ventricle as their site of predilection. They may be multiple in the basal meninges, and constitute a "cysticercus meningitis." It is usual for these cysts to shrink and to become calcified and obsolete in from 3 to 6 years. Less commonly, the hydatid of *Tænia echinococcus* is found. It is usually single, may reach a large size and present the signs of a slowly growing tumour with eosinophilia.

Infectious granulomata.—Tuberculomata are more common in the young; but they may occur at any age. They vary in size from that of a millet seed to that of a hen's egg, and are more often found in the posterior fossa of the skull than above the tentorium. When large, coagulation necrosis and caseation occur in the centre, and on section the tumour presents a dry yellowish crumbling or even diffuent centre, with a greyish-red peripheral

growing zone, where are located living tubercle bacilli and actively growing tubercles. The tuberculoma has an important aspect in connection with operation for extirpation. They are often situated favourably for extirpation, yet in every case where this operation has been performed the patient has succumbed to tuberculous meningitis, often after recovery from an apparently completely successful operation. Such a tumour recognised on decompression should be left severely alone.

Syphiloma.—This is not a common intracranial tumour. It grows most commonly from the meninges, and is therefore a surface lesion, though it may burrow deeply in the brain tissue. It is most commonly found above the tentorium. It is occasionally very hard in consistency, and tends in many cases to scar and become obsolete. It is sometimes impossible to distinguish this tumour from a tuberculoma without the aid of the microscope and the serum reaction.

Actinomycomata and tumours from streptothrix infection occur in very rare cases.

Symptoms.—The rates of growth of the different kinds of tumour vary widely. Some cases run their course from onset of symptoms to fatal termination within a few weeks, while in others there is evidence of gradual growth over a period of years. In the latter group it may be only in the final stage that the true nature of the illness becomes apparent, and only in retrospect that earlier symptoms assume their real significance. This perhaps is especially so in the case of those tumours which for months or years have manifested their presence only by generalised epileptiform fits. In yet other cases, an intracranial tumour may remain latent during life, being revealed unexpectedly at post-mortem examination.

Between these two extremes a great variety of symptom-complexes may be presented by an intracranial tumour. Thus, it may first show itself by producing signs of raised intracranial tension alone—that is, by general signs, or by signs of a gradually progressive local lesion alone—that is, by focal signs. Whichever of these two elements is initially lacking will probably appear later. A third manner in which a tumour may first signal its existence is—as has been mentioned—by the occurrence of generalised epileptiform fits in the absence of any other symptoms and signs. In this instance, also, general and focal signs will probably ultimately make their appearance. Again, a sudden onset of symptoms from hæmorrhage into a glioma, or from œdema of surrounding brain, may usher in the clinical course of a tumour within the skull.

The age of the patient is not without influence in determining the symptomatology and clinical course of a tumour. Thus, in childhood the early appearance of greatly raised intracranial tension—that is, of general symptoms, is the rule. This is mainly due to the fact that at this age the tumour is commonly in the fourth ventricle, and is thus favourably placed to produce internal hydrocephalus. In elderly persons, on the other hand, the picture of tumour is apt to be blurred, general signs are late in development, and focal signs are indistinct. Possibly the presence of a background of cerebral arterial degeneration and its associated cerebral change are responsible for this blurring of clinical outline. It may be supposed that the tumour does not write its mark upon a clean slate where there is arterial and cerebral degeneration already present.

GENERAL MANIFESTATIONS.—These symptoms are the result of raising of the intracranial pressure, and accordingly fail when there is no considerable raising. Therefore they tend to be absent in all tumours of the brain stem, in infiltrating tumours of the centrum ovale, and also in advanced age and in the subjects of marked arterial disease. They consist in the following signs: Papilloedema, headache, vomiting, loss of vivacity and mental drowsiness, nasal irritation, giddiness, alteration of pulse-rate, of blood pressure, respiration, and general convulsion.

Papilloedema.—This is by far the most constantly present of all the general manifestations. Papilloedema appears to be a stasis œdema of the nerve-head owing to the increased intracranial pressure forcing the cerebro-spinal fluid into the meningeal sheath which invests the optic nerve, and into the perivascular spaces which accompany the central vessels of the nerve. The nerve sheath becomes distended, and venous stasis occurs. On ophthalmoscopic examination the earliest changes are increased redness of the disk, distension of the veins, loss of distinctness of the nasal margin of the disk, with disappearance of the physiological pit. As the process increases the whole margin of the disk becomes lost. It enlarges in area, and becomes visibly swollen and presents the appearance of a mole-hill as seen from above. The point of emergence of the vessels, at the centre of the disk, becomes buried by white exudation, which occurs also all over the disk, and taking a form determined by the radiating nerve fibrils, gives the disk the appearance of being striated in a radial fashion, like a chrysanthemum. A similar exudate may rupture the membrana limitans interna in little droplets at the macula, and coagulating as it comes in contact with the vitreous humour, produce the characteristic radially arranged macular figure of "macular fan," exactly similar to that seen in renal disease. The venous congestion of the retina leads to multiple hæmorrhages, which infiltrate along the radially arranged nerve fibres, and for this reason are flame-shaped. With the outpouring of much exudation, the disk becomes white. In the course of time the hæmorrhages become white flame-shaped scars, the whole disk contracts, the swelling disappears, and the disk becomes white, flat and atrophic, and distinguished only from that of primary optic atrophy by the scarred remains of the exudate at its edge, producing a fluffy outline like that of torn cotton-wool, along the vessels and at the centre. In the early stages of papilloedema, even though there be considerable swelling of the disk, vision may be little impaired. As the process increases however, in proportion to the degree of the swelling to the amount of the exudate, and to the length of time the papilloedema has lasted in a severe condition, vision becomes impaired, and blindness results. Peripheral constriction of the visual fields, large pupil and dimness of vision, are the signs that, if the papilloedema be not speedily relieved, blindness will certainly result. Perfect vision may be retained for a time, even with a high degree of papilloedema. So important is papilloedema in the diagnosis of tumour of the brain, that it is necessary to bear constantly in mind all other causes which may give rise to it.

Papilloedema may occur in certain general intracranial conditions other than tumour. In meningitis it occurs as a late sign, and rarely before the tenth day, and as so many cases of meningitis do not survive so long, it is chiefly met with in the more chronic forms, such as tuberculous meningitis,

and untreated cases of meningococcal meningitis. Abscess may also cause papilloedema ; but it is by no means common in this condition.

Apart from intracranial disease papilloedema occurs in the following conditions : (1) Local conditions of the retina and optic nerves. In connection with tuberculoma of the retina in the neighbourhood of the disk, the most intense papilloedema may be found. Retrobulbar neuritis occurring close behind the disk may cause a similar condition, especially if it be of a syphilitic nature. In these conditions the papilloedema is often unilateral, but in any of them it may be bilateral. (2) Renal disease may give a retinal picture of intense papilloedema, macular figure and hæmorrhages, sometimes quite indistinguishable from that due to tumour. This is often seen in the small white kidney of young subjects, and sometimes in small red kidney, but there is no form of renal disease, even including tuberculous, amyloid and lardaceous kidney, in which papilloedema has not been observed. (3) Anæmic states of every kind sometimes give rise to papilloedema. As regards groups (2) and (3), it is essential to emphasise the facts that papilloedema, headache and vomiting may occur as a symptom-complex, both in renal disease and in anæmic states. (4) Septicæmic conditions, and especially those producing arthritis. Of these infective endocarditis is the most common ; but it has occurred with every form of septicæmic arthritis, and even in cases of acute rheumatism. (5) Further, papilloedema has been noted in connection with tumours, and with compressions and fracture-dislocations of the cervical cord, and also with acute myelitis.

The retinal changes in diabetes are always, and those in renal disease often, distinguishable from papilloedema resulting from increased intracranial pressure. In diabetes the change is essentially a hæmorrhagic retinitis from degeneration of vessels, sometimes with waxy-looking exudation in circinate patches ; and in renal disease it is often a general œdema of papilla and retina, with hæmorrhages and white patches far away from the disk. The papilloedema resulting from increased intracranial pressure is always bilateral, though it may appear in one eye before the other, unless there be local pressure upon one optic nerve, which always delays or prevents papilloedema appearing in that eye. Otherwise, an earlier commencement upon one side is of no localising value whatever.

Headache.—Although this symptom is a characteristic concomitant of raised intracranial pressure, it cannot be directly attributed to this, since a lumbar puncture which lowers this pressure may lead to increase of headache. It is probable that the pain arises from stimulation of sensory nerves in the walls of the cerebral arteries by changes in tension. The dura mater is probably not the sensitive structure responsible for headache in cases of intracranial tumour. After destruction of the fifth nerve by Gasserectomy, headache never again occurs upon that side. The sensation may vary from a mere feeling of fullness of the head to the most agonising pain. It is more often remittent than continuous, and may be absent for long periods together. It is rarely localised to any definite region, except when the growth actually involves the bone, or when pressure has caused local thinning of the bone, when local pain and tenderness on pressure may occur. Usually it is referred indefinitely to the frontal or to the occipital or to the vertical region. When occipital it may be associated with pain and stiffness of the neck, and head retraction. This is due to a general pressure effect, and does not indicate any

localisation. Headache may be entirely absent, even in the presence of severe papilloedema. It may precede the development of papilloedema, but more often it is later in its appearance.

Vomiting.—Only two-thirds of all cases of intracranial tumour present vomiting as a symptom. It rarely occurs in the absence of the two chief signs of increased intracranial pressure, papilloedema and headache. When the headaches are severe, it may be associated with much nausea, and the attacks are often referred to by the patient as "bilious attacks." Usually a result of increased pressure, it may be directly produced by lesions of the cerebellum, irritation of the vestibular nerve, and by the visual disorientation resulting from diplopia. As a symptom of intracranial tumour it hardly deserves the cardinal importance which has been assigned to it in most descriptions of this disease.

Loss of vivacity and mental drowsiness.—Even when intellectual capacity shows not the slightest impairment, there is from the first onset of symptoms a loss of vivacity, a slight heaviness and an absence of restlessness which is of value in diagnosis. It is almost unheard of for a tumour patient to suffer from insomnia. As the symptoms increase, so do heaviness and drowsiness, though a perfect but slow cerebration may persist until the latest stages of the disease.

Giddiness.—Though this is an inconstant sign, it is often met with, and it may be due to vestibular irritation, when it amounts to an actual vertigo, or it may be a sense of general unsteadiness. It is met with most often in sub-tentorial tumours, but may be quite a general symptom when very high pressure exists.

Convulsions.—As has been mentioned, generalised epileptiform fits, indistinguishable from those of idiopathic epilepsy, may usher in the clinical course of tumour, and may be present for long periods as the sole indication of tumour. The onset of such fits in an apparently healthy middle-aged individual, never before subject to them, should always give rise to the suspicion of tumour of the brain. When later on in the course of the illness general and focal signs of tumour appear, the convulsions may not increase in frequency, and it is not possible to regard them as a sign of raised intracranial tension. They more probably arise from local circulatory disturbances. Beyond saying that they are most frequently found in tumours of the cerebral hemispheres, no localising value can be attributed to them.

Blood-pressure, pulse-rate and respiration.—There is a slight compensatory increase of the blood-pressure for every raising of the intracranial pressure, so that the cerebral circulation may be kept going. The failure of such compensation is often the cause of the sudden death which occurs in tumour cases. The pulse-rate is in the inverse ratio of the blood pressure, and, therefore, of the intracranial pressure, and the pulse is slower than normal, where pressure is above normal. Respiration tends to be slow, and when the physiological condition of the intracranial contents is much disturbed, it tends to become irregular, grouped, and may show the wax and wane of movements which bears the name of Cheyne-Stokes respiration.

FOCAL SIGNS.—These have been fully described in the section upon the localisation of lesions of the brain. In connection with localisation, however, it is important to recognise certain possible sources of fallacy in making a diagnosis. Blindness from papilloedema prevents any localisation by means

of the visual functions. Papilloedema usually causes at one stage great peripheral constriction of the visual fields which might be attributed to a bilateral lesion of the cuneus, and it may cause altitudinal hemianopia, *i.e.* blindness of the upper half of both fields, by sagging of the exudation into the lower part of the retina. Jacksonian epilepsy may occur in long-standing cases without any relation to the position of the tumour.

Paralyses of cranial nerves may be serious pitfalls. They are of value in localisation when occurring early, and in association with alternate hemiplegias, and paralyses of the eighth, ninth, tenth, eleventh and twelfth are always of sure localising value. Paralysis of the sixth cranial nerve, perhaps, should always be disregarded as a localising sign for the following reasons: When the intracranial pressure increases from the presence of a growth, the first effect is that any superfluous cerebro-spinal fluid, of which there is normally very little, is expelled from the skull. Later, with further increasing pressure, since the only escape from the rigid skull is by its only opening, the foramen magnum, the medulla and cerebellum are pushed backwards towards the foramen magnum and come to fill up this aperture as with a cork. In all long-standing cases of increased pressure, the cerebellum will be found on autopsy, and especially when hardened *in situ*, to be deeply marked by the edge of the foramen, part of the cerebellum and medulla actually occupying the spinal canal. Corking up of the foramen magnum in this way offers a marked impediment to the flow of cerebro-spinal fluid, and is a most important factor in the production of hydrocephalus, secondary to tumour. Bearing this in mind the immediately fatal effects which have followed lumbar puncture in long standing cases of high intracranial pressure will be at once understood and for ever avoided. This shifting backwards of the medulla and cerebellum will cause stretching of those cranial nerves attached to the medulla, in proportion as they are directed antero-posteriorly, and take a straight course between their attachments to the dura mater and their origin from the medulla, and of these the sixth nerves will be most affected, and afterwards the third, seventh and fifth in that order. These nerves will not only be stretched, but are subject to the increased pressure also, and they may accordingly cease function simply as the result of the increased pressure. Special mention should be made of tumours of the pituitary body and stalk. In the previous section the localising signs of lesions in the region of the optic chiasma have been enumerated, but since the different varieties of tumour in this locality have their own typical symptom-complexes, the following table may be useful in differentiating them.

Diagnosis.—The differential diagnosis of intracranial tumour has to be made—(1) from other conditions causing papilloedema, (2) from other conditions causing headache, and (3) from other local lesions causing local signs within the brain. Renal disease, conditions of severe anæmia, encephalitis and meningitis may on occasion give rise to a combination of all three of these, very easily confused with the papilloedema, headache and vomiting of cerebral tumour.

Hydrocephalus is only distinguishable from intracranial tumour by the enlargement of the head which takes place in young subjects, but when the skull is rigidly closed, the symptoms are identical with those of a non-localisable tumour.

DIFFERENTIAL DIAGNOSIS OF TUMOURS IN THE PITUITARY REGION
(WALSHE)

	ADENOMA.			PITUITARY STALK TUMOUR.	MENINGIOMA.	GLIOMA OF OPTIC CHIASMA. (rare)
	Chromophobe.	Chromophile.	Mixed Cell.			
Age Incidence.	From adolescence onwards.			From 10 years to early adult life.	From 30 years onwards.	Usually in childhood.
Fundus Oculi.	Primary optic atrophy.			Papilloedema in children; usually primary optic atrophy in adults.	Primary optic atrophy.	Primary optic atrophy.
Visual Fields.	Bitemporal hemianopia.			Bitemporal hemianopia.	Bitemporal hemianopia.	Bitemporal hemianopia, proceeding to early blindness.
	(—Occasionally homonymous hemianopia—)					
Pressure Symptoms.	Absent, or late.			Early and severe, except in adults.	Absent, or late.	Absent, or late.
Glandular Symptoms.	Hypopituitarism.	Hypertrophic or Hypopituit.	Mixed.	Hypopituitarism.	Nil.	Nil.
Situation.	Sellar.			Suprasellar.	Suprasellar.	Suprasellar.
Radiological.	General enlargement and deepening of sella.			Shadows above and in sella. Sella shallow, and with uneven floor.	Commonly no change.	Enlargement of sella forwards beneath ant. clinoid processes.

Intracranial abscess is not often confused with tumour when it has an obvious cause in the vicinity of the brain, from bone disease, or an embolic cause at a distance, such as ulceration of the lung. It is an acute disease and rarely develops an increasing papilloedema. Accuracy in the early diagnosis of tumour cases depends upon the pertinacity with which every case of headache, every case of "fits" and indeed every case which shows any nervous symptom whatsoever, is systematically examined for signs of organic disease, and importantly upon that skill and practice with the ophthalmoscope which is so easily acquired with patience and a little determination. The presence of a tumour having been determined, the necessity is to localise it.

Above the tentorium tumours may be difficult or impossible to localise. So far as decompression is concerned the least indication, however slight, should determine the position of decompression. The external surface of the head should be carefully examined, and especially after it has been shaved, for now and then important indications of the position of a tumour may be afforded, for tumours may grow from the bone, or when internal may cause local absorption of the bone, and bulging of the skull. X-Ray examination should not be omitted, though it does not often afford important information. Tapping of the lateral ventricles, with analysis of their content as to protein concentration, and the introduction of air into the ventricles, with subsequent radiogram, and especially the injection of a thorium salt into the internal carotid artery in the neck, with immediate radio-instantogram, which shows the cerebral arteries and points out any region evascularised by local pressure, are all methods of value. Ventriculography is dangerous, and should only be performed when immediate decompression is practicable if found to be necessary.

The determination of the nature of a growth may be difficult or impossible, and length of clinical history may be a fallacious guide, since a slow-growing tumour may be long latent, and sudden in its production of symptoms. It may be recalled, however, that in childhood medulloblastoma is the most frequently occurring of all intracranial tumours, and that it has a very characteristic picture: headache, vomiting, papilloedema, bilateral external rectus palsy and unsteadiness of gait. Again, a well-marked picture of a progressive frontal lobe lesion generally indicates the presence of a glioma, and the same may be said of the symptom-complex of a temporo-sphenoidal lobe lesion. But there can rarely be any certainty as to the pathological nature of a tumour, even when it is a secondary carcinoma, since the primary growth may be latent and unsuspected.

It is important to remember that the finding of a positive Wassermann reaction in the serum of a patient presenting signs of intracranial tumour does not necessarily—or probably—indicate that the growth is a gumma. Both syphilis and intracranial tumour are common diseases, and their occasional association is less rare than cerebral gumma.

Course and Prognosis.—An intracranial tumour usually causes increasing symptoms, which progress with exacerbations and remissions, until papilloedema ends in blindness, and until the pathological intracranial condition becomes incompatible with even vegetative existence. At any time death may occur from vascular lesions, acute cedema or sudden raising of pressure. Tumours occasionally become obsolete—thus a tuberculoma may become scarred and calcified, and a glioma may become calcified or cystic; but this result is too rare for consideration within the grounds of practical perspective. The average duration rarely exceeds a year after the diagnosis has become possible.

Treatment.—The natural termination of a case of intracranial tumour is death, and the ideal of treatment must be the successful removal of the growth. Failing this, and it is frequently impossible, all that can be hoped for is the relief of headache and sickness, and delaying of blindness.

In respect of the radical, surgical treatment of tumours, it will be remembered that probably more than half (if we include glioma and secondary carcinoma) are in the brain substance, and can be extirpated only by

mutilating operations, which may leave in their wake grave physical and mental disabilities. The success of such an extirpation cannot, therefore, be adequately expressed in terms of "survival period"—as it is not infrequently assessed—but rather in terms of the kind of existence which is prolonged. This may be purely vegetative, and distressing to the patient and his relatives alike. We may say, then, that the treatment of the gliomas is, and must of necessity always remain, the forlorn hope of surgery. On the other hand, signal successes have been obtained in the case of the meningioma, the auditory nerve tumour, the pituitary adenoma, and a few cystic astrocytomas (particularly of the cerebellum).

It will be seen, therefore, how important it is to be able with some precision to determine the type of tumour present in any given case. When this is not possible, an exploratory operation is often justified. But it would be a mistake to suppose that surgical intervention is a matter of routine in every case in which intracranial tumour is diagnosed. Each case must be considered on its merits.

Failing the possibility of a successful removal, the palliative operation of decompression may be needed to relieve the symptoms caused by raised intracranial tension. This consists in the free removal of bone, and the incising of the dura mater, over the region of the tumour when this is known, or, failing localisation, in the right subtemporal region. For brain-stem tumours, decompression is not only useless, but also dangerous.

Relief of pressure by dehydration.—There are circumstances in which it may be desirable and necessary to reduce the brain volume and the intracranial pressure; for example, to relieve pressure headache, to avert impending coma or death, to render the patient capable of co-operating in his examination and thus facilitating a localising diagnosis, and finally to make surgical procedures more easy. Weed and M'Kibben have shown that the foregoing may be done by administering hypertonic solutions. In the ordinary case, the rectal injection of from 2 to 3 ounces of magnesium sulphate dissolved in 8 ounces of water may be tried. But for a very rapid effect, intravenous injection of from 50 to 75 c.c. of a 50 per cent. solution of dextrose, or of a 15 per cent. solution of sodium chloride, is effective. Pain and vomiting may be relieved with the various analgesics of the coal-tar series. When intracranial pressure becomes so high as to cause agonising pain, pulselessness and impending death, morphine in full doses will always relieve, and it is not dangerous. Convulsions should be combated with administration of bromides.

HYDROCEPHALUS

Definition.—The term "hydrocephalus" denotes a uniform distension of the ventricular system of the brain by the accumulation of cerebro-spinal fluid within it; and this distension is associated, sooner or later, with an expansion of the cranial bones and enlargement of the skull.

Hydrocephalus was formerly divided into acute and chronic, acute being applied to the condition of tuberculous meningitis. But since any marked degree of ventricular distension is unusual in that affection, and enlargement of the head very rarely occurs, this term has fallen out of use. In the majority of cases in which general atrophy of the cerebral tissues

occurs, fluid accumulates both in the ventricles and in the sub-arachnoid space; but such compensatory enlargement is not to be regarded as, in any sense, of the same nature as true hydrocephalus. Such accumulation of fluid is found in cases of cerebral diplegia and general paralysis of the insane in children, and it also occurs in the brains of old people. It is merely the result of wasting and shrinkage of the brain-tissue, and the accumulation of fluid takes place in order to fill up the space which is vacated within the rigid skull.

The enlargement of the head, which is not uncommonly found in rickets, has no connection with hydrocephalus. It is probably the result of mal-nutrition of cranial bones, which grow irregularly, and, being unduly soft, yield somewhat to the intracranial pressure. In rare cases of moderate degree, ventricular distension has been met with, but the enlargement of the head is never progressive, and the symptoms of hydrocephalus are absent.

According to their clinical aspect, cases of hydrocephalus may be placed in one of three groups—(1) congenital hydrocephalus, in which the enlargement of the head is present at the time of birth; (2) acquired primary hydrocephalus, which may appear at any period of life; and (3) secondary hydrocephalus. Under the name of secondary hydrocephalus may be grouped together all cases in which there is obstruction in the usual path by which the cerebro-spinal fluid leaves the ventricular cavities, or to the venous outflow from the choroid plexuses. But it is by no means clear that such obstruction is the sole or even the important agent in producing the ventricular distension.

Ætiology.—Hereditary influences are of importance in the causation of congenital hydrocephalus. This disease frequently affects several children of the same parents, and it may even appear as a striking family disease, affecting members of several generations of the same stock. Spina bifida, meningocele and hydromyelia are of frequent occurrence in association with this disease, and arrested and irregular development of the brain stem and cerebellum are the rule. Among other bodily deformities not infrequently associated with congenital hydrocephalus, may be mentioned harelip, cleft palate, talipes, rectal and testicular ectopia and imperforate anus. In a few cases definitely syphilitic lesions of the ependyma in the region of the brain stem have been found. The causation of primary hydrocephalus occurring after the time of birth is often obscure. The majority of the cases occur in childhood, yet no period of life seems to be exempt. In children, acute infective diseases, and especially gastro-intestinal infections, may occur as antecedents of hydrocephalus. In adults, syphilis stands in important relation in certain cases, some of which have been examined pathologically.

The causes of secondary hydrocephalus are, first, the sclerosing forms of meningitis, especially posterior basic and epidemic meningitis, very rarely tubercular; secondly, intracranial neoplasms encroaching upon the ventricular system, especially tumours of the brain stem and subtentorial region; thirdly, adhesive phlebitis of the cerebral blood-sinuses.

Pathology.—Hydrocephalus is directly due to an excess of cerebro-spinal fluid present within the ventricular system of the brain. The fluid is normally secreted by the choroid plexuses of the ventricles, which overflow into the subarachnoid space through the foramina of Luschka and Majendie in the roof of the fourth ventricle. It then fills the basal cisterns

and passing forwards between the tentorium cerebelli and the brain-stem flows up over the cerebral hemispheres. It leaves the subarachnoid space through the arachnoid villi, which pierce the walls of the dural venous sinuses and discharge their contents into the venous blood. Some of the fluid passes downwards into the spinal subarachnoid space, but its absorption is probably wholly within the skull by the channels mentioned. Obstruction to the absorption of the cerebrospinal fluid into the blood stream is the essential factor. This obstruction may arise at various points. There may be congenital atresia of the aqueduct of Sylvius which traverses the brain stem and joins the third and fourth ventricles. It may arise from occlusion of the foramina of Luschka and Majendie by adhesions resulting from an old acute lepto-meningitis. It will be apparent from these considerations that congenital hydrocephalus must be due either to atresia of the aqueduct, or to adhesions blocking the exit of the fluid from the ventricular system, these adhesions being the result of some pre-natal inflammatory process. Acquired hydrocephalus may follow an acute lepto-meningitis which has been productive of adhesions at either, or both, of the situations named above. It may also follow some other mode of blocking of the exits from the ventricular system. Such blocking is commonly produced by an intracranial tumour. The distension of the ventricles which necessarily ensues upon defective absorption of cerebro-spinal fluid from any of these causes is naturally maximal in the infant or child in whom the state of the skull vault allows of expansion of the intracranial space and of an enormous degree of stretching of the cerebral walls of the ventricles, especially of the lateral ventricles.

The quantity of fluid which is found in the ventricles after death varies greatly, a usual quantity being from 15 to 20 ounces. In long-standing cases with great cranial enlargement, very large quantities have been met with. The characters of the fluid do not differ greatly from those of normal cerebro-spinal fluid. Its density varies from 1008 to 1010. It is clear, colourless or slightly yellowish, and the reaction is alkaline. It contains a very small quantity of albumin and a comparatively large quantity of alkaline chlorides.

The dilatation of the lateral ventricles is always more extensive than that of the third ventricle, and is usually symmetrical upon the two sides, and it affects the body of the ventricle more than the cornua, so that the central cortex is the most thinned. The foramina of Monro are greatly enlarged and the anterior pillars wasted.

The convolutions are flattened, and the sulci indistinct. The thickness of the cerebral substance is much reduced. In advanced cases, the cerebral hemispheres have the appearance of a thin-walled sac, which collapses entirely when the contained fluid is allowed to escape. In a few cases, the aqueduct has been found closed, as if by antecedent adhesive ependymitis.

Symptoms.—The clinical manifestations of hydrocephalus fall into two groups, which result, respectively, from the effects of the abnormal intracranial pressure, first upon the brain-case, and secondly upon the nervous structures. In the congenital form, the enlargement of the head is the first noticeable feature; and this is true also of some cases of acquired hydrocephalus in young children. In most cases of acquired hydrocephalus, on the other hand, the nervous symptoms are first in evidence—namely, per-

sistent headache, vomiting, mental impairment, convulsions and sometimes papilloedema. The evidence of cranial enlargement may succeed these symptoms, and the older the subject, and consequently the more resistant the cranial walls, the more severe are the nervous symptoms, and the later is the cranial enlargement in appearing. In some cases of congenital hydrocephalus, enlargement of the head takes place during intra-uterine life, and it may be so great as to make delivery impossible without destruction of the head. More frequently, the cranial enlargement, not noted at the time of birth, becomes evident during the first few weeks of life.

Enlargement of the head is the most striking feature of hydrocephalus in children. The increase usually affects all the diameters of the cranial cavity, and is most marked on the vertex and least at the base. Trousseau compared the opening out of the cranial bones, which occurs as the head enlarges, to falling back of the petals of an opening flower. The forehead is large, rounded, and projects forwards; the temporal fossæ are obliterated, and the parietal eminences carried backwards. The vertex is often somewhat flattened, as also may be the occipital region. The direction of the external auditory meatus alters with the increasing size of the head; normally directed obliquely forwards, it comes to look directly inwards, or even obliquely backwards in severe cases. The head is frequently asymmetrical. In young children the sutures may be widely open, and then there is marked bulging along those lines and at the fontanelles. The skull may attain enormous dimensions, and many examples are recorded in which the circumference has been from 60 to 90 cm. The face is characteristically triangular, contrasting markedly with the forehead. Wasting of the facial subcutaneous tissues and retarded development of the maxilla and mandible often render this contrast still more striking. Bulging of the orbital plates of the frontal bone presses down the eyeballs, so that the pupils become more or less covered by the lower lids, and a band of the sclerotic may be visible between the iris and the upper lid. The hydrocephalic child often uses his hands to depress the cheeks, and so draw down the lower lids out of the position in which they impair the line of vision. The hair of the head becomes scanty, the subcutaneous veins of the scalp are often greatly developed and distended, and sometimes a vortex of distended veins radiates from the region of the anterior fontanelle. The general nutrition is poor, and bodily development retarded, in proportion to the severity of the effect of the intracranial pressure upon the nervous system. Auscultation may reveal a cephalic bruit, but this is neither a characteristic nor a constant sign in hydrocephalus, for it is frequently met with in rickety children, and may be present in a normal subject.

The nervous disorders which appear during the course of hydrocephalus are both variable and inconstant, and acute symptoms are of rare occurrence if the disease appears at an age at which the skull is still yielding. On the other hand, if the ventricular distension commences when the growth and ossification of the skull are complete, the nervous symptoms which arise are very severe, and resemble closely the general effects of intracranial growths. In secondary hydrocephalus, the symptoms due to this condition emerge from those of the preceding meningitis or sinus thrombosis, or are blended with those of the coexisting intracranial growth.

In children, the nervous symptoms of hydrocephalus, whether it be congenital or acquired, may be summed up in the following list, the symptoms

being frequent in the order in which they are enumerated; convulsion, mental failure, spastic paralysis of the limbs, headache, optic atrophy, nystagmus, vomiting, papilloedema. There is no constancy in the occurrence of these symptoms. Convulsion may not occur at all, and mental acuity may be unimpaired. Spastic weakness occurs in less than one-half of the cases, whilst optic atrophy is met with still more rarely, and papilloedema is distinctly unusual.

Convulsion.—While it is to be borne in mind that the whole course of hydrocephalus in children may run without the occurrence of convulsion, yet in the majority of cases this symptom is conspicuous. In some of the post-natal cases the symptoms of cerebral disorder are ushered in by convulsion, and it is probable that such convulsions are the immediate expressions of the morbid process, of which the primary hydrocephalus is the final result. The convulsions which recur at intervals throughout the course of the majority of cases of hydrocephalus result from a condition of functional instability of the cerebral cortex, which long-continued increased intracranial pressure brings about. The convulsions are usually general, with loss of consciousness.

All degrees of mental reduction occur, from the least noticeable to complete idiocy. The more severe forms of mental impairment are met with in congenital cases, and especially when cerebral agenesis, porencephaly and teratological defects are associated. The psychical reduction is less prominent the greater the age at which the symptoms commence, and, as a rule, the intelligence is far greater than the severity of symptoms (cranial enlargement, paresis, etc.) might lead one to expect. Cerebration is usually slow and the disposition placid, and periods of somnolence are of common occurrence.

The effect of long-continued ventricular distension in many cases is to cause degeneration of the pyramidal system, and, according to its degree, the latter entails bilateral spastic paralysis with contracture. The first signs of the onset of this event are exaggeration of the deep reflexes, and the change in type of the plantar reflexes from the flexor to the extensor response. The lower extremities are affected earlier and to a greater extent than are the upper, and at one period of the disease a case may present the picture of cerebral paraplegic rigidity comparable with that of Little's disease. The upper extremities are affected later. The paresis of the limbs is almost always symmetrical and equal upon the two sides. Sensibility is generally normal.

Vision is interfered with in a considerable proportion of the cases. The enlargement of the infundibular portion of the third ventricle, by pressure upon the inner borders of the converging optic tracts, may cause bitemporal hemianopia with atrophy of the nasal portions of both optic disks, this condition subsequently progressing to complete blindness and complete optic atrophy. More often the increased intracranial pressure causes atrophy of the optic tracts and secondary atrophy of the optic disks.

In other cases, optic atrophy is the result of papilloedema. In late childhood and in adult life papilloedema is the rule, and optic atrophy seems always to be consecutive to this. Strabismus is commonly present in congenital cases, and it is most frequently convergent. Nystagmus is met with in the subjects of hydrocephalus who are blind from optic atrophy, and it is of

frequent occurrence in long-standing cases in which spastic paresis is well-marked.

Headache is often complained of, and especially during the early days of illness in acquired cases, but this symptom never dominates the clinical picture in children, and is never so severe and persistent as that arising from the presence of an intracranial growth. Cerebral vomiting is of comparatively rare occurrence.

When one considers the profound anatomical alterations which take place in the advanced stages of the disease, the occurrence in some cases of unusual symptoms indicative of interference with the functions of the cerebellum, brain stem and cranial nerves is easily explicable. Unilateral or bilateral ataxy, vertigo, deafness, anosmia and paralysis of cranial nerves, are the most important of such unusual symptoms.

The signs of failure of the nervous system as a whole usher in the fatal result in severe cases. For some days or perhaps weeks before death, hebetude may become profound; spastic paresis gives place to flaccid paralysis with muscular wasting, the deep reflexes disappear, and the sphincter mechanism loses its control and subsequently its tone.

Hydrocephalus which commences in late childhood or in adult life presents an aspect widely different from that just described. At these periods of life, the bones of the skull are firm and resistant, and the sutures resist for a long time before yielding to the increased intracranial pressure. The general symptoms are acute, and the course of the disease is often rapid to a fatal termination. There is usually no enlargement of the head to aid the diagnosis, and the symptoms—headache, vomiting and papilloedema—resemble those of a non-localisable intracranial growth.

The headache is severe and usually paroxysmal, and it may be so intense as to cause sudden death, while, not infrequently, the sufferer loses all control during the paroxysms. Speaking generally, the headache is of much greater severity in adult hydrocephalus than in intracranial growth. Similarly, vomiting is apt to be more severe and persistent than that associated with cerebral growth. In many of the cases, a fatal result occurs before enlargement of the head, and before cerebral degeneration has produced further signs of spastic paresis than an increase of the deep reflexes, foot clonus and the change of the plantar reflexes to the extensor type. General convulsions and attacks of coma are not rare.

Diagnosis.—Where enlargement of the head is manifest the diagnosis of the disease presents no difficulties. The large head of rickets is easily distinguishable from hydrocephalus by its different conformation, by the association of the other signs of rickets, by the absence of nervous symptoms, by its non-progressive nature, and by the results of anti-rachitic treatment. The distinction between the primary and the secondary forms of hydrocephalus in children should present no difficulty, if a correct history of the early symptoms can be obtained. The initial manifestations in the primary form are slight, and cannot be confused with those of meningitis or of sinus thrombosis. Intracranial growths which cause early and marked hydrocephalus are situated in some part of the brain-stem from the third ventricle to the medulla, and growth in such a position must of necessity produce such early pathognomonic localising signs as to leave no excuse for erroneous diagnosis, save imperfect observation.

In adults the absence of cranial enlargement in most of the cases make it impossible to separate the malady with certainty from intracranial growths. It must be borne in mind, however, that headache, vomiting and papilloedema of rapid progress are not necessarily signs of intracranial growth, but may be the symptoms of primary hydrocephalus.

Prognosis.—This depends upon the cause of the hydrocephalus, upon the degree of severity of the symptoms, and upon whether it is progressive or not. In all severe and progressive cases the prognosis is hopeless, and the same is true of hydrocephalus secondary to inoperable neoplasm. In some of the slighter cases, both of the congenital and of the acquired form, the process becomes arrested, and the patient may attain to adult life with the possession of all his faculties. In cases in which the disease becomes stationary, the prognosis as regards mental capacity and the continuance of recurring convulsion has to be considered. If the mental capacity at the time of arrest is fair, it is not likely to deteriorate further, unless epilepsy is established. When mental reduction is marked at the time of arrest, a great degree of improvement cannot be reasonably expected.

Treatment.—While some cases of hydrocephalus cease to progress, and the symptoms disappear permanently under medical treatment, a like result has occurred in cases in which no treatment has been applied.

The importance of syphilis in the ætiology of hydrocephalus suggests the employment of anti-syphilitic treatment.

The results of surgical interference for the relief of pressure and to attempt the re-establishment of a way out for the cerebro-spinal fluid have been, up to the present, so unfavourable, that many writers and authorities consider such measures unjustifiable. It must be borne in mind, however, that in severe and progressive cases one is dealing with a necessarily fatal malady, and a few encouraging results have been published, which appear to justify further investigation. Paracentesis of the ventricle is both useless and dangerous, for when relief follows the operation it is only temporary, and where cerebral tension is very high an immediately fatal result may supervene. Repeated lumbar puncture is advisable in the earlier days; but this is only possible in cases in which the theca is in free communication with the ventricular space.

ENCEPHALITIS

Acute inflammation of the brain occurs under widely different clinical associations. It may occur as a primary disease or as a complication of known infective processes, affecting the system locally, generally, or as an associated event in diseases of the meninges. As a primary condition it is met with in the form of lethargic encephalitis. It is the constant result of trauma to the skull, if this be sufficiently severe. It is found as the result of infection of the brain with pyogenic organisms, either from local sources in the neighbourhood of the brain (septic bone disease), or from pyæmia, and may be then either suppurative (brain abscess) or non-suppurative. Infections by many of the specific fevers may cause it, and especially measles and scarlet fever. Acute encephalitis may occur in rare cases as the sole manifestation of cerebral syphilis. In all forms of meningitis there is some degree of extension of the inflammation into the brain tissue, and this

assumes an important degree in tuberculous meningitis, and sometimes in epidemic meningitis. The symptoms common to all forms of encephalitis are the general symptoms of severe intracranial disease—headache, somnolence, coma, irritability, convulsions, delirium and vomiting; and, in addition, local symptoms of irritation and paralysis, which are determined by the position and extent of the lesions.

1. SUPPURATIVE ENCEPHALITIS

Synonym.—Intracranial abscess.

Ætiology.—Suppuration within the brain substance is never primary, but the result of extension of infection from neighbouring tissues or by the blood stream from foci of infection in distant organs. In rare cases, the focus of original infection is undiscoverable.

The following are the important causal factors :

1. *Trauma.*—In the case of penetrating wounds the missile may be the source of the infection. Lacerated wound with fracture may allow of infection from the surface or from the middle ear, nose or pharynx. In these cases, meningitis often occurs in addition to abscess. Though not traumatic in a strict sense, any local lesion of the brain may become a locus resistentiæ minoris for the settling down of suppurative organisms derived from the blood stream, and in this way abscess has followed upon vascular lesions and the lesions of primary encephalitis.

2. *Extension from infected regions* in the immediate vicinity. The important cause of infection is any form of infective disease in the bones or soft tissues of the skull, calvarium and surrounding regions. Caries of the petrous bone from middle ear disease is the most common cause, while septic conditions of the nasal cavities and their accessory sinuses, or of any of the bones of the skull, suppuration of the scalp, orbital cellulitis and carbuncle of the neck are other causes. The exact manner of advent of the infection into the brain substance may differ in different cases. It may be by a septic thrombosis of a vein communicating between the infected region and the brain, or by extension along lymphatics similarly communicating, or by direct extension, as when the temporal lobe becomes adherent to the tegmen tympani, or it may be trans-meningeal by the direct transference of organisms across the meningeal space, without general meningitis occurring. That this latter mode of infection is a common one is suggested by the facts, that when the primary disease affects the upper surface of the petrous bone, the abscess is in the temporal lobe, and when the posterior aspect of the temporal bone is affected the abscess is in the cerebellum; and, most importantly, in all these cases of abscess from extension, the cerebro-spinal fluid shows the presence of polymorphonuclear leucocytes, thus showing that the meninges have been infected, although no symptoms of meningitis arise.

3. *Pyæmic states.*—Abscess of the brain does not often occur in symptomatic pyæmia. It may occur in infective endocarditis, and then multiple abscesses may be found. Sometimes in this condition multiple small spots of encephalitis, containing many polymorphonuclear cells but not definite abscesses, are met with. Much more commonly, abscess results from a single septic embolus from chronic pulmonary infection, such as bronchiectasis,

empyema and lung abscess. In rare cases metastatic abscess may arise from bone disease, liver abscess and in the course of specific fevers. The micro-organisms responsible for the infection are usually streptococcus, pneumococcus and staphylococcus, and often the infection is mixed. *B. coli* is sometimes found, and in rare cases streptothrix and oidium albicans.

Pathology.—The abscesses which result from local disease of the skull bones and surrounding tissues may be extradural, subdural or encephalic: in the first two cases they are invariably situated in the immediate vicinity of the antecedent seat of infection. The extradural abscess may reach a very considerable size and may burst externally, or into the meninges or into a blood sinus. The subdural abscess is confined in meningeal adhesions between the dura mater and pia mater. The contiguous surface of the brain is generally softened and has often disappeared, the abscess cavity extending deeply into the brain substance. This variety rarely has any capsule on the cerebral side. Encephalic abscess commences generally in the subsulcine white matter of the temporal lobe, and lateral lobe of the cerebellum. In one-half of all cases the abscess is in the temporal lobe, and in one-third in the lateral lobe of the cerebellum. The remainder are divided between the parietal lobe, the pons Varolii and the frontal lobe, in order of diminishing frequency. The size of the abscess varies up to that of a hen's egg. A recently formed abscess is irregular in shape with ill-defined limits, but in about 7 days it shows a definite capsule which may rapidly become of considerable thickness. The interior of the abscess cavity is usually of a greyish-green colour, and the pus is greenish and often fetid. The surrounding brain tissue is always cedematous and often softened. Rupture occurs in about one-sixth of all cases that are not afforded operative interference, and the rupture takes place most commonly into the ventricle and less frequently into the arachnoid space.

Symptoms.—An encephalic abscess has its origin in inflammation, and constitutes, when developed, a foreign body within the skull. Death may result from the effects of continually increasing intracranial pressure and wide interference with cerebral function, or from spread of the infection from the abscess. The symptoms may be grouped in four classes—(1) those of local suppuration; (2) those due to increased intracranial pressure; (3) localising signs dependent upon the position of the abscess; and (4) those of terminal extension of the infective process.

In extradural and subdural cases, the symptoms are generally acute and the course is rapid; the signs of pressure are severe, whilst localising signs are rare and a state of latency is not observed. In the majority of encephalic abscesses, on the contrary, the signs of initial suppuration are slight and are apt to be swamped by the symptoms of the preceding disease, otitis media, empyema, infective endocarditis, etc., and for this reason may be easily overlooked. A latent period in which symptoms are insignificant or completely absent may follow, and last for weeks or months. In the end, the latent period is broken, either by an acute outburst of symptoms, the result of extending infection, or the signs of progressive intracranial tumour arise.

The general symptoms which are likely to appear when a brain abscess is developing or emerging from a latent condition are pyrexia, which may be associated with rigor, headache, vomiting, irritability, vertigo, drowsiness deepening into coma, slowing of the pulse, respiratory and cardiac irregu-

larity, convulsions rarely, and papilloedema as a late sign. In addition, there is a leucocytosis of the polymorphonuclear variety in the blood, and a small number of polymorphonuclear leucocytes in the cerebro-spinal fluid in those cases arising by extension from disease of the cranial bones, but not in metastatic abscesses. The general symptoms vary much in their intensity and in the individual incidence of each of them, and in metastatic abscesses they may be almost absent, the local signs alone giving the indication that a cerebral lesion is present. Headache is rarely absent, and may be intense with spreading abscess. Vomiting is also an almost constant sign. Drowsiness is one of the most valuable of all the indications when any cause for the occurrence of cerebral abscess is present. Slowing of the pulse is also an important indication of a rising intracranial pressure. Papilloedema occurs late, and is often not present in acute abscesses at the time when diagnosis is all-important from a surgical point of view. It rarely occurs until an abscess has been present for a week, and is generally of low grade. With half-latent chronic abscesses, and with metastatic abscesses which attain a large size, it may be intense.

Local signs.—Generally speaking, the more recent and acute the abscess is the less definite are the local signs. In more chronic abscess, and in metastatic abscess, the local signs are usually more distinct. When there is a local cause for the abscess this constitutes an important localising sign, since abscess forms almost always in the immediate vicinity of site of infection. Thus rhinogenic abscesses are situated in the frontal region, and otitic abscesses are almost invariably situated either in the temporal lobe or in the lateral lobe of the cerebellum of the same side as the ear disease.

Metastatic brain abscesses may be situated anywhere, but they are more common in the region of distribution of the Sylvian artery, and in my experience have been more common in the posterior part of this supply—that is, in the parietal and occipital lobes. Metastatic abscess is sometimes preceded by definite indications of the embolism which gives rise to the abscess, such as local convulsion, local transient weakness or loss of consciousness, and such an event may precede the signs of abscess by many weeks. The local signs of lesion in the various regions of the brain are described in the section under that heading. Local diagnosis is often difficult owing to the condition of somnolence preventing the possibility of accurate examination. In these circumstances such slight signs as the absence of the abdominal reflex on one side, the presence of an extensor response in the plantar reflex of one side, or any aphasic signs, are important indications of temporal lesions; and unilateral hypotonia and nystagmus and attitudes, of cerebellar lesions. The initial signs and symptoms of encephalic abscess may lessen and disappear, and the abscess is said to become latent. The latency may be complete, or it may be broken by occasional headaches and transitory symptoms indicative of intracranial mischief. Much more commonly the abscess grows, and death occurs invariably in the absence of surgical interference, either from increasing intracranial pressure or from the rupture of the abscess, either into the ventricle, or on to the surface, with the production of general meningitis.

Diagnosis.—In those cases where there is no local cause for the formation of an intracranial abscess by direct extension, and no distant cause known or discoverable for the formation of a metastatic abscess, diagnosis is difficult,

and the distinction of an abscess from a tumour can hardly be made with certainty. The presence of pyrexia and of a polymorphonuclear leucocytosis in the blood may suggest the diagnosis in some cases. Where, however, the common antecedent causes of abscess exist in the form of ear disease, etc., or suppurative chronic lung disease, the diagnosis is relatively simple. For example, the advent of local or general intracranial signs in a case of chronic bronchiectasis from the first leaves no alternative diagnosis. When ear disease or local septic conditions of the region of the skull are present, local and general intracranial signs are due either to meningitis, abscess, sinus thrombosis, osteomyelitis of the base of the skull, or rarely to acute otitis.

Meningitis can be at once distinguished not only on account of the more irritative and rapidly oncoming symptoms, which differ somewhat from those of abscess, such as head retraction and rigidity of the neck, Kernig's sign, delirium and tremors, but by the lumbar puncture which gives the turbid cerebro-spinal fluid, containing polymorphonuclear cells and organisms in quantity. It must not be lost sight of that an abscess at any stage of its formation may be complicated by the development of general suppurative meningitis. Sinus thrombosis is usually accompanied by much oscillation of temperature, and by repeated rigors and oedema, and tenderness in the region of the emissary veins of the blocked sinus may be present. The diagnosis of the latter condition is not of vital importance, and its consideration should cause no delay in summoning the aid of the surgeon. The presence of any symptoms of intracranial disturbance, where tympanic septic disease exists, calls for immediate surgical interference, and the surgeon, after cleaning out the diseased tympanum, completes the diagnosis by examining the lateral sinus, both the temporal lobes and the lateral cerebellar hemisphere, and proceeds to those measures which the results of his exploration indicate. Acute otitis media may give rise to severe intracranial symptoms like those of meningitis, convulsions even occurring which may subside dramatically after perforation of the tympanic membrane, but it must be remembered that the chronic and not the acute forms of otitis give rise to septic extension to the brain. Osteomyelitis of the base of the skull extends from chronic bone disease in the region of the ear or nose. There is much pain in the base of the skull, and sometimes many cranial nerves are implicated at their foramina of exit. Skiagraphy will indicate the loss of bony structure. The malady is a chronic one, and usually ends in a terminal meningitis.

Prognosis.—Cerebral abscesses with very thick walls and inspissated or even calcified contents have been found post mortem, many years after the presumed time of formation of the abscess. Spontaneous evacuation of an abscess through the diseased ear, or through a sinus in the area of the local disease causing the abscess, has been followed by recovery. It is probable that no abscess becomes permanently quiescent after it has given rise to severe symptoms. The prognosis in cases of cerebral abscess, therefore, is that a fatal result will occur, unless successful surgical interference is possible. If the abscess is reached and drained, recovery often occurs rapidly, but this is never certain, for extensive perifocal softening, meningitis and sinus thrombosis may occur. Moreover, a general suppurative encephalitis may extend, in spite of draining the abscess.

Treatment.—The most rigorous prophylaxis should be employed that all patients suffering from septic nasal and ear diseases, and infective disease of the scalp and cranial bones, shall not pass out of observation until such disease is beyond all doubt cured. The only treatment for developed abscess is exploration and drainage. The liability to the occurrence of septic meningitis may perhaps be lessened by the administration of hexamine. The usual measures for the relief of pain should be employed.

2. LETHARGIC ENCEPHALITIS (see p. 1576)

MENINGITIS

Definition.—The inflammatory processes to which we apply the name of meningitis are infective in origin, and usually have their seat in the leptomeninges—the pia-arachnoid. A true inflammatory lesion of the dura mater, that is, pachymeningitis, is much less common, and is usually a localised process due to the direct spread of infection from adjacent bone.

Acute leptomeningitis, on the other hand, is usually generalised, and even when it arises from a local focus of infection it spreads rapidly throughout the subarachnoid space, this spread being facilitated by the cerebro-spinal fluid and also by the negligible bactericidal potency of this fluid. Further, the inflammation not only produces its characteristic changes in the pia-arachnoid, but also greatly changes the composition of the cerebro-spinal fluid. These changes may be said to reflect with considerable accuracy the nature and cause of the meningitis, and thus it is that the examination of this fluid has so great a diagnostic value. Acute leptomeningitis may be a primary infection, or may be secondary to some infective lesion elsewhere in the body.

Pachymeningitis may be cranial or spinal, and is usually secondary to either syphilis, tuberculous disease of bone, or middle-ear suppuration. The condition formerly known as “pachymeningitis interna hæmorrhagica” is now regarded as traumatic and not inflammatory in origin, and is described under the heading of chronic subdural hæmatoma (cf. p. 1593).

The fine infiltration of the pia-arachnoid by the cells of secondary carcinoma, of glioma, or sarcoma has been spoken of as a meningitis, but although such an infiltration may give rise to symptoms resembling those of a true meningitis, the term is not strictly accurate, though it is well to bear in mind that this form of new growth does occur and give a picture of meningeal irritation.

EXAMINATION OF CEREbro-SPINAL FLUID

The normal cerebro-spinal fluid is clear and colourless. As obtained by lumbar puncture, it is found to be under a pressure of from 60 to 150 mms. of water and to contain from 0 to 5 cells (lymphocytes) per c.cm. Its chemical composition is as follows:

Protein (mainly

albumin)	.	0.01 per cent.	(10 to 20 mgrms. per 100 c.c.)
Glucose	.	0.05 to 0.08	„ (50 to 80 mgrms. per 100 c.c.)
Chlorides	.	0.72 to 0.75	„ (725 to 750 mgrms. per 100 c.c.)

The lumbar puncture is made in the first interspinous space above a line joining the highest points of the iliac crests, which is the space between the third and fourth lumbar spines, or it may be made with equal rectitude in the space between the second and third lumbar spines. The needle should be introduced exactly in the middle line and at right angles to the surface, close to the upper spinous process of the interval used. Normally the fluid escapes drop by drop. If it runs rapidly or spurts out, this is an index of the increase of the cerebro-spinal pressure. Such an increase is met with in all meningeal inflammation, congestion and hæmorrhage, and in increased intracranial pressure. But it is also produced by coughing or straining, and its accurate measurement by a manometer is now part of the routine examination of the fluid.

Queckenstedt's phenomenon.—If, with a manometer in attachment to a lumbar puncture needle, the jugular veins be compressed, an immediate rise in cerebro-spinal fluid pressure in the normal person will be noted, the pressure rising as high as 300 or 400 mms. of fluid. It falls as rapidly when compression is released. If there be a block in the spinal subarachnoid space, or in the exits from the ventricular system, there will be no rise (complete block), or a slight rise with a delayed fall (incomplete block). Again, if the removal of a few c.cms. (4 to 8) of cerebro-spinal fluid is followed by a persistent fall in pressure of about 50 per cent., there is probably a block in the spinal subarachnoid space. These two tests afford valuable information in cases of suspected "obstructive" hydrocephalus, or of spinal block from tumour, or other local disease.

Increase of Protein Content.—This is of high importance, and occurs in all conditions of meningitis, and especially when the thecal space is obstructed by tumour, pressure from without or meningeal adhesions. In pathological conditions the protein content may reach 0.8 per cent. or more. A high protein content sometimes associated with xanthochromia, in the absence of cellular elements, is highly characteristic of thecal obstruction and is known as "Froin's syndrome." Lethargic encephalitis does not give any increase of protein.

Xanthochromia is a yellow colour of the cerebro-spinal fluid, and it is met with when the cerebro-spinal space is obstructed by tumours of the cord or meninges, or by external pressure, and in some forms of polyneuritis and meningitis. The yellow colour may result from an extravasation of blood either into the arachnoid or into the central nervous system.

Spontaneous coagulation of the fluid is met with in some cases of meningitis, when there is spinal obstruction, and in some varieties of acute polyneuritis.

Blood may occur from every condition of hæmorrhage, injury and encephalitis, and sometimes in meningitis. Blood that has been long shed into the cerebro-spinal fluid tends to become brownish and later yellowish. Leucocytosis is indicative of meningitis and often occurs in encephalitis, and in the neurotropic virus infections and in mumps. A lymphocytosis is characteristic of tuberculous and syphilitic meningitis, poliomyelitis, lethargic encephalitis, sinus thrombosis and mumps. A polymorphonuclear cytolysis occurs in meningococcal meningitis and all the suppurative forms of meningitis. In tuberculous meningitis there is often a mixed cytolysis at first, in which the polymorphonuclear cells may form 60 per cent. of the total. In

poliomyelitis, the lymphocytosis disappears after the end of a week. In lethargic encephalitis there is frequently no lymphocytosis.

Pus may be present in quantity in all the septic forms of meningitis, and especially in pneumococcal and epidemic meningitis.

Decrease in Glucose Content.—All conditions of meningitis cause decrease in the glucose.

Alteration of Chloride Content.—Diminution in chlorides is highly characteristic of tuberculous meningitis, and a reduction below 0.65 per cent. is pathognomonic of that condition. Increase in chlorides occurs in uræmia and other conditions of salt retention.

Lange's Colloidal Gold Reaction.—In neurosyphilis and in some cases of disseminated sclerosis, the globulin fraction of the total protein of the cerebro-spinal fluid increases and may almost equal the albumin fraction. The high globulin content gives the fluid a power of precipitating colloids from suspension. The estimation of this power in the case of colloidal gold is the basis of Lange's test. To ten dilutions of cerebro-spinal fluid (from 1 in 10 to 1 in 10,000) constant amounts of colloidal gold are added, and the mixtures allowed to stand for 24 hours. The form of the precipitation curves has a differentiating value. Thus in general paralysis the first six dilutions are precipitated (paretic curve), in tabes dorsalis, the third and fourth dilutions show the maximal precipitation (luetie curve); in meningitis, the sixth to eighth dilutions are precipitated (meningitic curve). In disseminated sclerosis the combination of negative Wassermann reactions in blood and fluid and a paretic curve in the fluid is frequently found.

The nature of the organismal content is determined (1) by the direct examination of films made from the centrifugalised fluid, (2) by cultures from the fluid, and (3) by the inoculation of animals from the fluid.

NAME.	ORGANISM.	CEREBRO-SPINAL FLUID.
Tuberculous meningitis	Tubercle bacillus.	(Clear or turbid. Lymphocytes, either alone or in greater numbers than polymorphs. Tubercle bacilli diminished. Chlorides diminished.
Pneumococcal meningitis	Pneumococcus.	Turbid. Polymorpho-nuclear leucocytes. Pneumococci.
Meningococcal meningitis		{ Clear or turbid. Polymorpho-nuclear leucocytes. Intracellular diplococci.
Sporadic or posterior-basal.	Still's diplococcus.	
Epidemic or "Spotted Fever."	Weichselbaum's diplococcus.	
Pyogenic meningitis	{ Staphylococcus. Streptococcus. B. influenza. Gonococcus. Streptothrix.	Turbid. Polymorpho-nuclear leucocytes. Organisms.
Syphilitic meningitis	Spiruchæa pallida.	Clear. Lymphocytes only. Wassermann reaction +.
Other forms	{ B. typhosus. B. enteritidis. }	Turbid. Polymorphs.
Rheumatic meningitis	Diplococcus rheumaticus.	
Serous meningitis		Clear. Few cells. Sterile.
Traumatic meningitis		

The Wassermann reaction in the fluid is positive in all conditions of recent syphilitic disease impinging upon the meninges, and always in general paralysis. Though often positive in tabes, it may be found negative. Lumbar puncture is dangerous in cases of long-standing increased intracranial pressure, and if performed in such cases a minimum of fluid should be withdrawn. It may in some cases cause severe headache of long duration. It may be difficult or impossible to perform when there is bone disease of the lumbar vertebrae.

The most useful classification of the varieties of meningitis is according to the nature of the micro-organism producing the inflammation.

1. TUBERCULOUS MENINGITIS

This disease results from the general invasion of the cerebro-spinal leptomeninges by the tubercle bacillus, and this organism invariably arrives in the meninges by the blood stream from some previously existing focus of tuberculous infection within the system, and most commonly from caseous tracheo-bronchial glands and tuberculous bone disease. Occurring at all ages, it is the form by far the most frequently met with in the second and third years of life. The characteristic features of the cerebro-spinal fluid are, that it is usually under considerable pressure, it is clear or only slightly turbid, has no visible deposit before centrifugalisation, but it often forms a fine flocculent clot. It contains an excess of albumin. The normal sugar is generally absent. There is a pleocytosis with a high proportion of lymphocytes, 70 to 80 per cent. being of this nature, and the rest being polymorphonuclears. Careful examination will almost always reveal the presence of the tubercle bacillus.

Ætiology.—The inheritance of a lowered resistance to the invasion of the tubercle bacillus is an important factor, especially when such a tendency exists in both parents. The sexes are equally affected. Tuberculous meningitis is rare during the first year of life, and especially during the first 6 months of life, when posterior basal meningitis is most common. Its greatest incidence is during the second and third years. It is common throughout childhood and early adult life, after which it becomes increasingly rare. The primary focus from which the organisms are spread to the meninges is most commonly a tuberculous mesenteric or bronchial gland.

Sometimes the source of infection is tuberculous disease of the lungs, of the abdomen, of the ear, of the joints, or of bone. Operations upon the sites of tuberculous disease may directly cause the dissemination of the tubercle bacilli, and especially surgical procedures upon tuberculous intracranial tumours, upon spinal caries, and upon tuberculous disease of bones and joints. The acute specific fevers, and especially measles, are sometimes the exciting causes of the disease. Injury to the head sometimes determines the attack.

Pathology.—In tuberculous meningitis three kinds of lesions of the meninges may be met with—(1) grey tubercles unassociated with inflammatory deposit; (2) tuberculous meningitis characterised by the presence of tuberculous granulations associated with a fibrinous and purulent exudation—the superficial tissue of the nervous system underlying the meninges is in this case always involved; (3) tuberculous tumours of any size up to that

of a pigeon's egg. It is not uncommon to find such a tumour to be the focus of widely spread meningitis. The three kinds of lesions may coexist in the same case.

The flattening of the convolutions and the dry sticky feel of the surface of the brain are highly characteristic. The disease affects the pia-arachnoid and its processes, the small vessels entering the surface of the brain and the superficial tissues of the latter. Occasionally a few tubercles are found upon the inner surface of the dura mater. Generally the convexity of the brain escapes, or is little affected. In the intercerebral space, around the optic chiasma, covering the tips of the temporal lobes, along the commencement of the Sylvian fissures and around the brain stem, there is an inflammatory exudation of tough consistency and of a pale yellowish-green colour. Spreading from the edge of this in decreasing numbers, grey tubercles are seen in the pia-arachnoid, particularly along the Sylvian fissures. They may be found wherever the pia-arachnoid extends (the convexity, as a rule, excepted), but except at the base of the brain they are not, as a rule, accompanied by the characteristic tough exudation.

The brain as a whole is soft, and local softening of the walls of the ventricles, of the velum interpositum, and of the fornix is often present. This softening is caused by spreading of the tuberculous process from the pia-arachnoid to the small vessels of the surface of the brain, on the walls of which tubercles develop, sometimes in such numbers that a small entering vessel, when observed under low magnification, after the brain tissue has been removed by careful washing, may resemble a bunch of grapes, each grape being a tuberculous nodule. Thrombosis is a common event in the vessels so involved, and softening follows. Some degree of thrombosis in the superior longitudinal sinus and in the veins of Galen is commonly present. It is probably owing to the softening of the nervous tissues that occlusion of the foramina by which the cerebro-spinal fluid leaves the ventricles does not often take place, and that, therefore, a condition of hydrocephalus does not occur in this form of meningitis. The cranial nerve palsies which are so frequently met with in this disease are the result of implication by adhesions and local interference with the blood supply of the nerves at the base of the brain, by the newly formed adhesive tissue. In the majority of cases the membranes of the spinal cord are affected, and the most common situation of the tubercles is upon the inner surface of the theca, and in the pia covering the lumbar enlargement.

Symptoms.—The onset is usually gradual, with signs of vague and slight illness. In children, general apathy and neglect of amusements and play, headache, loss of appetite, dullness, fretfulness, restlessness at night with grinding of the teeth during sleep, headache, vomiting and pyrexia are common symptoms. In older subjects, lassitude, depression, mental alteration, perversity and hysterical¹ manifestations are common. Constipation is usually present, and the breath has a peculiar fetor. The facial expression is one of illness and frowning discomfort, and there is disinclination to talk. Young children may be speechless for days together. As a rule, in this stage of the disease young children complain of nothing, and delirium is rare; but as age advances, delirium increases in frequency, and headache, usually frontal is increasingly complained of. These slight and vague symptoms may last from a few days to several weeks, and constitute what has been called

the prodromal stage of the malady. An early disappearance of the knee and ankle jerks, and the occurrence of retention or urine are often early signs and should be looked for in suspected cases. In those cases which are said to being acutely, careful inquiry will generally reveal that some symptoms such as the above have preceded the acute onset. The further development of the disease is marked by the appearance of a lethargy, which soon deepens into a stupor, from which it is difficult or impossible to arouse the patient. Vomiting is of frequent occurrence, and headache may be severe. The child lies upon its side in the "cramped" position, resenting any disturbance. The expression becomes vacant, with wide-open eyes and dilated pupils, as if fixed upon some distant object. There is often some retraction of the angles of the mouth, and there is frequently a bright malar flush. In the later stages the limbs are generally extended and rigid. Stiffness of the neck is the rule, and head retraction may occur, but this is never so marked as in posterior basal meningitis. The abdomen is always markedly retracted and a *tâche cérébrale* is often conspicuous. A single sharp cry, apparently causeless, called the hydrocephalic cry, and which is common in all forms of meningitis and also in other infantile intracranial affections, is sometimes heard.

Ocular phenomena make their appearance towards the end of the first week of the developed disease. All varieties of varying and persistent strabismus and ptosis are met with, paralysis of the external rectus being the most common. Rolling movements and independent movements of the eyeballs may occur. None of these signs is constantly present. The pupils may be contracted at first, and may show varying inequality, but in the later stages they are dilated. Papillœdema is often present towards the end of the second week, if the patient survives so long. It is of moderate intensity, the height of the swelling rarely exceeding two dioptries. Choroidal tubercles sometimes occur.

Convulsions are common in every stage of the disease in children, but rare in adult cases. They may be the first symptom of the onset, but are more often met with in the later stages of the disease. They may be local or general. Repeated rhythmic movements are frequent, and are specially noticeable in connection with the mouth, where sucking and champing movements and grinding of the teeth are common. Rhythmic movements of the limbs may also occur. Coarse tremor upon movement of the limbs is the rule, and spasmodic twitching of the muscles is frequent. In rare cases, movements exactly like those of chorea occur. Kernig's sign is usually present.

The temperature is usually raised one or two degrees, but it presents no characteristic features, some cases being apyrexial throughout. Irregularity of the pulse is the rule, and is of considerable diagnostic importance. Rapid in the early stages, it tends to become unduly slow in the stage of coma, and again rapid as death nears. Cheyne-Stokes breathing and grouped breathing are common. Constipation is usually a marked and persistent feature.

Course.—The course of tuberculous meningitis is progressive to an invariably fatal termination in from a few days to 3 weeks after the appearance of definite symptoms, and no case of recovery is known to me in which the diagnosis has been unquestionably proved by the recovery of the tubercle bacillus from the cerebro-spinal fluid.

Diagnosis.—The early symptoms of the disease may give rise to difficulty in diagnosis, but the latter is relatively simple when the disease is advanced. The diseases liable to be confused with tuberculous meningitis at its commencement are gastro-intestinal catarrh, the exanthemata—especially enteric fever—and pneumonia. It must be borne in mind that in children convulsion, strabismus, head retraction and stiffness of the neck, with pyrexia, may be symptomatic of many maladies apart from meningitis, especially of apical pneumonia. In enteric fever the temperature is higher and the headache more severe, and irritability and resentment of interference are not present; the decubitus is usually dorsal. Widal's test is of importance in this connection. When distinctive signs of intracranial disease have appeared the diagnosis has to be made from other forms of meningitis, sinus thrombosis, tumour, abscess and middle-ear disease. Careful examination of the retina and of the tympanic membranes is then necessary. In all cases the diagnosis must be made certain by the examination of the cerebro-spinal fluid, which will be found to contain lymphocytes in excess and tubercle bacilli. These organisms are sometimes difficult to isolate from the fluid, but their presence can be readily demonstrated by injecting the fluid into the subcutaneous tissue of guinea-pigs, when the characteristic lesion of tubercle results. It must be remembered that in some cases the polymorphonuclear leucocytes may be in excess, but these cases are at once distinguished from other forms of meningitis by the presence of numerous lymphocytes, by the absence of the meningococcus and of the other germs producing suppuration, and by the presence of the tubercle bacillus. Pirquet's skin reaction is often absent in tuberculous meningitis.

Treatment.—From the unvarying fatal issue of the malady, treatment can only be directed towards the relief of symptoms. Temporary improvement and relief of headache may be brought about by lumbar puncture, which may for this purpose be repeated several times. Bromides, chloral, aspirin and other analgesic drugs may be used to relieve the headache, check the convulsion and diminish the restlessness. Hexamine in large doses, inunction of mercury and administration of tuberculin have been largely used, but without any success. General treatment must be that which will secure such comfort as is possible for the patient. Where swallowing is difficult nasal feeding should be adopted.

2. PNEUMOCOCCAL MENINGITIS

Pneumococcal infection of the meninges most commonly follows upon a similar infection elsewhere in the body, empyema and pneumococcal otitis being the commonest lesions, while pneumonia, abdominal infection, abscess and joint infection are less common. In one-third of the cases, however, the meningeal infection is primary. The characteristics of the cerebro-spinal fluid are that it is purulent and sometimes so thick that it will not flow through the needle. It is greenish-yellow in colour, contains a large amount of albumin, and multitudinous polymorphonuclear cells, among which the characteristic pneumococcus is found. In fulminant rapidly fatal cases the fluid may be turbid from the presence of pneumococci alone, no reaction in the form of pleocytosis being present.

Ætiology.—The disease may occur at any age. It is sometimes a terminal

event of a pneumococcal infection elsewhere, and passes almost unnoticed, or is discovered only at the autopsy. Meningitis which follows operations upon the nose and disease of the nasal bones is usually of the pneumococcal variety.

Pathology.—The surface of the brain and spinal cord is highly characteristic. Usually the whole surface of the vertex and of the base is covered with a thick, tenacious, greenish-yellow pus, which is contained in the meshes of the arachnoid, and between this and the dura. The ventricles often contain pus. A similar exudation is found upon the spinal cord, more especially upon the dorsal aspect, and in the cervical and lumbo-sacral regions. The major affection of the vertex of the brain is the peculiarity of this disease, and only in the rarest cases is the base alone affected. The exudation is characterised by the greater amount of fibrin than in other forms of meningitis.

Symptoms.—The symptoms are those which are common to all forms of meningitis. Some of the cases are indistinguishable symptomatically from cases of tuberculous meningitis. Others run a very rapid course and present few features other than headache, vomiting and pyrexia, with rapidly oncoming and quickly fatal coma. In others again, the meningeal symptoms are concealed in the terminal asthenia of a previously existing pneumococcal infection elsewhere, such as empyema, purulent pericarditis or peritonitis.

Diagnosis.—This rests upon the presence of signs of meningitis or the existence of coma alone, together with a cerebro-spinal fluid which is purulent from the presence of polymorphonuclear leucocytes, containing the pneumococcus.

No case of recovery from this form of meningitis has hitherto been recorded. Lumbar puncture and intrathecal injections of anti-pneumococcal serum may be performed, but on account of the thick nature of the exudate, little relief must be expected from the former, while the latter cannot possibly avail except in primary cases.

3. MENINGOCOCCAL MENINGITIS (see p. 36)

4. PYOGENIC MENINGITIS

Apart from meningococcal and pneumococcal infections, suppurative meningitis may result from the invasion of the meninges by staphylococci, streptococci, gonococci, *B. influenza*, *B. anthracis* and streptothrix.

Staphylococcal and streptococcal infections are by far the most common. They may result in young children from septic conditions of the umbilicus and from infections of the skin. Usually they are due to extension of an infection of adjacent structures to the meninges, and follow disease of cranial and spinal bones, especially caries of the middle ear, erysipelas and other infections of the scalp, wounds of the meninges, especially bullet wounds, rupture of intracranial abscess, and they may occur in the course of a general septicæmia.

Pathology.—The pathology of these conditions does not materially differ from that of pneumococcal meningitis. In all cases the exudation

is purulent, and in the meningitis due to *B. anthracis* it is of a red colour, due to concomitant blood effusion. The cerebro-spinal fluid contains large numbers of polymorphonuclear leucocytes, together with the micro-organism responsible for each variety. Suppurative meningitis resulting from bone disease and from wounds of the meninges may be localised by the formation of meningeal adhesions, and an intrameningeal abscess may result. Such an abscess situated upon the upper surface of the temporal bone is not an uncommon result of caries of the middle ear.

The clinical aspect is that common to all forms of acute meningitis, high pyrexia, rigors and delirium being conspicuous. The course is rapid to an almost invariably fatal termination. In the localised form where drainage can be ensured and extension of the infection prevented, recovery should take place. Several cases of recovery from gonococcal meningitis have been reported. Influenzal meningitis is invariably fatal.

Diagnosis.—This depends upon the presence of the clinical signs of meningitis and of a cerebro-spinal fluid containing polymorphonuclear leucocytes in large quantities, and upon the recognition in this fluid of the several micro-organisms responsible, by microscopic and cultivation methods. The recognition of *B. influenzae* requires that cultures should be made upon some blood medium, for otherwise the organism may be easily overlooked and the fluid reported as sterile. Further, the presence of some well-known cause for suppurative meningitis, such as ear disease, staphylococcal infection, etc., suggests the diagnosis.

Acute otitis media may give rise to symptoms closely resembling those of meningitis, such as headache, pyrexia, vomiting, head retraction and delirium. In such cases examination of the ear, which should be made a routine in all cases where meningitis is suspected, will reveal tympanic distension, the relief of which is followed by a speedy disappearance of the symptoms. In this connection it must be borne in mind that meningitis and intracranial abscess never follow directly upon acute otitis, but they are the sequelæ of chronic otitis, which has resulted in caries of the temporal bone. When evidences of caries of the middle ear are present in a case presenting cerebral symptoms, distinction has to be made between meningitis and abscess of the brain or cerebellum. Here the presence of localising symptoms, either temporal or cerebellar, and the presence of papilloedema and any tendency to a temporary abatement of the symptoms point to the existence of an abscess, and further lumbar puncture will in all but the rarest cases settle the point. In cases of abscess in which cells and organisms are found in the cerebro-spinal fluid, these exist in small numbers only, as compared with the copious cells and organisms present in the fluid of suppurative meningitis.

Treatment.—In cases of meningitis secondary to temporal caries, the source of infection should be at once cleared out by surgical procedure. Repeated lumbar puncture may relieve symptoms, and injection of an anti-serum to the organism present may be tried. Vaccines may also be used.

The *treatment and prognosis* of all the foregoing forms of meningitis, except tuberculous meningitis, have undergone a great change since the introduction of sulphanilamide and its derivations. The mode of employment of these drugs is dealt with on p. 49 (cerebro-spinal fever).

5. BENIGN ASEPTIC MENINGITIS

Synonyms.—Epidemic Serous Meningitis; Benign Lymphocytic Meningitis.

Ætiology.—This is unknown. No organisms have been found in the cerebro-spinal fluid. The malady so named appears of wide distribution.

Pathology.—Since recovery is the rule, nothing much is known of this, but lymphocytic infiltration of the lepto-meninges has been found in one fatal case.

Symptoms.—Children are mostly affected, but no age appears exempt. The onset is abrupt, with headache, sickness and fever. The typical signs of meningeal irritation are present, neck and spine rigidity, Kernig's sign, irritability and restlessness and sometimes delirium. Somnolence is unusual. In young children convulsions may occur. The fever mounts to 102 or 103 and fluctuates. It may disappear in 2 or 3 days, or persist for 3 weeks. Lumbar puncture yields a cerebro-spinal fluid under pressure, usually clear but sometimes opalescent. The cell count ranges from 50 to 1500 per c.mm. After the first two or three days these cells are almost wholly lymphocytes. The sugar and chloride contents remain at normal height, thus differing from the findings in other forms of acute lepto-meningitis, and resembling the findings in acute poliomyelitis.

Diagnosis.—This depends upon the cerebro-spinal fluid findings and upon the benign course of the illness. For a few days differentiation from poliomyelitis may be impossible.

Prognosis.—Recovery is the rule.

Treatment.—Repeated lumbar puncture reduces the intracranial tension. Beyond this, only general nursing care is needed.

6. SYPHILITIC MENINGITIS

Meningitis due to infection by the *Spirochæta pallida* is one of the characteristic lesions met with in practically all cases of syphilitic disease of the central nervous system, and plays its part in the production of the symptom complexes of these maladies, from acute cerebral syphilis and acute myelitis to general paralysis and locomotor ataxy. It may occur at any period after infection, but one-half of the cases occur during the first four years. In a few cases the symptoms have been noticed coincidently with the syphilitic roseola.

Pathology.—The morbid process consists essentially in an infiltration of the meninges with lymphocytes and plasma cells, spreading from the perivascular lymphatics where the spirochætes multiply freely, and it may lead to scarring and opacity of the membranes, with consequent strangling of the nerves and vessels and occlusion of the arachnoid space, or to massive gummatous formation in the meninges. It is essentially a chronic form of meningitis though it may result in the production of acute symptoms. A marked feature is that the meningeal changes may be found actively progressive in one spot, and equally regressive in another. The disease may be local or diffuse, and it may attack the dura (pachymeningitis) and involve the overlying bone, or it may spread from the pia-arachnoid into the sublying nervous tissue (meningo-encephalitis).

The cerebro-spinal fluid is characteristic. It is usually under increased

pressure, is clear and colourless, and contains lymphocytes and no other cell forms. The number of the lymphocytes present is in direct proportion to the activity of the meningeal syphilis. The spirochæte has rarely been found in the fluid, yet inoculation of apes with the fluid has proved successful.

Symptoms.—Apart from those conditions of nervous syphilis in which meningitis is associated with arterial disease, the formation of massive gummata and neuronie degeneration, syphilitic meningitis may be described as giving rise clinically to the following conditions :

1. *Headache.*

2. *Hydrocephalus.*—In those acute cases of cerebral syphilis characterised by rapidly oncoming headache, vomiting and papilloedema, mental reduction and somnolence without localising symptoms, and which respond readily to treatment, it seems certain that ventricular distension, consequent upon adhesive meningitis and ependymitis, is responsible. A more slowly oncoming ventricular occlusion may give rise to symptoms which cannot be distinguished from those caused by a non-localisable intracranial tumour. Syphilitic meningeal occlusion may give rise to typical hydrocephalus, and a considerable proportion of the cases of infantile hydrocephalus are of this nature and are due to congenital syphilis. A few cases are recorded in which chronic hydrocephalus of this nature has occurred in adult life.

3. *Infantile syphilitic meningitis.*—This is a chronic malady which commences insidiously during the first few months of life, with signs of general nervous deterioration. The appearance of the brain is very characteristic. The membrane over the vertex is opaque and thickened and adherent to the cortex. The gyri are shrunken, the sulci wide and the surface of the brain has in parts the appearance of wash-leather. The child does not get on, and takes an ever-decreasing notice of its surroundings. Power of movement lessens, the limbs become rigid and the clinical aspect comes to resemble exactly that of a severe cerebral diplegia. Convulsions are of frequent occurrence. The diagnosis is not difficult, for the signs of meningitis are obvious and those of congenital syphilis may be present. There is an excess of lymphocytes in the cerebro-spinal fluid, both in which and in the blood there is a positive Wassermann reaction. The prognosis in any case where the symptoms have become marked is most unfavourable.

4. *Adult syphilitic meningitis,* with a symptom-complex closely resembling that of tuberculous meningitis, has been reported on many occasions. In some of the cases the onset coincided with the appearance of the syphilitic roseola. The diagnosis depends upon the presence of signs of active syphilis, upon the cerebro-spinal lymphocytosis and upon the existence of a positive Wassermann reaction. The prognosis under appropriate treatment is good.

5. *Paralysis of cranial nerves.*—This common and often isolated symptom of nervous syphilis may result from sclerosing basal meningitis or from the presence of a gumma in the course of the nerve. Several of the nerves may be involved together in one patch of meningitis. Any of the cranial nerves may be affected from the olfactory to the hypoglossal, but the third or oculomotor nerve is by far the most frequently attacked.

Treatment.—The treatment of the above conditions is that appropriate for nervous syphilis in general (p. 1636). The combined administration of mercury by inunction and of arsenic compounds by intravenous injection

gives the best results. Iodide of potassium is not nearly so useful as when massive gummata are present, and, moreover, it seems to increase the scarring process. Its use should be avoided until the patient has been under the influence of mercury for some time.

7. OTHER FORMS OF MENINGITIS

Meningitis due to the typhoid bacillus is a rare malady. It may occur as a primary disease, but is usually a complication in the course of enteric fever. It is to be remembered that while many cases of enteric fever present cerebral symptoms, in very few can meningitis be proved to exist. The meningeal exudation, generally serous, is sometimes purulent. The cerebro-spinal fluid contains lymphocytes, and Eberth's bacillus is present. The symptoms resemble those of acute meningitis in general. The diagnosis depends upon the presence of enteric fever, of Widal's reaction and the discovery of Eberth's bacillus in the cerebro-spinal fluid. The malady is generally fatal, but a considerable number of recoveries have occurred, especially in children. In rare cases symptoms of meningitis occur in the course of rheumatic infection, and Poynton and Paine have brought forward evidence that such symptoms are the result of infection of the meninges with the *Diplococcus rheumaticus*. The term "serous meningitis" is applied to those cases of meningitis in which the cerebro-spinal fluid is clear and sterile. In such cases recovery is the rule, and the symptoms are not rarely rapidly relieved by lumbar puncture. The term "meningism" is used for a group of cases which present symptoms of meningitis and in which no pathological change can be found either in the cerebro-spinal fluid, or, if death occur, in the meninges or cerebral tissue. It is met with in children in association with acute febrile diseases, and is presumably due to the toxin present. Recovery is usually rapid and complete.

VIRUS DISEASES OF THE NERVOUS SYSTEM

Certain viruses have a selective affinity for the nervous system and are therefore spoken of as "neurotropic." They act upon the nerve cell, and to a less degree upon glia cells, but not upon the white matter. They are capable of multiplication and of exerting their pathogenic action only within the nerve cell, where their life and activity are short-lived. Thus, the infections to which they give rise are known as self-limited.

The essential lesion resulting from their presence is an acute necrosis of the nerve cell, leading to the death and destruction or to the damage of the cell. A secondary glial and vascular reaction ensues as a result of which lymphocytes pass into the cerebro-spinal fluid from the perivascular spaces in the affected regions of the nervous system.

Such virus infections are primary infections of the nerve cell and not invasions of the central nervous system secondary to general infection. The portal of entry in all cases, except that of rabies, is probably the nasopharynx by the mechanism of "droplet infection." The virus is carried thence via the olfactory filaments into the olfactory bulbs and tracts. Gaining direct access

to the brain in this way it seems certain that the virus is thereupon transported within the system through axis cylinders. This at least appears to be the mechanism in the most closely studied of the virus infections of the nervous system, acute poliomyelitis.

The so-called post-infective encephalitis that may follow the acute exanthemata is not a true virus infection of the nervous system, since the lesion is a demyelination and not an attack upon the nerve cell. This form of encephalitis, therefore, is probably to be regarded as an intoxication of the nervous system associated with a systemic virus infection.

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ACUTE ANTERIOR POLIOMYELITIS

Synonyms.—Infantile Paralysis ; Heine-Medin Disease.

Definition.—An infection of the nervous system by a neurotropic virus which acts primarily upon the nerve cell, the interstitial tissues of the nervous system and the leptomeninges being secondarily involved. Contrary to what was formerly believed, there is no initial phase of general systemic infection ; from the outset, poliomyelitis is a disease of the nerve cell. The virus is an obligatory intracellular parasite, and has a special affinity for the ventral horn cells of the spinal cord, within which it multiplies during its brief period of activity.

Ætiology.—A constant feature of the disease is its incidence in young children. They appear to be immune during the first year of life, susceptibility being greatest in the second, third and fourth years, and thereafter declining. Cases during adolescence and early adult life are not uncommon, but the disease is rare after middle life. It is possible that a very large proportion of the community has at some time been infected by poliomyelitis, only a very small proportion (less than 1 per cent.) having developed symptoms of infection. From 50 to 80 per cent. of the adult population possess serum containing protective antibodies, and this fact is taken as presumptive (though not certain) evidence of previous infection. That such clinically latent infection is possible may be seen from the occurrence of the many "abortive" cases of poliomyelitis in all epidemic outbreaks. The disease is much more prevalent during the hotter months of the summer, usually the months of August and September in the Northern hemisphere, and the months of March and April in the Southern. It is spread by human carriage by infected persons in the initial stage of their illness, and probably also by healthy carriers. The nasopharyngeal secretions are infective during the first ten days of the illness, and spread probably takes place by what is known as "droplet infection." It has been said that case-to-case infection does not occur, but the long-continued case-incidence in certain small, enclosed communities strongly suggests that, however difficult it may be to trace, such infection does in fact occur, and must be allowed for in dealing with outbreaks of the disease. There is some evidence that milk may also act as a channel of conveyance of the virus.

THE PATH OF INFECTION.—It is exceptional for the virus to be found in the blood or cerebro-spinal fluid, and it is not by either of these channels that it reaches the central nervous system. The portal of entry into the body is

the nasopharynx, whence the virus enters the free ends of the olfactory nerve filaments, and travels by the axis cylinders directly into the olfactory bulb, and thence into the brain. The subsequent passage of the virus to the ventral nerve cells is believed to be entirely axonal.

It is believed that *all* the symptoms of the declared disease, from the first onset to the appearance of paralysis, are due to the effects of the virus acting upon nerve cells in its passage from the site of entry into the brain, until it reaches its site of election in the ventral horn cells of the lumbar region of the cord.

Pathology.—The virus of poliomyelitis is an obligatory intracellular parasite, and its action takes place entirely within the nerve cell. The changes seen in the nervous system vary with the virulence of the infection. In very severe infections the nerve cells undergo acute necrosis. If the experimentally infected animal be destroyed at this initial stage no lesions other than these cell changes are found, and the rapidly ensuing cellular exudation and meningeal infiltration seen in fatal human cases are not present. But the necrosis of nerve cells in the surviving patient is naturally shortly followed by phagocytic processes, and amœboid (microglial) cells and polymorphonuclear leucocytes rapidly invade the affected areas and clear away the dead nerve cells. It is essential to bear in mind, however, that these processes are secondary and not, as used to be thought, the primary and essential lesion of poliomyelitis. In less severe infections, less acute forms of nerve cell changes are seen, and with these the cellular exudation is almost wholly of amœboid microglia cells. These phagocytic cells fill the perivascular spaces in the affected parts of the cord. Together with leucocytes, these cells finally overflow into the cerebro-spinal fluid. They appear here even before the development of paralysis; and it is this early indication of meningeal infiltration that led to the view formerly held that a meningitis preceded the involvement of the nervous system. In the affected regions of the grey matter of the ventral horns, some cells always remain unaffected by the virus.

Lesions in the viscera have been described: namely, hyperplasia of the lymphoid tissue and splenic enlargement—but these are not constant, and their presence at the final stage of the malady is of uncertain significance. They are no longer held to indicate the occurrence of systemic infection in poliomyelitis.

Cerebro-spinal fluid.—The fluid is clear, colourless, or faintly yellow, and under high pressure, and the titre of chlorides and sugar is normal. The protein content is increased. There is usually a pleocytosis from a moderate to a very large number of cells, which disappears rapidly with convalescence and is usually gone in a fortnight. It is usually described as polymorphonuclear at first and rapidly becoming lymphocytic, but in our experience this is certainly not the rule. We have seen the fluid normal throughout in indubitable cases; we have seen a pure lymphocytosis throughout and from the preparalytic stage; and we have seen high polymorph pleocytosis, both early and late, and also with a relapse on the tenth day. The nature and number of the cells seem not to afford any prognostic indications.

Blood.—In the early stages of the malady, there is a constant and very marked polymorpho-nuclear pleocytosis, which may reach as high as 30,000, with lymphocytic leucopenia. This leucocytosis disappears when the fever abates.

Symptoms.—It has been the practice to divide the clinical course of poliomyelitis into an initial stage of general infection; a second stage of meningeal involvement; and a final stage of paralysis from invasion of the nervous tissues. Cases in which recovery ensued after a brief febrile illness and without paralytic manifestations were spoken of as “abortive,” it being supposed that the infection had died out before the virus had invaded the spinal cord. This classification, based as it was upon an erroneous pathology, may now be abandoned in favour of a division into preparalytic and paralytic stages; it being understood that in both stages, and therefore also in abortive cases, the virus has in fact invaded the nervous system before any symptoms whatever have appeared, and that even the symptoms of the preparalytic stage are almost certainly the expression of the action of the virus upon the nervous system. In short, there is no clinical stage of poliomyelitis in which the virus has not already gained access to nerve cells, and begun to exert its pathogenic effects. The failure of serum therapy in the past is probably to be explained by this fact.

The preparalytic stage.—This may last from 1 to 4 or 5 days. It is almost invariably febrile, the temperature rising to 103 or 104 degrees. The pyrexia lasts for from 1 to 3 days and then declines, sometimes finally, sometimes recurring for a day or two as paralysis develops.

To those who have studied the disease in the course of a large epidemic there can be no doubt that the clinical picture of the opening phase is pathognomonic, and clearly to be distinguished from that of other febrile illnesses of childhood. Draper has given a very vivid account of this stage, from which the following statement is taken. The child is commonly flushed and miserable, and may be drowsy, but it presents a typical appearance of mingled apprehension and restlessness, and may be very irritable. In severe infections the child breathes rapidly, appears pre-occupied and in a state of tenseness. An ataxic tremor and involuntary muscular jerkings may be present. Extreme fearfulness, and confused and alarming dreams are common. The child is hypersensitive to even the lightest touch and resents being moved. Vomiting, probably of central origin, may also be present. Headache, pain in the neck and back, stiffness of the spine and pain in the back on active or passive flexion, diminution of tendon jerks, and some diffuse weakness all appear in sequence, and finally paralysis suddenly appears in one or more groups of muscles. On the hypothesis that the sequence of symptoms in poliomyelitis from the opening of the clinical picture until the appearance of paralysis accurately represents the passage of the virus through the nervous system from olfactory bulb to ventral horn cell, Faber has drawn up the following table of symptoms.

Progress of Virus.

Characteristic Symptoms.

Olfactory bulb to hypothalamus and brain stem.	{	Fever, drowsiness, alternating with restlessness, sweating, general hyperæsthesia, apprehension, fear, irritability, heightened sensibility to pain, headache and vomiting.
Posterior columns of cord. Posterior root ganglia.		Localised pains and tenderness, disturbances of pain sensibility, pain on flexion of spine, Kernig's sign, transiently increased tendon jerks. Temperature may fall now.

*Progress of Virus.**Characteristic Symptoms.*

Anterior horn cells.	{ Twitchings, tremor, unsteady movements.
No further spread :	{ Flaccid paralysis, loss of tendon jerks.
Infection dies out.	{ Diminution of extent of paralysis, disappearance of pain and tenderness.

It would probably be premature to say that every stage in this progress of the virus has been finally and conclusively proved, but it is of course quite reasonable to take the view that all the initial symptoms of poliomyelitis are capable of being produced by an infection wholly confined to the nervous system; in other words, the assumption that there is an initial stage of general infection is superfluous, and without supporting evidence.

The paralytic stage.—It was formerly the practice to differentiate a number of types of poliomyelitis according to the localisation of the paralytic symptoms. Thus there were cerebral, cerebellar, brain-stem, spinal and neuritic types. As Weston Hurst has pointed out, it is impossible to produce in the experimental monkey, no matter what the site of inoculation of the virus, any but a spinal type of paralysis, and it is extremely doubtful if in the human disease there are any other than brain-stem and spinal cases. The remaining cases are probably due to some other cause than the virus of poliomyelitis, and they will not be considered here.

1. *SPINAL FORM.*—In young children, the paralysis is often not apparent until the second or third day of the illness. In older children and in adults, the paralysis is usually present within 24 hours of the onset. The paralysis is always of the flaccid variety, with loss of the deep reflexes in the region of the paralysis, and subsequent atrophy of the muscles if it is lasting; it develops very rapidly in most of the cases, and seems to have its full limit of distribution at the moment of its appearance, which facts correspond exactly with the experimental pathology. In some cases, however, the paralysis spreads rapidly from its original site, either in ascending or, more rarely, in descending fashion. The ascending cases are very liable to be terminated with fatal bulbar involvement. In rare cases, relapse occurs, and the paralysis, after remaining stationary for several days, may spread suddenly to other regions. This event, which is due to a recrudescence of the infection, has also been observed in experimental poliomyelitis. It is often stated that relapsing cases are usually fatal, but in a very considerable experience of such cases we have never seen a fatal issue, which on theoretical grounds should be highly improbable, on account of the rapid development of immunity after infection.

The paralysis is generally much more widely spread at the onset than it is destined to be permanently. At first all four limbs may be completely helpless, and later there may be complete recovery in all but one limb. The widely spread temporary paralysis is due to a recoverable affection of the nerve cells, whereas the permanent palsy is the result of an actual destruction of the cells by a necrotic lesion. The paralysis may affect any muscles of the body, but those of the legs are by far the most commonly involved, while those supplied by the nuclei of the brain stem are never permanently paralysed. The trunk muscles may be affected alone, giving rise to spinal deformity, usually of a scoliotic or kypho-scoliotic type. Thus, poliomyelitis

comes to be one of the very common causes of spinal curvature in the young. The narrowing down of the initial paralysis begins to show itself after the end of the first week, and any muscle which will recover useful power will have done so before the end of the third month. The paralysed muscles undergo atrophy, which is more rapid and complete in those cases in which there will be no subsequent recovery; they give the reaction of degeneration. They are flaccid from the first, and in the course of time tend to develop a variable degree of contracture, and yet it is common to see a limb which remains permanently flail-like. Any muscle which shows a response to faradism 3 weeks after the onset will completely recover. When a limb is paralysed, there is usually a considerable degree of vasomotor paralysis, and there may be subsequent retardation of growth. Considerable deformities of the body and limbs may arise as the result of the loss of support, which results from the paralysis, from the action of unopposed muscles, and from the contractures. Such deformity may involve actual dislocation of joints, as in the shoulder joint, where the deltoid is paralysed and the pectorals escape.

The local lesion of the spinal cord is by no means confined to the grey matter, and may involve the contiguous white matter of the lateral column sufficiently to give rise to signs of lesion of the pyramidal tract, and in rare cases of lesion of other neighbouring tracts, such as the spino-thalamic tract with a result in a Brown-Séquard's syndrome of pyramidal deficiency upon the same side and loss of pain and temperature sense on the opposite side below the lesion. Paralysis of the cervical sympathetic is not rare when the lower part of the cervical enlargement is involved, with the usual signs of a small pupil and low-lying lid on the affected side. It is, however, generally a transient event.

Disturbances of sensibility of an objective kind are rare, and are almost always transient, and amount to blunting of pain and temperature sensibility, from involvement of the spino-thalamic tracts which are continuous to the ventral horns. Subjective disturbances are common, and consist of severe local pains in the limbs, back and neck. Tenderness of the muscles, and pain on moving the joints are sometimes very prominent, and may persist for many weeks. The dominance of the clinical picture by persistent pains in the periphery constitutes the so-called "neuritic" form of poliomyelitis. Sphincter paralysis is quite common in the early stages in cases in which the lumbo-sacral enlargement is affected, but it is always rapidly transient.

The reflexes, both superficial and deep, are at first lost in the affected region, and indeed are generally absent throughout the body in the early stages of a severe case, from the general effect of the virus upon the nerve elements. In the later stages they return, or remain permanently absent, according as the muscles recover or not. Any sign of a returning reflex, either deep or superficial, in the early days of the illness is a most useful prognostic indication that the muscles concerned with the reflex will entirely recover.

2. THE BRAIN-STEM FORM.—In this type, the incidence of the lesions is upon the grey matter of the brain stem from the medulla to the region of the red nucleus. The general symptoms of the onset are as in the spinal form. In place of the paralysis of trunk and limbs there is bulbar, facial, trigeminal or ocular paralysis, according to the situation of the lesions. An extensive lesion of the medulla itself proves very rapidly fatal. Lesions of

the upper brain stem are more commonly survived, and the resulting clinical pictures are, in order of frequency of occurrence, facial paralysis, spastic tremulousness from involvement of the upper part of the brain stem, and lastly ocular paralysis with nystagmus.

Course.—Most commonly within a few days of the onset of the paralysis a very considerable remission occurs, and the paralysis becomes much narrowed down in its limits; thus, with an initial paralysis of all four limbs and trunk, the limbs recovered rapidly, leaving a permanent partial paralysis of the trunk, and in a case where both legs were paralysed, the one recovered power within the first week, leaving the other permanently crippled. Sometimes, however, there is no rapid improvement or narrowing of the region of paralysis whatever.

The paralysis remaining after the rapid improvement is final, and admits of such improvement only as may occur from the recovery of a few cells which have escaped destruction upon the confines of the inflammatory lesions, and such recovery is very slow and never reaches more than a slight degree. A certain slow improvement in those paralysed muscles which retain some voluntary power is often observable, and is referable to hypertrophy of function in those elements which remain and to the acquisition of the aptitude which necessity produces. On the other hand, children afflicted with this disease during the period of active growth will often show what seems to be a progressive diminution of power in the weak muscles, and which is, in reality, a relative failure of these muscles under the strain of the increasing weight and length of the body and limbs.

Death is uncommon at any stage in the spinal form of poliomyelitis except during epidemics, when severe general symptoms are followed by widely spread paralysis, involving all the respiratory muscles, and in these cases it takes place on the first day of appearance of the paralysis. Weakness of the respiratory muscles and especially total intercostal palsy is not infrequently an indirect cause of death, even at long periods after the onset, if bronchitis or broncho-pneumonia occur.

Diagnosis.—During the stage of general pyrexial symptoms and before the paralytic manifestations appear, a definite diagnosis can hardly be made; but it may be suggested by the time of year, by the prevalence of an epidemic, and by the combination of a polymorpho-nuclear leucocytosis in the blood with a lymphocytosis in the cerebro-spinal fluid. When the paralysis first sets in, the diagnosis has to be made from acute rheumatism, in which the painful joints may cause an appearance of severe paralysis. In the same way, syphilitic pseudo-paralysis (acute syphilitic epiphysitis) may be diagnosed from poliomyelitis. From acute polyneuritis and Landry's paralysis, both of which maladies may have a pyrexial onset with similar general symptoms, poliomyelitis can generally be distinguished by the sudden onset of the paralysis and by the absence of any spreading tendency, and probably by the lymphocytosis in the cerebro-spinal fluid, and later on by the permanent atrophic paralysis. In the rare spreading types of poliomyelitis, the latter two points alone serve to make the diagnosis.

From almost all of the local lesions of the spinal cord, membranes and roots, whether these are of rapid onset, as for example hæmatomyelia and acute myelitis, or of slow onset, such as tumour, inflammation and pressure, poliomyelitis is at once distinguished by the absence of the conspicuous

sensory loss and sphincter trouble which accompany the former diseases. In the final stage of permanent muscular paralysis and atrophy, deformities and contractures, poliomyelitis presents little difficulty of diagnosis, but it should be borne in mind how frequently deformities of the trunk and especially lateral curvature of the spine have their origin in slight attacks of this malady where the lesions are confined to the dorsal region.

Poliomyelitis may simulate meningitis so closely as to be hardly distinguishable. The skin in the former malady may be suggestively flushed and pink. A sterile cerebro-spinal fluid with no micro-organisms and with a mixed lymphocytic and degenerating polymorph pleocytosis and with the chlorides and sugar content normal can hardly be from any other case than one of poliomyelitis.

Prognosis.—It is rare for complete recovery to occur in any case of spinal poliomyelitis in which paralysis has once set in. Though recovery may be nearly complete, yet there seems always to be some region in which permanent muscular atrophy persists, and in cases which otherwise clear up, this is frequently in the spinal muscles, giving rise to a lateral curvature. From this condition of nearly complete recovery to one in which there is not the slightest recovery from the initial paralysis, there is every gradation. The prognosis is not influenced by the severity or otherwise of the general symptoms, for the paralysis may be slight where the general symptoms are severe, and vice versa. Incomplete paralysis or the presence of reflex action, either superficial or deep, in any region at the end of the first week after the paralysis has set in, is a sure indication that useful recovery will occur in that region. Those regions which remain completely paralysed for several weeks after the onset are certain to remain permanently disabled. The prognosis as to the eventual usefulness of disabled limbs, or as to eventual power of walking, depends upon a consideration of the muscles which are permanently paralysed, as to whether they are essential muscles or not, and whether they can be assisted or supplanted by any mechanical apparatus which is light enough for the weak limbs to carry.

Second attacks of poliomyelitis are exceedingly rare, but two such cases have been recorded by Eshner and by Sanz. The occurrence of progressive muscular atrophy in subjects who have in early life been afflicted with poliomyelitis is not very rare, and it is usual for the progressive atrophy to commence in the region originally affected by the poliomyelitis. Potts has recorded a series of 28 such cases, and several others are to be found among the records of the National Hospital.

Treatment.—In the acute stage, the patient should be kept at rest upon a soft bed and fed upon a diet suitable to the febrile condition. Since the malady is an infectious, specific fever, and since the virus is known to exist upon the nasal, buccal and respiratory mucous membranes, and is presumably spread therefrom, bed and utensil isolation is necessary, with sterilisation of any contamination from the mucous membranes and mild daily disinfection of the mouth and nose. Salicylates, especially in the form of aspirin, will relieve the pain and fever, and seem to be decidedly beneficial. If pain be very severe there is no contra-indication to the use of morphine. If the respiratory muscles are seriously involved, belladonna or atropine is of great service both in stimulating the respiratory mechanism and in checking accumulation of bronchial secretions.

In cases in which the respiratory musculature is involved and in which a fatal issue may for this reason ensue, apparatus has been devised to effect an artificial respiration in the hope that the paralysis may recede and the respiratory musculature resume its function. It is particularly during epidemic outbreaks that such cases are seen, the paralysis progressively ascending from the lower limbs, or spreading by "jumps" at short intervals. There are two main types of apparatus, the Drinker respirator and its derivatives which consists of a closed compartment in which the patient lies recumbent, his head protruding through a rubber collar. A motor then produces alternating air pressures which passively move the chest. Another type, the Bragg-Paull respirator, consists of a rubber apparatus strapped round the chest which, again by the use of a motor, by alternate inflation and emptying, moves the chest. There may be a few cases in which a really good result can be achieved, namely, a useful measure of general muscular recovery ensues and the patient can take up a more or less active life. More numerous, however, are those in which although sufficient active thoracic movement to sustain life returns, the patient remains bedridden for life. Cases are on record also in which survival depends upon permanent retention in a Drinker respirator. It is probable, therefore, that such machines will find their greatest field of usefulness in other maladies than poliomyelitis, such, for example, as carbon monoxide poisoning and other essentially temporary causes of respiratory weakness. A modification of the Drinker machine is now provided in most hospitals throughout the country.

It has recently been claimed by Contat in Switzerland that heavy dosage with potassium chlorate during the pre-paralytic stage of the illness averts the development of paralysis. It is known that the virus of poliomyelitis is highly sensitive to oxidising agents. The patient is given nasal instillations of 2 per cent. solution of the substance, 5 drops four times daily. By mouth the dose ranges from 5 grains in the 24 hours in an infant to a total daily dosage of from 60 to 80 grains in an adult. This total is spread over frequent small doses during the 24-hour period. The administration is begun as early as possible and continued during the febrile period (2 to 3 days), and progressively diminished to cease on the fifth or sixth day. It is said that no albumin, red cells or casts are found in the urine during this medication. Contat also employed potassium chlorate as a prophylactic in contacts.

Serum therapy.—Since Netter first initiated this method of treatment some twenty-five years ago, the administration of the serum of individuals who had recovered from a known attack of poliomyelitis ("convalescent serum") has been a widely used method of treatment. Convalescent serum has been experimentally found to contain protective antibodies, and it has been thought that it may prevent the development of paralysis if administered during the preparalytic stage, and may limit the extent and severity of paralysis if given early in the paralytic stage. Recent re-assessment of the clinical evidence strongly suggests that these beliefs are illusory, and that serum therapy is unavailing at any stage of the malady. This result is indeed what the experimental evidence should have led us to expect. For Flexner found that the administration of convalescent serum to an inoculated monkey protected only if it were given at the same times as the dose of virus, or within 12 hours and *before* the appearance of symptoms. Administered after this moment, it was uniformly ineffective. But it is only after the

appearance of symptoms that it can be given in the human subject. Again, the theory adopted to rationalize serum therapy was that poliomyelitis was a general infection, with later involvement of the nervous system. Serum administered in the preparalytic stage was thought to prevent invasion of the nervous system. We know now that the nervous system has already been invaded before any symptoms whatever develop, and that this supposed prevention is from the nature of things quite impossible. Recent controlled observations in New York, in which 50 per cent. of the patients in an extensive epidemic were given serum and the remaining cases denied it, have indicated that serum therapy has no influence upon the course of the individual case.

Kleinschmidt, in his analysis of the 1938 epidemic in Cologne, adds further confirmation to this judgment.

Rest and posture.—It is all-important to secure as complete physiological rest as is possible for the weak or paralysed muscles for some time after the onset. Even in the slightest cases, the patient should be kept in bed for at least 3 weeks, during which time attempts at volitional movements should be discouraged. The posture of the paralysed region should be such as to secure the relaxation of the paralysed muscles; for if they are kept stretched by the action of opponent muscles which are not paralysed, recovery is greatly hindered. Appropriate postures can be secured by pillows, sandbags, splints and other devices. After a few weeks have elapsed, massage and passive movements should be regularly employed and re-educational exercises commenced, where there is sufficient power. Electrical treatment in any form is of very doubtful value. Re-education should be assisted by every appropriate mechanical device, but it must be carefully borne in mind that every mechanical apparatus which overweights the weak limb places a millstone around the neck of recovery. The lightest possible shoes should be worn, and if splints are indicated the excellent and almost weightless, moulded, celluloid splints should be employed, to the absolute exclusion of all heavier varieties. In the re-education of the legs for walking, a walking-machine on wheels is a necessity. Contractures and deformities, which hinder useful action, should be dealt with by passive movements, splinting, tenotomies and other surgical procedures.

There have recently been recorded several cases of acute bulbar poliomyelitis in children upon whom tonsillectomy and adenoidectomy had been performed during the course of a local outbreak of poliomyelitis. It appears that this operation opens a port of entry to the virus. The onset of the disease is at an interval of 10 to 20 days after operation, and the issue is commonly fatal. The performance of these operative procedures upon children is therefore strongly contra-indicated during outbreaks of poliomyelitis.

LETHARGIC ENCEPHALITIS

Synonym.—Epidemic Encephalitis.

Definition.—An acute febrile disease, occurring sporadically and epidemically, due to the infection of the nervous system, presumably from the nasal passages and by a purely axonic route, by a virus, which can be inoculated into the nervous system of monkeys, reproducing the disease. The

clinical aspect is that of a lasting, as opposed to an evanescent, infection, producing chiefly inflammatory reaction, and principally incident upon the upper parts of the nervous system, the cerebrum, basal ganglia and brain stem. Though very definite, it is remarkably polymorphic, and it is sometimes mono-symptomatic, and its type has changed greatly during the passage of an epidemic. The absence of evidence forthcoming of case-to-case infection has necessitated the assumption that infection is transferred by carriers, or by those in the pre-symptomatic stage of infection only.

History.—When we read of the influenza epidemic which swept over Europe in 1580 and which was accompanied by a malady so peculiar as to gain the title of “*schlafkrankheit*,” and afterwards of the epidemic described by Sydenham in 1675 as “*febris comatosa*,” the “sleeping sickness” of Tübingen in 1712 and Dubini’s epidemic of the fatal “electrical chorea” in Northern Italy in 1846, we cannot but agree with von Economo’s conclusion that these epidemics were epidemics of lethargic encephalitis. The subsequent epidemics of Mauthner’s “*Nona*” in Piedmont in 1891, and also Pfuhl-Leichtenstern’s “*hæmorrhagic encephalitis*” in 1905 have been proved identical with lethargic encephalitis, both clinically and pathologically. The malady became pandemic from 1917, reaching a maximum in 1920, since when it has gradually declined and it is now comparatively rare, if indeed any true cases occur. We have, however, seen a good many end-results of cases which had their commencement from 1910 onwards, showing that in England this malady was increasingly present, though unrecognised.

Ætiology.—During the period of its incidence, the disease occurred both sporadically and epidemically, with no centre of spread. It was more prevalent in the cold season of the year. No age is exempt from the malady, and cases have occurred in the seventh decade of life, but it is rare in young children and seems to be most incident in the first half of adult life. Infection presumably takes place, as in poliomyelitis, from human vectors alone, and by droplet infection. When once the virus has gained access to the nervous system by a peripheral axonic route, it is “*virus en cage*,” to use Economo’s term. It is imprisoned within the nervous system and cannot get out, but it may there survive for very long periods, giving rise to second and third attacks after apparent recovery, or to exacerbations of symptoms after long intervals of remission, or to insidious and progressive severe abrogation of nervous function long years in train of slight trivial and evanescent symptoms which marked the epoch of infection. Whether the infection thus pent up in the nervous system does on occasion manage to escape from the peripheral nerve terminals, as it does regularly into the saliva in the case of rabies and often into the skin in the case of herpes, and so get free, has not been determined.

The height of the epidemic incidence of lethargic encephalitis has many times coincided with a severe epidemic of influenza, but no further connection between the two conditions is known. Claimed at one time as an aberrant form of poliomyelitis infection, von Economo’s disease has proved quite distinct, both in its age incidence, seasonal prevalence, morbid anatomy and symptomatology. Economo first succeeded in transferring the disease to the monkey by intracerebral inoculation in 1916, and Loewe and Straus first proved that the infective agent was filtrable.

Pathology.—The pressure and quantity of the cerebro-spinal fluid are

always increased, and in a few of the cases blood or the products of hæmorrhage are present. In a third of our cases the cell count has been normal. In the rest there has been a moderate lymphocytic pleocytosis, with little or no protein increase, the titre of the sugar tending to a high normal and that of the chlorides being normal. No prognostic indications can be derived from the nature of the fluid. The vessels of the brain are markedly congested and full of blood, and the colour shows a characteristic change from the normal throughout the whole of the grey matter, varying from a rosy flush to a deep salmon-pink, giving rise to the term "the rose-coloured brain." When hardened in formalin, this colour becomes a heavy purple grey. Both subdural and deeply seated hæmorrhages are occasionally found. Economo describes the anatomical picture as one of unvarying constancy. It is that of an oedematous and congested brain, with all the grey matter conspicuously reddened in contrast to the white matter, which is of normal colour. There is a non-purulent and, properly speaking, a non-hæmorrhagic inflammation of the whole grey matter exclusively, the white matter being uninvolved. There is most conspicuous perivascular lymphocytic cuffing remarkable for the absence of any polymorphs, with an intense cellular infiltration of the grey matter with elements of the microglia, while the neuroglia is unaltered and demyelination does not occur. Accompanying and succeeding these inflammatory changes is a certain measure of neuronophagia, with primary loss of the ganglion cells.

Symptoms.—In the acute forms of the malady the onset is often ushered in by general symptoms, such as shivering, malaise, headache, and fever and bodily pains, a characteristic thickly coated white tongue and constipation, and sometimes vomiting and persistent hiccough. This train of symptoms usually appears in the story as an attack of "influenza." The pyrexia does not usually last longer than a week. Countless such attacks of "influenza," distinguishable only by the occurrence of transient diplopia, or of slight somnolence, and often even without any such distinguishing features, have been completely recovered from at the time, but have been followed, after long intervals, by the slow onset of the Parkinsonism of lethargic encephalitis. Again, the epoch of infection may apparently give rise to no symptoms at all, and long afterwards an insidious onset of Parkinsonism ensues, as has happened nowadays in many of the examples of Parkinsonism in childhood.

So many and varied may be the clinical aspects of this disease that it is useful to consider the separation of clinical types which Economo has laid down :

A. Acute Types.

- (1) The somnolent and ophthalmoplegic type.
- (2) The hyperkinetic type. Spontaneous involuntary movements, sleeplessness, great mental unrest, delirium and mania are here characteristic.
- (3) The amyostatic and hyperkinetic type. In this type Parkinsonian tremor and rigidity, salivation and the greasy face are conspicuous.
- (4) The cerebellar type. The symptomatology is that of the cerebellum, and recovery usually occurs.
- (5) The bulbar type.
- (6) The ophthalmoplegic type.

- (7) The neuritic type, which simulates acute fibrositis.
- (8) The mono-symptomatic type :
 - (a) Characterised by persistent trismus.
 - (b) Characterised by persistent hiccough.

B. Chronic Types.

- (1) The progressive Parkinsonian type.
- (2) The mental type.

A combination of all the types is very common.

THE NERVOUS SIGNS.—*Mental symptoms.*—An increasing lethargy, which often becomes very deep, is present in many of the cases. In this condition the patient will lie for days without stirring a muscle, taking no heed of his surroundings and passing the dejecta under him unheeding. Yet when roused by command and vigorous bodily stirring, he will wake up and hold a very intelligent conversation, lapsing back at once when he is left alone, even though his mouth be half full of unswallowed food. In this condition, *flexibilitas cerea* may often be demonstrated in the limbs. The lethargy may last for three weeks or longer even in patients who completely recover. It passes away gradually. Unrousable coma is invariably a sign of impending dissolution. Subsequent memory of events during the early days of the lethargy may be remarkably retained. Insomnia may be a troublesome early symptom, and even when the patients are markedly lethargic they will complain that they cannot sleep. Lethargy, however, may be completely absent and the early mental state be that of vivacious excitement and talkativeness. Irritability and restlessness may be present. In some cases the first nervous sign may be delirium or mental aberration, which may rapidly develop into acute and violent mania ; such cases are rapidly fatal. In cases which recover after severe symptoms, considerable mental reduction and self-obvious mental change may persist, but we have not seen any case in which insanity has resulted. Indeed, it has been said that no sufferer from this disease ever regains his original mentality, and it is a common experience to find personality very seriously changed in the way of mental reduction. Complete incapacity for any sustained work, entire change of character, anti-social tendencies, moral perversion and depressed neurasthenic states are not uncommon sequels of the disease. (See also page 1833.)

Convulsions are very rare, but they may undoubtedly occur as in other forms of encephalitis. Indeed, the initial clinical picture may be dominated by convulsion, and closely resemble "status epilepticus" from other causes.

Ophthalmoplegia and other paralyses in the region of the cranial nerves are most often nuclear in type, but peripheral paralysis of any cranial nerve may be met with, most commonly unilateral paralysis of the facial nerve. The pupils may show every abnormality which a lesion of the nervous system can produce. Inequality, unroundness, eccentricity and loss of light reflex and ciliary paralysis may occur. The loss of light reflex may be unilateral. The external ophthalmoplegia, being nuclear in origin, involves both eyes in terms of their conjugate movements, and the upward and downward movements are as a rule more severely impaired than are the lateral movements. Bilateral ptosis is very usual, and is a most important and valuable early indication of the disease. The common error is to consider it part of the sleepy state. The nuclear ophthalmoplegia is often irregular, giving

rise to strabismus and diplopia. Either in addition to the above or existing alone, there may be peripheral paralysis of any of the oculo-motor nerve trunks. The degree of the ophthalmoplegia varies in different cases from slight diplopia with hardly noticeable strabismus to complete paralysis of both eyes. It may be rapidly transient or permanently severe. In severe cases which survive there is always some improvement in the degree of paralysis in the course of time.

Vision.—The diplopia and loss of accommodation cause much defect of vision, but many of the patients complain of a loss of vision in each eye, which is too great for any such explanation, the cause of which is not yet explicable. Papilloedema has been reported in a few cases, in one of which at least meningeal hæmorrhage had occurred. It is transient and never reaches a high degree.

Oculogyric crises.—This term is applied to recurring attacks of tonic conjugate deviation of the eyes, most commonly upwards, sometimes upwards and to one side, less commonly downwards, or downwards and to one side. We have observed one case of a child in which the oculogyric spasm always proceeded to a torsion spasm of neck, trunk and limbs so severe as to roll the patient out of bed on to the ground with each access. The spasm is met with in the chronic stage of the malady, and there is always some degree of Parkinsonism. The attacks may last from a few seconds to 4 hours or more, and may occur frequently or at intervals of several days, and as the eyes are commonly fixed in an upward direction, they are peculiarly incapacitating. They have not been reported in any other disease than lethargic encephalitis, and may constitute, with some slight facial Parkinsonism, the sole sequels of this malady. We have not found any treatment which influences the frequency or severity of the attacks.

Bilateral nuclear facial paralysis and bulbar paralysis are not uncommon. Paralysis of any individual cranial nerve may occur, and also of any individual spinal root. Such paralysees always completely recover in the course of time.

Symptoms indicative of lesion of the basal ganglia are among the most common features of the disease, and they are often the most persistent. These consist of weakness of movement, rigidity with slowness of movement, and spontaneous involuntary movements. The weakness, rigidity and slowness of movement give rise to a peculiar immobility of facial and bodily expression and movement. The face is mask-like, the neck stiff and the head moved little and slowly, the trunk bent forward and stiff, the arms held away from the trunk, the whole appearance of the patient closely resembling that of paralysis agitans. Rapid fluttering of the eyelids when gently closed is characteristic of this condition. The spontaneous involuntary movements may be of a rhythmic tremulous nature, as in paralysis agitans, or there may be slow rhythmic, choreiform, athetoid, myoclonic, irregular or highly complicated movements: these may be met with at any stage of the malady, but most commonly appear some little time after the acute stage has passed away. Fibrillation and fascicular twitching of the muscles is very common in the acute stage. In cases where bulbar symptoms, either of a spastic or flaccid kind, are present, hypersalivation of the nature of a true sialorrhœa is often a most troublesome, though transient, symptom.

In addition to the above common symptoms and signs, other indications of involvement of the cerebral hemispheres may occur. Bilateral spasticity

with signs of involvement of the pyramidal systems, increased jerks, lost abdominal reflexes and extensor plantar responses are common. Hemiplegia, aphasia and hemianopia may occur, presumably as the result of local sub-cortical hæmorrhages. Meningeal symptoms may be very marked in the early stages, such as suboccipital headache, painful stiffness of the neck, head retraction, vomiting and Kernig's sign. Indeed, we have seen rapidly fatal cases in which the clinical picture throughout was hardly distinguishable from that of acute meningitis, but none of these cases showed any leucocytosis in the cerebro-spinal fluid. A major incidence of the lesions upon the cerebellum gives rise to the picture of acute cerebellar ataxy following a lethargic onset, and the end-result may be a condition closely resembling a usual type of disseminate sclerosis. Such cases make a good recovery in the course of time.

Peripheral pains are sometimes very severe and are usually quite local. They may be the first signs of the illness, and several of our patients had been treated for trigeminal neuralgia, brachial neuritis or sciatica before any other sign of the malady appeared. These pains may persist for months after recovery. They are obviously due to the lesion around the nerve roots which has been already referred to.

Spinal symptoms.—Since the lesions have been found in the spinal cord, it is only to be expected that focal spinal lesions should be met with in rare cases. These are usually acute atrophic paralyses similar to those of poliomyelitis. Those that we have seen have completely recovered. It has been argued, however, that this atrophic palsy is due to a lesion of the spinal roots. More severe lesions may apparently give rise to a condition resembling acute transverse myelitis.

Sphincters.—The incontinence which is almost constantly present, even when the lethargy is far from deep, is the result of the lethargy. Transient conscious dysuria is however not infrequent in the early stages of the disease. The deep reflexes may be lost in severe cases during the acute stages, and they are usually absent in premortal conditions. Otherwise they tend to be exaggerated, especially if involvement of the pyramidal system be present. The condition of the abdominal and plantar reflexes depends upon the presence or absence of lesions affecting the pyramidal tracts. In the former case, the abdominal reflexes will be absent and the plantar reflexes of the extensor type.

Attention must be drawn to a group of cases in which the initial manifestations of the disease are so slight as not even to interfere with the daily work or to call for medical attention, and yet in the course of months, or it may be even years, the most serious and completely incapacitating paralysis appears. A patient of ours noticed that he saw double, and did not feel very well for a few weeks. He recovered, but, two years later, had to give up work, by reason of a slowly oncoming Parkinsonism, which became extreme. A similar result in the slow and late development of grievous symptoms may follow any attack of lethargic encephalitis and make the prognosis in this malady very difficult.

Sequelæ.—The disabilities which this malady may leave in its wake seem endless and ever increasing as clinical experience widens. The mental, paralytic and Parkinsonian end-results have already been referred to, but special mention must be made of involuntary spontaneous movements,

recurring rhythmic movements, spasms and altered respiratory rhythm. Ceaseless rhythmic pulsatile movements may occur in any muscle, movements like those of convulsive tic may incapacitate the patients. Hideous recurring spasms may appear, sometimes local, sometimes general. Torticollis may occur. An unduly rapid respiratory rhythm may be established. (See also p. 1705.)

Course.—The course of the disease is extremely variable. It may be a slight transient illness lasting but a few days, and leaving no sequelæ after a few weeks; or a most malignant disease, fatal in a few days. In others, symptoms indicative of fresh lesions may occur repeatedly weeks and even months after the onset.

Diagnosis.—A diagnosis of lethargic encephalitis is even at this date (1941) not rarely made, but must be received with the very greatest reserve. Under this title most neurologists have encountered a great variety of nervous disease, including intracranial tumour, cerebral abscess, subdural hæmatoma, tuberculous meningitis, and the like. In typical cases the diagnosis presents no difficulty, the rousable lethargy, incontinence, ophthalmoplegia and negative, lymphocytic, or blood-containing cerebro-spinal fluid being so characteristic as to preclude possibility of error. The less usual forms of the malady, and especially those with very gradual onset and slight symptoms, often present great difficulty and require much care and full knowledge of the possible symptomatology of the disease for their recognition. There is no specific laboratory test for the malady, and the diagnosis must be based upon clinical grounds. Where meningeal symptoms are prominent, distinction has to be made from other forms of meningitis and from poliomyelitis. Here, the cerebro-spinal fluid is of the highest importance, as no polymorpho-nuclear leucocytes occur in lethargic encephalitis. In cases commencing with peripheral pains, excitement, maniacal symptoms or convulsions, careful look-out should be kept for the advent of ptosis, ophthalmoplegia, or lethargy, the appearance of which, following such symptoms, should at once suggest the diagnosis. It must be borne in mind that the clinical picture of the disease may be dominated by a hemiplegic condition, and that an apoplexy may occur during the acute stage of the disease. Slight cases of the disease are frequently unrecognised, or are indeed unrecognisable in the early stages, but here the diagnosis can often be made with certainty from the end-results; the peculiar ophthalmoplegia, the spontaneous involuntary movements, and the paralysis agitans-like syndrome being almost pathognomonic of the malady.

Prognosis.—A rapid onset and quick development of severe symptoms, marked pyrexia, delirium and maniacal excitement are bad prognostic signs and indicate a rapidly fatal issue. After the third week of the disease, the probabilities are all in favour of survival. The prognosis, however, as to how much permanent damage to the nervous system will eventually remain is hardly possible, since slow improvement may go on for months and even years. Of the acute cases occurring at the height of an epidemic, 40 per cent. are quickly fatal, 30 per cent. are reduced to chronic invalidism, and 30 per cent. recover completely (Economo). The spontaneous movements, even when very marked, may clear up in from 3 months to a year. The weakness, rigidity and tremors, which form the paralysis agitans-like picture in many of the cases, persist indefinitely.

Treatment.—Nothing being known of the infectivity and mode of spread of the disease, isolation and disinfection are not usually employed. Each case must in England be immediately notified to the public health authorities. No treatment is known which has any specific influence upon the disease. Intravenous injection of collosol iodine solution (150 c.c. for a dose), repeated on the second and fourth days, has been advocated, and is certainly without harmful effects. Intravenous sodium salicylate, in 15-grain doses in normal saline daily, certainly seems to clear up the symptoms in some cases and may do permanent good. It remains therefore to use those measures which will help to keep the patient alive and those which relieve symptoms. Relief of the constipation is most important and is often followed by striking improvement in the symptoms. After the acute stage, treatment is concerned with combating the physical and mental listlessness and depression, removing the rigidity with massage, passive movements and exercise, and withal brightening the days of a convalescence which is often long, tedious and hard to bear.

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W. J. ADIE.

Revised by F. M. R. WALSH.

HERPES ZOSTER

Synonym.—Zoster; Shingles.

Definition.—An acute infection of the posterior root ganglion, probably by a neurotropic virus, leading to severe pain in the distribution of the corresponding posterior root, and to the appearance of a crop of vesicles in the cutaneous distribution of the root.

Ætiology.—The virus of zoster stands in some as yet undetermined relation to that of chicken-pox, and the appearance of the latter malady in a susceptible subject some 14 days after contact with a case of zoster has been too frequently recorded to be of the nature of coincidence.

The disease is seen at all ages, but according to Head is perhaps most common in adolescents. In elderly patients it is frequently a more serious as well as a more painful affection than in young persons. It may arise without discoverable cause and with a febrile reaction and considerable malaise. It may also occur apparently "symptomatically" during the course of arsenical medication, or during such illnesses as pneumonia, tabes dorsalis and tuberculosis.

Pathology.—The essential lesion is an acute inflammation of the dorsal root ganglion of the same histological character as the lesion of acute anterior poliomyelitis. There are degenerative nerve cell changes, with accompanying microglial reaction and perivascular infiltration with round cells. Later, degenerative changes occur in the fibres of the dorsal roots and of the peripheral sensory nerves. The Gasserian ganglion and the thoracic and upper two or three lumbar ganglia are most often affected. There is an increased protein and lymphocyte count in the cerebro-spinal fluid.

Symptoms.—There may be an onset with fever which persists for 2, 3 or even 4 days. There is from the first pain at the place at which later

the herpetic eruption is to appear. This occurs on the third or fourth day of the illness. At first the rash is a patchy erythema, upon which appear small vesicles filled with clear fluid. From the fifth to the tenth day the vesicles dry up and shrink progressively until a scab is formed. This finally drops off, sometimes leaving considerable scarring. These scars may be anæsthetic to touch, pinprick and temperature sense. The pain before and during the evolution of the cutaneous lesion may be intense. It is of a burning and itching quality, and in frail and elderly persons it may persist as a most intractable post-herpetic neuralgia for months or even years.

Zoster of the ophthalmic division of the fifth nerve is most commonly found in elderly persons. Corneal vesicles may form and burst, giving rise to ulcers, which may spread and end in residual scarring (nebulæ), which impairs vision.

Herpes of the geniculate ganglion occasionally occurs. The vesicles are found in the pinna, and there is pain in this region, over the mastoid, and sometimes in the fauces (see p. 1512).

Localised paralysis may accompany herpes. Thus, in ophthalmic herpes there is occasionally third-nerve palsy, with ptosis and squint. In geniculate herpes, facial palsy and loss of taste over the anterior two-thirds of the tongue is the rule. In herpes of the lower thoracic ganglia there may be paralysis of the oblique abdominal muscles on the affected side. The marked local bulging of the abdominal wall which ensues resembles at first sight the presence of an abdominal tumour. These paralysees do not invariably clear up, though the facial palsy of geniculate herpes does so more frequently than the paralysis of the abdominal muscles.

Treatment.—The course of the cutaneous lesions is not influenced by treatment, which is directed to keeping the vesicles dry and free from infection. For this purpose a dusting-powder of starch or zinc oxide, or a collodion dressing may be used.

During the acute stage, pain may be relieved by aspirin or phenacetin, but the post-herpetic neuralgia of some elderly and debilitated patients may prove intractable, and so severe as to render life scarcely tolerable. A neurosis may be grafted on this pain and render the situation extremely difficult for the doctor. Various local remedies have been recommended, including rays of all kinds, local heat, electrical currents, analgesic applications, and so on. But none can be relied upon to afford material relief, though the neurotic patient may obtain some comfort from any of them that commands his faith. It may be necessary to keep the patient under some such mixture as the following: tinct. gelsemi min. 10, phenazone grs. 10, phenobarbitone. sol. gr. $\frac{1}{2}$, sod. bromid. grs. 10. Aq. chlorof. ad fl. oz. 1 t. d. s.

A warning may be sounded against prolonged bromide medication for elderly subjects. It sometimes renders them confused and feeble, and these debilitating effects may take some weeks to pass off even after bromide is withdrawn from administration. For such patients, small and carefully adjusted doses of phenobarbitone are probably better.

RABIES

Synonyms.—Hydrophobia ; Lyssa.

Definition.—This is an infective disease due to a filtrable virus which is located in the salivary glands and central nervous system. It is transmitted to man and most warm-blooded animals through infective saliva of canines or blood-lapping bats. There is a long and variable incubation period, and a short pyrexial illness of sudden onset characterised by fever, nervous exaltation and violent muscular spasms involving the œsophagus and respiratory system. Once symptoms have supervened, the patient invariably succumbs.

Ætiology.—The disease is generally transmitted either by the licking of a freshly abraded surface of skin or the bite of an infected dog. In Eastern Europe and the Orient, wolves not uncommonly transmit the disease and, owing to extensive laceration of the tissues, a greater proportion of people bitten by them develop the disease than with either dogs or jackals. It has been estimated that wolf bites entail a mortality of 80 per cent. In Trinidad, in 1925 an epidemic of paralytic rabies in man was attributed to the bites of vampire bats, cattle being the original source of infection.

The virus of rabies.—This is now known to belong to the class of neurotropic viruses that have a special affinity for attacking the grey matter of the nervous system. To this class belong also the viruses of acute anterior poliomyelitis and of Borna disease.

Pasteur, in 1881, discovered that rabies could be transferred in series from animal to animal by subdural inoculation of emulsions of central nervous tissue derived from an infected dog. In rabbits, after some twenty passages, the virus became modified : firstly, the incubation period of ordinary street virus which varied from 8 to 60 days was reduced to 7 days ; and, secondly, it lost its capacity to reproduce the disease on subcutaneous inoculation. Such a virus is known as fixed virus or virus fixé. Street virus, on the other hand, is transmitted from the local wound via the peripheral nerves to the central nervous system, and if the sciatic nerve be inoculated the lumbar cord becomes infectious several days before the virus can be demonstrated in the brain (di Vestea and Zagari). This accounts for the fact that cases bitten about the face, head and neck have such a short incubation period. The salivary virus is filtrable through coarse Chamberland and Berkefeld filters—not so, emulsions of infective brain tissue. The virus is destroyed at 50° C. and is attenuated by drying—a fact made use of in the preparation of anti-rabic vaccine by the Pasteur method.

Pathology.—Excess of cerebro-spinal fluid, petechial hæmorrhages of the piaarachnoid and injection of its vessels may be found at autopsy. Histological examination reveals cellular infiltration of the perivascular lymph spaces as well as Negri bodies within the cytoplasm of the nerve cells and their processes. These bodies were described by Negri in 1903. They are globular or ovoid structures, of variable diameter (0·5–25·0 microns), and are especially common in the Purkinje cells of the cerebellum and the hippocampus. Though demonstrable in the brain of 97 per cent. of dogs infected with street virus, they never appear in the salivary glands—a fact which supports the view that they are not parasitic protozoa, as Negri suggested, but rather some kind of cell inclusion or degenerative structure.

Symptoms.—The period intervening between the bite and the clinical

manifestations varies from 1 to 2 months as a rule, the limits being 11 days to over a year. Face, head and neck bites have a shorter incubation period than those on the upper extremity, and arm bites a shorter incubation than those implicating the leg. The onset is generally sudden, but prodromal symptoms are sometimes noted for a day or two before a hydrophobic syndrome appears. For convenience, three stages are described.

1. *The invasion stage.*—This includes prodromal features such as pain in the scar, fever, headache, rapid pulse, anxiety, restlessless, insomnia, irregular and sighing respirations, and phases of rushed speaking.

2. *The stage of excitation.*—This supervenes in 24 to 48 hours. There is intense restlessness, mental excitement, hyperæsthesia and hydrophobia which consists of a sudden spasmodic spasm of the muscles of the mouth, pharynx and larynx and, to a greater or lesser degree, the whole respiratory musculature. A typical attack may be induced by offering the patient water. As the glass approaches the mouth, the head retracts in a series of spasmodic jerks associated with gasping respirations, while any water reaching the mouth is immediately ejected. The shoulders are elevated, the chest expanded, and the sterno-mastoid and platysma muscles contracted. Later, the synaptic resistance in the reflex arcs becomes so lowered that a variety of sensory stimuli such as a sudden sound, cold air, strong light, a strange smell, and even the suggestion of water may suffice to induce the attack. The voice is altered. Frothy saliva collects in the throat and mouth and is flung off the lips during the attacks which may be characterised by intense fury or the most profound terror. Later, opisthotonus and general respiratory spasm are superadded. In the interval the mind is clear, the patient remaining quietly at rest in bed. Examination of the central nervous system reveals, as a rule, nothing more than increased deep reflexes. Glycosuria is not uncommon, and vomiting, exhaustion and emaciation characterise the final stage of the illness. Death during the paroxysm may occur from dilatation of the right heart, though sometimes near the end the spasms ameliorate or cease altogether.

3. *Stage of paralysis.*—If the patient survives long enough, paralysis of various types, including Landry's ascending paralysis, paraplegia and hemiplegia, may supervene. The patient lies helpless and exhausted, and generally dies in coma. In man this stage is rarely seen in canine-transmitted rabies, but paralytic rabies is commonly encountered in the bat-transmitted variety in Trinidad.

In the Trinidad outbreak all the cases were of this variety, and all proved fatal. The onset is acute, with fever and headache. Numbness and burning sensations in one or both legs, paresis of the legs and retention of urine follow. After 2 or 3 days the paraplegia becomes more complete, and the plantar and tendon reflexes disappear. One limb is commonly affected before the other. In a few days the paralysis begins to ascend, involving the muscles of respiration, of articulation and deglutition. There is dyspnoea, restlessness and death. The sufferer remains conscious, but may be delirious. Sensory changes are of variable intensity. A final brief coma precedes the fatal issue. During this time the temperature swings round 103° F., and there is profuse sweating. Hydrophobic symptoms are exceptional, and when present slight. The cerebrospinal fluid yields an increased globulin content, but is otherwise normal. The duration of the illness is from 4 to 8 days.

Rabies in the dog.—These animals never show the hydrophobic syndrome

observed in man. The earliest manifestation appears to be a change in temperament, followed by irritation and exacerbations of vicious fury in which the animal runs amok, biting wildly anything in its path. Later, swallowing becomes difficult, the bark is altered, the jaw drops and general paralysis ensues. Death invariably follows some 2 to 5 days after the first symptoms appear. In dumb rabies the stage of excitation is absent.

Diagnosis.—As a rule, little difficulty is experienced in diagnosis, but occasionally tetanus, the cerebral type of typhus fever, bulbar paralysis from any cause, and datura and other poisonings encountered in Oriental countries may need differentiation. Lyssophobia or hysteroid counterfeiting of the disease generally manifests itself within the first 10 days, and is unaccompanied by fever or other serious features.

Prognosis.—By no means all patients bitten by rabid animals die, but once clinical manifestations appear the disease invariably ends fatally. Estimates varying from 5 to 33 per cent. have been made of the death-rate in untreated patients, but of those receiving early anti-rabic inoculations in Pasteur institutes, not more than 1 per cent. die. The mortality varies with the site of the bite, the interposition of clothing, the number of tooth-marks, the extent of tissue laceration and the rapidity with which efficient local treatment has been instituted. Head, face and neck bites are particularly dangerous, as well as bites from wolves and jackals.

Treatment.—This is entirely preventive, and in England the muzzling order and the strict quarantine of all imported dogs has led to the eradication of rabies. In endemic areas canine bites should be promptly treated, and the suspected dog chained up, muzzled, and kept under observation. Should the animal be alive at the end of 10 days it is proof that the bitten person has not been infected. This rule, universally followed in Pasteur institutes, is based (1) on the knowledge that the infected dog never survives longer than 6 days from the onset of its illness, and (2) that the saliva of a rabid dog is never infective for more than 4 days before the onset of symptoms. In suspicious cases, especially the head, face and neck bites, treatment should be commenced without delay and discontinued if the dog survives.

The virus of rabies differs from that of yellow fever in not passing through the intact skin, and where there is a history of being licked by an animal suspected of rabies prophylactic inoculation need not be advised unless fresh skin abrasions were present at the time.

Local treatment.—If seen within 30 minutes, bleeding should be encouraged by the application of a ligature just tight enough to obstruct the venous return and the parts bathed with permanganate solution. Subsequently, each tooth-mark should be probed separately and cauterised or treated with pure phenol. For 3 days the wound should not be sutured; this particularly applies in the case of face bites.

Anti-rabic vaccination.—Owing to the long incubation period, it is feasible to attempt immunising the patient either by the inoculation of attenuated, living, fixed virus, as in the Pasteur and Högyes methods, or by the injection of carbolised or etherised vaccines in which the fixed virus has been killed. The Pasteur treatment consists of a series of 18 injections of emulsions made from the spinal cord of rabbits which had been dried for periods of from 14 to 35 days. Semple introduced carbolised vaccine; the most potent preparation consists of a 5 per cent. carbolised suspension of sheep brain

infected with Paris virus. In mild cases the course consists of 2 c.c. injected subcutaneously each day for 7 days; in average cases of 5 c.c. for 14 days; and in severe cases, such as head, neck and face bites, in wolf bites, or in children bitten on the bare skin, 10 c.c. are injected daily for 14 days. Itchy swellings may appear at the site of the inoculations about the eleventh day, but other complications following inoculation are fortunately rare. Paralytic accidents, however, have been recorded with all methods; they include a mild facial neuritis, dorso-lumbar myelitis and an ascending paralysis of Landry's type which is fatal in about 30 per cent. of cases.

Treatment of the paroxysm.—No specific treatment is known. Measures directed to alleviate the suffering of the patient should be instituted. These include chloroform inhalations and morphine, hyoscine, chloral and atropine in large doses. Curare and tetrado-toxin have both been employed for the relief of spasms.

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CEREBRAL VASCULAR LESIONS

ANEURYSM

Pathology.—Miliary aneurysms, which are small seed-like dilatations of the finer arteries in the substance of the brain, are not uncommonly present in cerebral arterial degeneration. They are usually multiple. The extensive researches of A. G. Ellis in 1909 and of Pick in 1910 have proved conclusively that miliary aneurysms are of little practical importance apart from the associated arterial degeneration.

True aneurysms of the larger cerebral arteries are not uncommon and have in the past been the subject of a very extensive literature. In a recent analysis of over a thousand cases McDonald and Korb have ascertained the following facts:

(i) The arteries involved in order of frequency are the middle cerebral, basilar, anterior communicating, internal carotid, vertebral, anterior cerebral, posterior communicating and posterior cerebral.

(ii) Aneurysms occur at all ages; about 11 per cent. were below 20 years, 35 per cent. from 20 to 40 years and 54 per cent. above 40 years of age.

(iii) The sex incidence is approximately equal.

(iv) 63 per cent. of the vessels examined showed pathological changes, the remainder being described as normal. The predominant lesion was atheroma, and less common in order of frequency were mycotic aneurysm and syphilitic changes, only 5 per cent. showing changes of the last-named order. At all ages pathological changes in the affected arteries predominated, mycotic aneurysms being most common in young persons, atheromatous changes in older subjects.

These aneurysms may give rise to no symptoms during life, being merely post-mortem findings, they may rupture, or they may be of sufficient size to evoke tumour symptoms. Of these possibilities, rupture appears to be the most common, the artery most frequently affected in this way being the anterior communicating.

The aneurysms found on normal arteries are usually regarded as due to developmental defects in the wall, commonly seen at junctions on the circle of Willis. They may be multiple, and from their shape are spoken of as "berry" aneurysms. They may rupture freely, leading to a rapidly fatal issue, or they may leak recurrently. The wall may contain calcified material which shows as a ring shadow in the radiogram. Mycotic aneurysms arise from septic infection of the arterial wall, usually in association with septic endocarditis.

Symptoms.—Clinically, cerebral aneurysms fall into two main groups : (1) Those which rupture during life with the extravasation of blood, in greater or less amount, into the subarachnoid space, and sometimes also into the cerebral substance and into the ventricles. These form the so-called *apoplectic* group. (2) Those which produce symptoms in virtue of the pressure they exert on neighbouring structures, thus giving rise to paralytic symptoms. These form the *paralytic* group. It has to be added that this second group may ultimately rupture. There are also cerebral aneurysms that during life are clinically latent, being merely necropsy findings.

The clinical picture of subarachnoid hæmorrhage, following upon the rupture of cerebral aneurysms is now widely recognised, but that of the paralytic group is only now being clinically differentiated. Recent studies by G. Jefferson has clarified their clinical picture considerably, and in most cases their presence should be diagnosed.

1. SUBARACHNOID HÆMORRHAGE

Synonym.—Spontaneous Subarachnoid Hæmorrhage.

We have seen that bleeding into the subarachnoid space may be an accompaniment of head injuries in which the meninges are torn. It may also follow primary intraventricular hæmorrhage, but the usual cause of uncomplicated, or as it is sometimes called, spontaneous subarachnoid hæmorrhage is rupture of a cerebral aneurysm on the circle of Willis or on one of its component arteries. The situations in which such aneurysms are commonly found have been already enumerated. What has been called here the "berry" aneurysm may rupture suddenly and freely with the production of fatal apoplexy, or there may be recurrent leaking of blood in small amounts from such an aneurysm, leading to a syndrome of meningeal irritation.

1. *The apoplectic syndrome.*—The subject may have been subject to frequent headaches, or the episode may be quite unheralded until a sudden intense headache, rapidly followed by sudden lapse into unconsciousness, signals the free rupture of the aneurysm. It may be thought that an ordinary cerebral hæmorrhage has occurred when the comatose patient is first seen, but careful examination will generally reveal the absence of signs of a gross lesion in one cerebral hemisphere (cf. p. 1527) when this has occurred, whereas in uncomplicated subarachnoid hæmorrhage no signs of hemiplegia are present. On the other hand, a bilateral Babinski plantar response will be obtained and there will be marked neck rigidity. At first both pupils may be small and sluggish, but in fatal cases the pupils ultimately dilate. Lumbar puncture produces a fluid that resembles pure blood.

Recovery from hæmorrhage of this severity is by no means unknown.

In fatal cases death commonly ensues within 24 to 36 hours, or at some time during the first fortnight from fresh bleeding. If this period be safely passed the prognosis as to recovery is good. Some patients enjoy years of normal health after such an episode, while others are subject to recurrences of greater or less severity.

2. *The meningitic syndrome*.—In this case, the hæmorrhage is less abundant and therefore consciousness may not be lost. There is violent headache, restlessness, delirium, rigidity of neck and spine, Kernig's sign, bilateral extensor plantar responses, and sometimes diplopia and squint. Within a few hours, or somewhat later, ophthalmoscopic examination may reveal the presence of flame-shaped hæmorrhages in the nerve fibre layer of the retina, or massive hæmorrhage in the subhyaloid space. The last-named are characteristic of subarachnoid hæmorrhage. Both forms of hæmorrhage arise from the passage forwards of the extravasated blood into the subarachnoid space surrounding the optic nerves. A low grade papilloedema is occasionally also observed.

In non-fatal cases of both types, the temperature may remain raised for 7 or 8 days, and the urine for the first 48 hours only may contain abundant albumin and some sugar. The addition of these features to the clinical picture may, if the possibility of their occurrence is overlooked, lead to an erroneous diagnosis of uræmia or diabetes.

In small leaking hæmorrhages the cerebro-spinal fluid is more or less heavily bloodstained, and may for two or more weeks be discoloured, yellow or brownish according to the amount of blood originally present. As with all other conditions of high and rising intracranial pressure, sudden and unexpected death is a common event in undrained cases. Subarachnoid hæmorrhage does not always reach the general subarachnoid space freely. When effused upon the vertex, it may spread out and clot at its edges and from without, while the bleeding is still going on at its centre. Thus, a pancake-like hæmatoma accumulates upon the surface of the brain, thin and clotted at its edges, which prevents further spread, and still liquid and ever accumulating at its centre. Not infrequently the pressure of the growing liquid centre bursts into the hemisphere causing the addition of a sudden hemiplegia to the syndrome, and this has been named by Froin the "meningo-cerebral hæmorrhage." Draining of the cerebro-spinal fluid gives no relief when this pancake hæmatoma is present.

3. *The lumbago-sciatica syndrome*.—This rare condition, first described by Professor Arthur Hall, commences with pain and stiffness in the lumbar region, followed by pains in the legs, and sometimes the leg jerks are absent. For a week or more there may be no indication that the cause is intracranial, but thereafter in undrained cases the symptoms spread upwards to the arms and neck, and head retraction, headache and vomiting are added. Pyrexia is the rule. The diagnosis depends upon the characteristic cerebro-spinal fluid of subarachnoid hæmorrhage. The explanation of this syndrome is not easy. It may be that deposit of fibrin upon the roots of the lower theca is the cause of the quite local meningeal irritation. All the reported cases that have been treated with regular drainage by lumbar puncture have made good recovery.

4. *The recurring coma syndrome*.—The description of a typical case will best illustrate this condition. A man of 28, during a period of four years,

on four occasions, and at long intervals without any prodromal symptoms, fell unconscious in the street, and on each occasion he was taken to the nearest hospital where, on account of the persistent coma, lumbar puncture was done, with the discovery of blood in fair quantity in the cerebro-spinal fluid. On each occasion, the coma disappeared somewhat suddenly after 24 hours, and the patient insisted on leaving hospital and returning to work within a week, as he felt quite well. This patient came under my observation for the prevention of further attacks. The only abnormality found was a yellow cerebro-spinal fluid resulting from long antecedent hæmorrhage. The presence of leaking aneurysm has been pathologically proved in several similar cases.

Differential Diagnosis.—The recognition of subarachnoid hæmorrhage is an easy matter in those cases in which the train of symptoms calls at once for the examination of the cerebro-spinal fluid and blood is found in that fluid. The distinction of the apoplectic forms from other varieties of cerebral hæmorrhage can only be made: (1) by the age of the patient, practically all hæmorrhagic apoplexy being in the first half of life the result of ruptured aneurysm; and (2) by preceding symptoms, such as headache, diplopia, ophthalmoplegia and migrainous phenomena. In those cases in which blood does not escape into the cerebro-spinal fluid, as in very many of the subdural hæmorrhages, the diagnosis is both difficult and uncertain. The insidious onset of irregular headaches with periods of mental confusion and drowsiness alternating with period of recovery, especially if following a fall or slight blow on the head, should, during the second half of life, always suggest the possibility of subdural hæmorrhage. Adie has suggested that all cases of migraine with transient ophthalmoplegia are due to aneurysms. It seems certain that many of the numerous cases of sudden death occurring in ophthalmoplegic migraine have been the result of terminal hæmorrhage due to the presence of aneurysm. On the other hand, the majority of the cases of migraine with ophthalmoplegia make perfect recovery.

Prognosis.—When the aneurysm ruptures frankly and widely and the bleeding can be free, the outlook is hopeless, and death occurs in from a few minutes to a few hours; nor does drainage avert the consequences of so large an opening into a main arterial trunk. If, as so commonly happens, there is a slower leakage which perhaps is intermittent, the outlook will depend: (1) upon the cessation of the bleeding and the healing of the leak by clotting; and (2) upon the possibility of the free escape of the effused blood into the subarachnoid space and its removal by repeated lumbar drainage. In many of the cases of subarachnoid hæmorrhage, the bleeding ceases and the pressure and the dangerous results therefrom can be well relieved by lumbar drainage, repeated whenever the symptoms demand it, and healing of the aneurysm, by clotting and calcification, occurs with complete recovery. In other cases there may be repeated attacks of leaking at intervals of weeks, months or even years, and again many of such patients make good recovery in the end. When the bleeding is wholly or mainly subdural and when a subarachnoid hæmorrhage clots at its edges upon the surface of the brain, drainage and the relief of symptoms is impossible, and the prognosis is serious in the extreme but for the possibility that the site of the bleeding may be located and the clot turned out and the hæmorrhage arrested by surgical procedures.

Treatment.—In the case of subarachnoid hæmorrhage the patient must be kept absolutely at rest, with the administration of sedatives. An immediate injection of morphia is indicated when the patient is not comatose and has the usual intense headache. It also may be necessary on the recovery of consciousness on account of headache and restlessness to take this step. It is doubtful whether repeated lumbar puncture is advisable, as it may lead to recurrence of hæmorrhage, but if there be signs of raised or of rising intracranial tension (and progressive slowing of the pulse is generally a reliable indication of this), then lumbar puncture may be expedient. When recovery sets in, the patient should still be kept in bed for at least 3 or 4 weeks. During the first fortnight recurrent hæmorrhage is more likely to occur than later during the course of convalescence.

2. UNRUPTURED ANEURYSM OF THE INTERNAL CAROTID ARTERY

Synonym.—The Paralytic Syndrome.

The aneurysms of this group, like the berry aneurysm, mostly occur at the bifurcation of an artery. In this case, on the internal carotid artery where the ophthalmic artery arises, or at the junction of the anterior cerebral and anterior communicating arteries. There seems no doubt that they are more often seen in women than in men, mostly in middle life.

Symptoms.—The onset may be sudden, in which event diagnosis is relatively easy; or the symptoms may develop so gradually as to make a diagnosis of intracranial new growth probable. The arrangement of the circle of Willis and its components is such that the visual pathway (optic nerves, chiasma and tracts) are very commonly affected by the development of an aneurysm in this situation. Similarly, when there is a carotid aneurysm the proximity of the carotid artery to the cavernous sinus commonly leads to ocular palsies.

ANATOMICAL CONSIDERATIONS.—The arrangement of the circle of Willis and its relations to the visual pathway (optic nerves, chiasma and tracts) are such that aneurysms in this situation usually exert pressure upon some part of this pathway and thus give rise to visual defects. Further, the proximity of the internal carotid artery to the cavernous sinus may lead in similar manner to ocular palsies and to disturbances in the field of the trigeminal nerve. The optic tracts pass forward above the posterior communicating arteries, the chiasma lies within the arterial circle, and the optic nerves pass forward below the anterior cerebral and anterior communicating arteries. In other words, the visual pathway in its passage forward passes through the circle Willis from behind and above, and leaves it anteriorly on its under side.

It will be seen, therefore, that there are supra-clinoid and sub-clinoid aneurysms of the carotid trunk. The latter arise in the cavernous sinus, do not commonly interfere with the visual pathway, but may compress the third nerve and the ophthalmic division of the trigeminal nerve. The supra-clinoid aneurysm arise from the carotid where the ophthalmic artery goes off or at the final bifurcation of the main trunk. In these circumstances the visual pathway is generally affected.

Symptoms.—The symptoms may evolve slowly, as though a new growth were in question, or may develop with dramatic suddenness. There may be

paralysis—partial or complete—of the third, fourth and sixth nerves with squint and ptosis, severe pain, and some sensory loss round the eye in the field of the first division of the trigeminal nerve. There may be slight proptosis of the eye, and rarely only some papillœdema. The visual field defects vary from case to case. There may be monocular blindness when the optic nerve alone is compressed, or monocular blindness with a temporal hemianopia in the eye on the side away from the lesion. Other recorded defects are bitemporal and homonymous hemianopia. Pain is a prominent symptom. Symptoms of pituitary dysfunction are usually not found. X-ray examination may reveal a ring calcification of the aneurysm, while angiography may show the shadow of the aneurysm.

Treatment.—Ligature of the carotid artery appears to be the only method of treatment of an unruptured aneurysm of this type.

CHRONIC SUBDURAL HÆMATOMA

Subdural hæmatoma stands in no ætiological relationship to subarachnoid hæmorrhage, but is invariably traumatic in origin, the hæmorrhage occurring from cerebral veins as these traverse the subdural space. Yet it is convenient to consider it at this juncture.

Ætiology.—The condition under consideration was formerly known as pachymeningitis hæmorrhagica, the name expressing the belief that an inflammatory process was in question. It was further supposed that the lesion was in some unexplained way peculiar to chronic alcoholic subjects and to sufferers from general paralysis.

W. Trotter was the first to point out that in reality the lesion was traumatic in origin, and that the frequent absence of a history of injury was what might be expected in the individuals specified above, who are both more than normally liable to falls and head injuries of a minor order and less than normally capable of recalling these injuries. In fact, subdural hæmatoma is found at all ages and is invariably traumatic. Falls on forehead or occiput, not at the time apparently productive of serious injury, may yet lead to a tearing of the cortical veins as these pass from the surface of the brain to enter the dural sinuses. The tear is commonly in the subdural space on one or both sides of the vertex (superior longitudinal surface). When after the shock of the fall the blood pressure rises again to normal and on subsequent occasions when there is a transient rise in this pressure blood leaks from the torn veins and collects in an ovoid mass on each side of the vertex.

Rarely such a subdural collection may be found at the base of the brain. The periphery of the blood clots thus formed tend to organise so that a fine capsule is built up round the hæmatoma. This may remain solid or its centre may liquefy with the formation of a cyst containing a thin brownish fluid. In time the dura over this cyst and the cyst walls become thickened and stained, and it is clear from the condition sometimes found at necropsy that a subdural hæmatoma may be compatible with many years of survival and may during life give rise to no clearly recognizable symptoms. Such hæmatomas may reach a large size, and a 10-ounce mass over each hemisphere has been observed.

Symptoms and Diagnosis.—The difficulty which still frequently sur-

rounds the diagnosis of subdural hæmatoma depends in large measure upon a general unawareness that it is a not uncommon lesion, and from a survival of the old and now obsolete notion that its occurrence is largely confined to sufferers from the two affections named above. It must be emphasised again, therefore, that subdural hæmatoma may follow an apparently trivial head injury in persons at all age periods ; that essentially its symptomatology is that of a space-occupying lesion, with a feature characteristic of hæmatoma : namely, a remarkable fluctuation in the course and severity of the symptoms ; that owing to the bilateral nature of the lesion the signs are apt to be difficult of localising interpretation ; and finally that in the presence of such a somewhat blurred picture of raised intracranial tension a history of head injury some days, weeks, or even months before the onset of symptoms should always give rise to the suspicion that a hæmatoma, and not a new growth, may be present.

There is almost invariably a clinically latent period in the development of a subdural hæmatoma. This may vary from a matter of days to one of weeks. On the whole, it may be said that in young persons, the latent period is apt to be shorter and the symptoms more severe and of more rapid evolution than is commonly the case in elderly subjects. In young subjects, too, there is usually no difficulty in obtaining a history of head injury, either a fall upon the head or a blow sustained at sport or in some other way. The initial symptom is usually headache, fluctuating in intensity, apt to be most severe on awaking in the morning or on physical exertion. With the passage of days or weeks this becomes more severe and soon other symptoms are added to it. The patient has days on which he is drowsy. He may pass rapidly into stupor or even coma, emerging again to become almost normal. Transient accesses of diplopia with squint may be noted. Examination during a period of maximal symptoms may reveal a papilloedema, sometimes severe in rapidly developing cases. The plantar responses may, on one or both sides, be of the extensor type. There may be an inequality of tendon jerks on the two sides, the abdominal reflexes may be diminished on one or both sides. They may even be absent. Periods of mental confusion may also occur.

This fluctuation in the severity of the symptoms, the fugitive character of the physical signs and the generally downhill tendency of the illness, despite the fluctuations are amongst the features which are characteristic of subdural hæmatoma and help to differentiate it from that of intracranial new growth. When the syndrome develops rapidly, it is more common to meet a marked slowing of the pulse than in new growth.

In most instances there is no trace of blood in the cerebro-spinal fluid.

In all cases there is a great liability to a rapid development of coma with a fatal issue. Yet, the occasional finding at necropsy of what is clearly a subdural hæmatoma of very long standing, unsuspected during life, shows that from time to time the sequence of events briefly reviewed above fails to develop. Though it has to be confessed that even in such cases it is highly probable that careful clinical examination and an awareness on the part of the examiner of the symptom-complex of hæmatoma might have made diagnosis possible during life.

Diagnosis and Treatment.—The features which should make clinical diagnosis possible have been described, but in certain doubtful cases certainty

can be obtained only by an exploratory operation. This may consist in bilateral trephine holes and tapping of the subdural space, or in the turning down of osteoplastic flaps and the evacuation of the cyst when found. It is clear that treatment is essentially surgical.

EMBOLISM

The majority of embolisms of the cerebral arteries occur in valvular heart disease, 89 per cent. (Saveliew). Embolism may also occur from detached portions of clot from an aneurysm, from thrombi in connection with atheroma or syphilis of the aorta, and from detached clots which may form in the region of the pulmonary veins and left heart where there is no cardiac valvular disease. This latter condition is not an infrequent cause of puerperal apoplexy. It occurs in suppurative and gangrenous conditions of the lungs, and is an essential factor in the production of "pulmogenic" cerebral abscess. Embolism is rather more frequent in women on account of the greater incidence of mitral stenosis in that sex, and from the puerperal cases.

The embolus comes from the left heart and may be a vegetation from a quite recent endocarditis, but is more commonly a detached vegetation from a chronic and especially from a septic endocarditis. Very frequently it is a detached portion of clot which has formed in the left auricle in mitral stenosis. The middle cerebral arteries are the usual sites of lodgment of the emboli, and the left middle cerebral is rather more frequently affected than is the right. Embolism of the other cerebral vessels may occur, but is extremely rare.

The pathological events which may follow the plugging of a cerebral vessel with an embolus are varied and are highly important. In the first place, secondary thrombosis may proceed from the embolus throughout the whole distal distribution of the vessel, and lead to complete softening of its area of supply, and the clinical aspect will be that of severe and unchanging damage to the brain. The softened area may shrink or may undergo cyst formation, or it may be completely absorbed, giving rise to a porencephalus. Secondly, the embolus in the absence of secondary thrombosis may become adherent to one spot of the vessel wall at the site of its lodgment, and retracting from the vessel wall elsewhere, the blood channel becomes reopened, and the clinical results of the embolus, at first very severe, may disappear with unexpected and dramatic rapidity, and complete recovery ensue. Thirdly, the embolus may contract at the site of its primary lodgment, and become detached and shifted on by the blood stream to find a second resting-place in a much smaller artery. This event is manifest clinically by rapid clearing up of the physical signs in many regions, with persistence or even deepening of the involvement of one particular region. For example, a severe and complete hemiplegia clears up suddenly on the third or fourth day, leaving a brachial monoplegia only. Fourthly, an embolus may be impacted at the termination of the internal carotid artery, giving rise to severe hemiplegia with blindness of the opposite eye from blocking of the ophthalmic artery—*carotid hemiplegia*. Owing to the re-establishment of the circulation by the circle of Willis the hemiplegia is likely to recover rapidly, while

the eye remains permanently blind owing to secondary thrombosis extending through the ophthalmic artery. When an embolus is finally lodged and completely occludes the artery, the condition, both pathologically, clinically and from the point of view of treatment, is one of thrombosis, the cause of the embolism being taken into consideration. As with thrombosis, the immediate result of permanent occlusion is a condition of infarct and acute œdema in the region from which the blood supply is cut off. The acute œdema causes local pressure and increased general intracranial pressure, and is a common cause of the transient coma which often supervenes a few hours after the stroke, in both embolic and thrombotic apoplexy.

Stroke from embolism is the most suddenly occurring of all apoplexies, and the ictus is not preceded by any prodromal cerebral symptoms. Consciousness is apt to be lost at once if the whole middle cerebral artery be occluded, especially if the lesion be upon the left side. Or it may be retained throughout if the embolus lodge in a small vessel only.

Diagnosis.—This rests upon the occurrence of sudden apoplexy without prodromal symptoms in the presence of an obvious cause for embolism such as cardiac valvular disease, aortitis, pulmonary thrombosis or the puerperal state. The diagnosis can be a matter of probability only in those conditions where either embolism or thrombosis is likely, such as enfeebled cardiac states and the puerperal state.

Prognosis.—The prognosis in cerebral embolism depends upon the size of the vessel which is plugged, as deduced from the severity of the initial symptoms and their extent, and upon the immediate pathological changes which occur in the obstructed vessel as above described; and according to the nature of these changes it may be the most severe and least recoverable, or, on the other hand, the least severe and most recoverable of all forms of apoplexy. The prognosis of the condition causing the embolism is often the more important.

Treatment.—The treatment is that of cerebral thrombosis (see p. 1606) together with that of the condition giving rise to the embolus.

ARTERIAL THROMBOSIS AND HÆMORRHAGE

Cerebral thrombosis and cerebral hæmorrhage seem hitherto to have been described in text-books of medicine as quite separate conditions, almost antagonistic and mutually incompatible, between which it was highly essential and even possible to make a differential diagnosis for the purpose of applying a very dissimilar line of treatment in the respective conditions, each line of treatment being the worst possible for the other condition. It cannot, however, be too forcibly pointed out that primary arterial thrombosis and primary arterial hæmorrhage depend in every case upon degeneration of the arterial wall, and that every condition of degeneration of the arterial wall may cause either thrombosis or hæmorrhage indifferently. It is a usual experience to find in patients who have had severe strokes that thrombosis was the cause of the earlier, and hæmorrhage of the final apoplexy. Even in that condition, which has always been held to be the most important antecedent of cerebral hæmorrhage—renal disease with

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high arterial tension—Janeway has recently found that thrombosis and not hæmorrhage was the cause of apoplexy in many of his cases.

On account, therefore, of the identity of the underlying pathological condition in every case, and the clinical association of thrombosis and hæmorrhage of the cerebral arteries, and the difficulty of distinguishing them clinically, the two conditions are here described together.

Ætiology and Pathology.—The arterial degeneration which may result in cerebral thrombosis and hæmorrhage is due to the following causes: (1) Syphilis, which is the commonest cause of thrombosis in the first half of adult life, and which is less commonly the cause of hæmorrhage. It may affect both the large and the small arteries, even to the smallest. All the coats of the artery are affected, and in the case of the finest vessels there is conspicuous lymphocyte accumulation or “cuffing” round the vessel. In the neighbourhood of the affected vessels there is always syphilitic cerebritis in the form of lymphocyte exudation and oedema, and meningitis, if the lesion come to the surface. This is the most recoverable of all thrombotic lesions of the brain. (2) Atheroma, which is the common cause both of thrombosis and of hæmorrhage in the second half of adult life, and which is by far the commonest cause of hæmorrhage. It must be especially borne in mind that cerebral atheroma may be local in the cerebral vessels, and unassociated with general atheroma of the systemic vessels. (3) Arterial hypertrophy, with secondary focal degeneration of the media, with or without its commonly associated renal disease, which is of the nature of “small white kidney” in children and younger adults, and of the various types of “granular kidney” in older subjects. (4) Abnormal conditions of the blood, especially when associated with feeble cardiac action and low blood pressure, as in the puerperal state, and in septicæmic conditions, and at the time of the menopause. Hæmorrhage into the brain may also complicate polycythæmia and acute leukæmia. (5) In association with new-growths of the brain, both thrombosis and hæmorrhage are common events, especially when the neoplasm is soft and rapidly growing. The vascular lesion may occur quite early in the course of the new-growth, and apoplexy may be the first sign of its presence. (6) Inflammatory conditions of any nature may cause thrombosis and hæmorrhage. The vascular lesions are usually small, but they may be extensive, and may cause death. (7) Traumatic lesions, such as the passage of a bullet through the brain, or a blow upon the head, or concussion from high explosives, may cause extensive thrombosis or hæmorrhage.

While cerebral hæmorrhage results often enough from the direct rupture of a true aneurysm, or of one of those irregular local thinnings of the vessel wall which is called a “false aneurysm,” and may take place from an artery the wall of which is softened by disease though there be neither thinning nor bulging of the vessel wall, yet it is probable that cerebral hæmorrhage is very often the direct consequence of thrombosis, and especially of thrombosis which has occurred some time previously. The sequence of events is as follows: An area of thrombosis occurs within the brain, and the usual softening and necrosis follow. On the confines of this area, the necrosis spreads to come in contact with the wall of a living and unthrombosed artery, perhaps of considerable size. The arterial wall of this vessel was nourished by the capillaries of the necrosed area, and with its nutrient supply now

cut off there is local degeneration of the wall of the living artery. Moreover, the shrinking of the necrosed area of brain causes loss of support to the degenerate wall of the vessel, which ruptures as the result, under the influence of any sudden increase of blood-pressure. It is for this reason that one commonly finds in patients who have had multiple attacks of apoplexy, that the final and fatal attack is one of hæmorrhage, and that the preceding attacks have been attacks of thrombosis. These spots of thrombosis, which cause hæmorrhage, need not be of large size, and they may be so small as hardly to cause symptoms on their occurrence. Marie first called attention to these small spots of thrombosis as *plaques jaunes*, small yellowish-brown spots, softened and sometimes cystic, and pointed out their importance as a cause of cerebral hæmorrhage. In a similar way the thrombosis of syphilitic arterial disease may cause subsequent hæmorrhage.

Syphilitic cerebral thrombosis is not usually a pure pathological process, for the vascular disease is often accompanied by acute syphilitic encephalitis, with much lymphocyte extravasation in the vicinity of the diseased vessels, and acute local œdema, which increase the evascularisation when thrombosis occurs. The symptoms of cerebral loss of function are not all due to the thrombosis, but are in part owing to the recoverable acute inflammatory condition, and it is for this reason that syphilitic apoplexy often shows much more recovery than do other forms of apoplexy.

Thrombosis is a more common cause of apoplexy than is hæmorrhage, but it is much more frequently survived, while hæmorrhage is frequently fatal, within from a few hours to a few days of its onset. It follows therefore, that in the autopsy room of a general hospital, hæmorrhage is seen much more often than is thrombosis, while in infirmaries, where the survived cases of apoplexy collect, thrombosis is almost invariably the lesion found to be primarily responsible for the apoplexy.

Thrombosis tends to occur when an habitually high blood pressure is temporarily lowered and the circulation less active, and is always strongly suggested when apoplexy occurs during sleep and conditions of quiet, and after exhaustion, exposure to cold, severe purgation, and in debilitated states generally. It is preceded by slowing of the circulation in the area affected, and this may be productive of prodromal symptoms. Or there may be slight local thromboses preceding the main thrombosis, also giving rise to prodromal symptoms. Thrombosis may thus have an ingravescent onset, especially when clotting occurs in distal branches of an artery and extends towards the main vessel; but, on the other hand, it may have an absolutely sudden onset when the clotting occurs primarily in a large artery. The immediate effect of the thrombosis is a condition of infarct with œdema, extending widely in the vicinity, and it is this œdema which causes the loss of consciousness so commonly seen a few hours after the apoplexy has occurred. The œdema tends to pass off in a few days, and the area bereft of circulation by the thrombosis tends to become narrowed by collateral circulation from surrounding regions, and any recovery of function within the affected region must be by collateral circulation from elsewhere. The affected area at an early stage is bright red in colour, and soon becomes soft and shrunken (red softening). Later, the blood pigments degenerate with the production of bilirubin and are partly absorbed, producing a yellow-coloured lesion (yellow softening). Finally, much of the thrombosed tissue becomes necrotic and is

absorbed, leaving one or several cystic cavities. These cavities are never so sharply defined as those resulting from embolism, because of the more complete necrosis occurring with the later lesion. Still, a severe arterial thrombosis occurring at an early age may result in a porencephaly. Cavities found in cases of apoplexy after years have elapsed, are too often attributed to hæmorrhage. In reality they are nearly all due to thrombosis or embolism. The cerebro-spinal fluid in thrombosis is never found to contain blood, but some little time after the apoplexy it is often coloured yellow or yellowish-brown from escape of changed blood pigments, when the lesion has reached the surface of the convexity or the surface of the ventricle.

Hæmorrhage, which is usually described as an apoplexy of sudden onset, may be so when the escape is from a large vessel. When the bleeding commences from a smaller vessel, the symptoms are not sudden in their onset, but gather rapidly. Such a hæmorrhage is much like an avalanche. Commencing from a small vessel the hæmorrhage tears a small cavity, and in so doing opens up fresh bleeding points, and with increasing destruction more and more bleeding occurs from every piece of torn tissue, until the hæmorrhage reaches such a size as to burst commonly into the ventricle and much more rarely on to the surface. Indeed, it is difficult to conceive how a hæmorrhage into such a soft and vascular tissue as is the brain should ever stop. As a matter of fact, it very rarely does so, but causes death in the first attack of hæmorrhagic apoplexy, within from a few hours to a few days after the onset, from widespread tearing up of the nervous system and bursting into the ventricle. One of the most important clinical distinctions between apoplexy due to thrombosis and apoplexy due to hæmorrhage is that the former is often survived, and that the latter is almost invariably fatal within a short time of the onset.

Hæmorrhage may occur anywhere within the nervous system, but its common seat of commencement is in the centrum semiovale, and the vessel which bursts is one of the perforating arteries, of which the lenticulo-striate which carries the name of the "artery of hæmorrhage" is the most common. Such bleedings are often called "capsular hæmorrhages." It must be pointed out that this term capsular refers to the region outside the corpus striatum or external capsule, and not to the compact internal capsule as it converges to the crus cerebri. The cerebro-spinal fluid in cases of hæmorrhage contains blood within a very short time of the onset, and lumbar puncture often withdraws what is practically pure blood in large quantities. I have found blood present in large quantities very often within an hour of the onset.

Both thrombosis and hæmorrhage may occur in any part of the brain, while massive embolism is rare, except in the middle cerebral artery. The semioval centre, the calcarine region and the pons are the common sites of both hæmorrhage and thrombosis in that order of frequency. Hæmorrhage is rare except in these regions, while thrombosis is not uncommonly met with elsewhere.

Symptoms.—The nature of the symptoms in apoplexy will depend upon the site of the vascular lesion; and as the semioval centre or region of the middle cerebral artery is the commonest site for all the vascular lesions, hemiplegia is the common result; and this is associated with aphasia, if the lesion

is in the left hemisphere, and involves or isolates the cortex. When the calcarine artery is the site of the lesion, hemianopia results; and this is apt to be accompanied by word-blindness, if the lesion be on the left side. Pontine apoplexy involves the appearance of double hemiplegia, bilateral ataxy and bilateral loss of sensibility, with signs of involvement of cranial nerve nuclei and cranial nerves. Cerebellar apoplexies and thrombosis of the posterior inferior cerebellar artery produce acute ataxy with forced movements and vomiting.

Prodromal symptoms in the form of transient weakness of one or both limbs of one side, transient aphasia and giddiness occur in thrombosis only. An ingravescent onset occurs in thrombosis only and when the clotting occurs in the periphery of arterial distribution first and spreads towards the main trunk. When commencing in the parietal region, tingling and numbness of an extremity first occur, followed by a spread of these symptoms over half of the body, and subsequent weakness deepening into hemiplegia. When commencing in the left temporal region gradually oncoming aphasia is first noticed, and when commencing in the ascending frontal convolution a peculiar sensation of heaviness in the limbs gradually increases until hemiplegia is obvious.

The onset in embolism is always instantaneous; it may be sudden in thrombosis, and in hæmorrhage from a large vessel. In hæmorrhage it is always rapid. Consciousness is lost or not, according to the severity of the initial lesion and the site it occupies, and to the magnitude of the processes which follow the initial lesion, namely, the œdema of embolism and thrombosis and tearing of the brain tissue in hæmorrhage. In hæmorrhage, consciousness is lost soon, and the rapid development of severe symptoms which progressively deepen, is a most important early indication that this is the nature of the lesion.

In calcarine thrombosis the initial symptoms may be so slight as to pass unnoticed by the patient, whose first indication of defect may be, that he runs into objects on his blind side.

Convulsion sometimes occurs at the onset, and this nearly always indicates thrombosis, rarely embolism, and never hæmorrhage. There may be some local spasm in the region of the cranial nerves in pontine hæmorrhage, but this is not convulsion.

Conjugate deviation of the eyes is a common feature of all apoplexy. When the lesion is irritative at its onset, and not too destructive, and always when convulsion occurs at the onset, there may be active conjugate deviation, the eyes being turned away from the side of the lesion and towards the paralysed or convulsed side in hemiplegic cases, or the blind side when hemianopia is present. But this active conjugate deviation lasts but a short while and is followed by a paralytic conjugate deviation in the opposite direction, both eyes being directed away from the paralysed side and towards the side of the lesion. This variety of conjugate deviation may last for a considerable time, but usually disappears with the onset of deep coma.

The pupils are often unequal; they may be contracted, or dilated widely, and may be insensitive to light. In severe apoplexy, when as the result of the cerebral shock or when hæmorrhage or œdema have so raised the pressure as to greatly reduce the physiological activity of all the intracranial elements with the production of deep coma, the pupils are widely dilated and insensi-

tive. In pontine lesions, the pupils are often contracted to pin-point size, and this condition is of important localising significance.

In proportion to the severity of the general intracranial disturbance, respiration tends to be hurried, noisy and stertorous, and with increasing pressure to become irregular, grouped or of the Cheyne-Stokes type. The blood pressure tends to be raised and the pulse full in all conditions of apoplexy, provided the heart will respond to the requirement of an increased blood pressure in the face of an increased intracranial pressure.

Swallowing is often impossible, and the sphincters may be relaxed or retention may occur.

In the usual variety of apoplexy where the lesion is in the area of the middle cerebral artery and the local sign of the lesion is hemiplegia, it will be obvious that when the general intracranial pressure becomes severe and the coma becomes deep, the hemiplegia becomes less apparent, or masked by the universal condition of paralysis consequent upon the general intracranial condition. The physician often sees the patient for the first time when there is considerable coma, and he must determine upon which side the lesion is situated, and endeavour to have some perspective as to prognosis by determining the severity of the lesion.

The following points will serve to determine the side of the lesion when these signs are present: (1) The paralytic conjugate deviation is towards the side of the lesion. (2) The corneal reflex, when any is present, is diminished or lost on the hemiplegic side. (3) Painful stimulation will elicit less response or no response upon the hemiplegic side (hemianæsthesia). (4) The patient may respond by blinking to a feint made with the observer's hands towards the patient's eyes upon the sound side, and not on the hemiplegic side (hemianopia). (5) The limbs on the hemiplegic side when raised and allowed to fall passively, do so in a more lifeless, inert and flaccid fashion than upon the sound side. (6) And when there is any difference between the knee-jerks, abdominal reflexes and plantar reflexes, the former tend to be diminished and lost on the hemiplegic side while the plantar reflex will be of the extensor type on the hemiplegic side. It must be remembered in this connection, that a severe lesion of one cerebral hemisphere abrogates for a time at least most of the functions of the whole hemisphere, and that the hemianæsthesia and hemianopia, here referred to, do not necessarily indicate that the destructive lesion involves the visual and sensory paths. And further, that the condition of coma due to increased intracranial pressure of itself causes such signs as bilateral loss of abdominal reflexes and knee-jerks, and bilateral extensor responses in the plantar reflex.

The severity of the lesion may be judged—(1) From the depth of the coma; (2) from the degree to which the patient responds to any form of stimulation and from the general signs of nervous depression present—for example, a condition of complete bilateral flaccidity with complete loss of all reflex action and of all response to stimulation indicates a most severe lesion; and (3) from signs of failure of respiration as shown by irregular, grouped or Cheyne-Stokes breathing. It is further important to arrive at a determination if possible as to whether the condition present is stationary, deepening or showing signs of amelioration.

Vomiting is not an uncommon occurrence in the early hours of apoplexy and before coma becomes deep. Hyperpyrexia is often seen in fatal cases

before the end. It is especially common and may reach a high degree in pontine apoplexy. It may be preceded by initial depression of temperature. It is of fatal prognostic import.

HEMIPLEGIA is the commonest sequel of vascular lesions of the brain. The signs which serve to indicate its presence in the comatose subject have already been enumerated.

After cerebral thrombosis it may happen that the initial hemiplegia is completely recovered from, but unless this recovery begins early and progresses rapidly it is not likely to be complete.

The essential feature of hemiplegia is the loss of voluntary movements, but as this loss begins to pass off, certain new features make their appearance. These are muscular hypertonus, increased tendon jerks, and associated movements.

The restoration of movements follows a certain order. Deviation of the tongue and facial asymmetry clear up early; next, the leg begins to recover; and finally—and often very incompletely—the arm. The return of movements in the limbs is selective. In both upper and lower limbs, movement at the proximal joints recovers soonest and most completely. In the leg, extension and plantar flexion recover more completely than flexion and dorsiflexion. As a result, the patient can often stand when he cannot lift the foot and leg to step properly, and has instead to circumduct the limb when walking. In the arm, flexion movements recover soonest and best, while the fine skilled movements of the hand and fingers are frequently lost for ever.

The development of hypertonus, or spasticity, is as selective as the return of movements. In the leg, the extensor group becomes spastic; in the arm, the flexor group. Thus, the arm tends to take up a position of adduction, with flexion at elbow, wrist and digits. The leg is always spastic in extension, and does not go into flexion contracture, as may happen in spastic paraplegia from spinal cord lesions. The degree of hypertonus varies, and is greatest when the loss of movement is greatest.

The tendon jerks are exaggerated, and there is clonus (knee and ankle) in the affected limbs. The Babinski plantar response persists, but the abdominal reflexes, which are initially lost on the affected side, sometimes return after a period of months.

The forced immobility of shoulder and distal joints in the arm may lead to the formation of adhesions.

The so-called associated movements are involuntary changes of attitude of the paralysed limbs which accompany forceful voluntary movements, or such involuntary movements as, yawning.

CEREBELLAR APOPLEXY.—This is usually the result of thrombosis of the posterior inferior cerebellar artery, which is a branch of the vertebral artery, and the clinical picture is very unlike that of cerebral apoplexy. The patient is seized with a sudden intense vertigo which carries him to the ground, as in Ménière's disease. Incessant vomiting and forced movements follow, the forced movements rotating the patient, so that he comes to rest prone, with that side of the face corresponding with the side of the cerebellar lesion in contact with the pillow. There is intense ataxy, usually bilateral at first, and later becoming confined to the limbs and trunk on the side of the lesion. The patient is unable to lift his head, or to maintain the sitting or standing

position. When placed in such a position he positively dives to the ground when released. Nystagmus with the long slow movement to the side of the lesion, and a short fast movement in the opposite direction is conspicuous, and the skew deviation of the eyes is sometimes seen. There is much general hypotonia of limbs and trunk which soon becomes limited to the side of the lesion. Head retraction, pain and stiffness of the neck and opisthotonos may occur. When the patient's condition recovers sufficiently to allow of examination, all the signs of a unilateral cerebellar lesion will be found. Consciousness is not often lost. Since the posterior inferior cerebellar artery also supplies the lateral region of the medulla, signs indicative of disturbance of this region are usually present, and these may dominate the clinical picture rather than the cerebellar signs. Chief amongst them are analgesia and ther-manæsthesia of the face and head, due to implication of the as yet uncrossed quinto-thalamic path, and of the limbs and body upon the opposite side, due to involvement of that part of the spinothalamic tract which has crossed below this level. Between these two areas of sensory loss there is often a gap where sensibility is normal, corresponding with that part of the spinothalamic tract which is crossing obliquely at this level, and therefore is too near the middle line to be affected. Paralysis of the motor vagus is often found from involvement of the nucleus ambiguus, and, from the extension of the lesion or of consecutive œdema towards and across the middle line, sometimes causes severe dysphagia and dysarthria, and one of the great dangers of this form of apoplexy is extension of the thrombosis to that part of the medulla which contains the respiratory and other vital centres. When, however, such extension does not take place, and if the destruction of the lateral lobe is not too extensive, the most remarkable recovery may take place.

Diagnosis.—*The nature of the lesion.*—Embolism should be diagnosed in all cases where there is an obvious cardiac valvular lesion, particularly mitral stenosis, septic endocarditis, aortic disease and aneurysm. It is true that syphilitic cerebral thrombosis may occur with syphilitic aortitis, but the combination is rare, for syphilitic aortitis usually occurs at a much later age than does syphilitic cerebral thrombosis.

Further conditions of cardiac feebleness and corresponding feebleness of circulation must obviously predispose to thrombosis if arterial disease be present. Mistakes in diagnosis will, however, not often occur, and they are not of moment to the patient, for embolism, when once the embolus is lodged, is for all purposes of treatment and prognosis the same condition as is thrombosis. Thrombosis should be diagnosed in all primary apoplexies in young syphilitic subjects, for syphilitic hæmorrhage usually occurs at some time considerably subsequent to a syphilitic thrombosis. In this connection the serum reaction and the cytology and reactions of the cerebro-spinal fluid are all-important in the diagnosis.

Thrombosis should be diagnosed, notwithstanding the presence of high arterial tension or renal disease, in all cases of apoplexy without organic cardiac valvular disease, when the onset occurs during sleep or under circumstances of quiet, depletion or exhaustion, and in all cases where prodromal symptoms are marked, or where the onset of the apoplexy is gradual, and in apoplexies occurring in advanced age, for then hæmorrhage is almost unknown. All slight apoplexies and nearly all those that survive the first 10 days after the ictus, are due to thrombosis.

Puerperal apoplexy and that occurring at the time of the menopause in women are mostly due to thrombosis.

The cerebro-spinal fluid affords important indications, since hæmorrhage into the brain in most of the cases soon bursts on to the surface or into the ventricle. If blood is absent from this fluid a few hours after the ictus, thrombosis or embolism is highly probable and hæmorrhage is very unlikely. Any infarct condition coming to the surface may in the course of time cause the fluid to be blood-tinged or yellow. It is important to bear in mind that the infarct conditions of embolism and thrombosis are followed by packing of the infarcted region with polymorphs, and that these may escape from the surface in such numbers as to load the cerebro-spinal fluid with such a high polymorph pleocytosis as to suggest the presence of suppurative meningitis. Hæmorrhage is a likely cause of apoplexy occurring during exertion, especially if it occurs at a moment of severe physical strain, or at the height of passion. It is always a probable lesion in cases where a previous thrombotic apoplexy has occurred, the final event, where multiple strokes have succeeded one another, being almost invariably hæmorrhage. An apoplexy with rapid onset and with symptoms rapidly deepening, with a quick onset of deep coma, and the development of pyrexia and signs of respiratory failure, is usually due to hæmorrhage. The certain test that an apoplexy is due to hæmorrhage is the presence of blood in quantity in the cerebro-spinal space as proved by lumbar puncture. In cases of small white kidney in the young and of granular kidney before the age of 50 years, where the blood tension is very high, and where there is severe retinitis, hæmorrhage is the most likely cause of stroke.

The position and extent of the lesion.—The position of the lesion may be judged by the nature of the initial signs, whether visual, sensory, motor or aphasic, cerebellar or pontine, and later by the permanent symptoms resulting from the lesion. It must be carefully borne in mind in this connection, that a severe lesion of a cerebral hemisphere may entirely abrogate the functions of that hemisphere, initially by a process of shock and afterwards by the occurrence of œdema in the vicinity of the lesion, which may spread widely.

The extent of the lesion may be gathered by the severity or otherwise of the early symptoms and their rate of increase, and by early or immediate loss of consciousness, and by the completeness of the paralysis resulting. The more severe the extent of the lesion the sooner do grave signs of general cerebral failure appear.

Differential Diagnosis.—The diagnosis of coma due to a cerebral vascular lesion is usually made without difficulty from the history, and from the presence of unequivocal signs of local lesion of the brain. In a patient without history, and when the coma has become so deep as to remove the unilaterality of physical signs, from the severity of the general intracranial pressure, the diagnosis may be difficult from other causes of coma such as uræmia and diabetes, poisoning by opium, alcohol and its derivatives and illuminating gas, and in cases of difficulty search is to be made for the usually obvious signs of these conditions. Uræmia may present especial difficulties, for it is often associated with cerebral vascular lesion, and transient hemiplegic attacks may occur in this condition. This is true also of the crises of essential hypertension, which are described in more detail on page 1608. Absolutely

sudden death which is so often recorded in death certificates as due to apoplexy, is usually associated with a stoppage of the heart following the obliteration of one of its coronary arteries. Apoplexy never causes sudden death. There is one recorded case of death from cerebral hæmorrhage in 5 minutes, but it is rare in any apoplexy for death to occur in under 2 hours. Other conditions causing hemiplegia with coma must be taken into consideration. Epilepsy and especially hemi-epilepsy may be followed by marked unilateral paralysis (Todd's paralysis), which may last for a considerable time. Here the history of recurring attacks and the complete recovery will easily prevent confusion.

Cerebral malaria and sunstroke may closely resemble apoplexy, and should always come to mind when rapid coma follows the development of cerebral symptoms in circumstances where these causes are likely.

The congestive attacks of general paralysis of the insane are peculiarly difficult to diagnose from apoplexy. Perhaps they are due to suddenly occurring acute cerebral local oedema. They are liable to mistaken diagnosis, of course, only when occurring as the initial manifestation of the disease. These attacks take the form of rapidly occurring attacks of hemiplegia, aphasia, hemianopia, hemianæsthesia or of some combination of these conditions, usually associated with initial convulsions and followed by coma. The diagnosis of a syphilitic thrombosis is made with reason on the positive serum reactions, and cerebro-spinal fluid examination. If energetically treated it recovers with marvellous rapidity and completeness, to slowly develop the characteristic signs of general paralysis. It is the too rapid recovery in a case of apparent syphilitic thrombosis which should suggest the possibility of the stroke being a congestive attack in general paralysis of the insane. In all cases of coma without history, especially when there are signs of local cerebral involvement, a very careful examination of the head should be made for traces of recent injury, and if signs of injury be found, the skull and meninges should be opened, and the nature of the lesion sought out and dealt with surgically.

Prognosis.—A majority of the cases of apoplexy from syphilitic thrombosis make a fair recovery, which obviously depends upon how much permanent thrombosis occurs in the lesion of acute syphilitic encephalitis which is responsible for this condition, and upon the early application of appropriate treatment for syphilis. In some of these cases even, no recovery occurs.

In embolism the course and prognosis depend upon the extent of the vascular supply cut off when the embolus comes finally to rest; and upon the amount of collateral circulation afforded, and upon the cardiac condition.

In thrombosis due to atheroma the apoplexy may be rapidly fatal from extension of the thrombosis and secondary oedema, which raise the intracranial pressure beyond the limits of survival. In cases which survive, considerable recovery may occur in proportion to the extent of the lesion, but in these subjects an apoplexy is usually the beginning of the end, since the underlying pathological causes, arterial disease and failing cardiac action, still exist and are not amenable to any radical treatment. It is astonishing, however, how many of the cases of apoplexy due to atheromatous thrombosis survive for years without any recurrence of the thrombosis or occurrence of hæmorrhage. In cases of hæmorrhage, the immediate prognosis

is the gravest possible, the great majority of the cases surviving but a few hours.

Treatment.—When arterial disease is known to be present, the only measure which can in any way tend to safeguard the patient from apoplexy is moderation in all things: in diet, alcohol, mental and physical exercises, and above all moderation in all measures tending to lower the blood pressure, for hæmorrhage is due not so much to the immediate high blood pressure as it is to an antecedent period of low blood pressure in a high tension subject, which has allowed of thrombosis and which, when the tension is high, at some subsequent period causes rupture of a vessel in the thrombosed area. It is highly probable that no treatment influences the course and fatal issue of apoplexy due to hæmorrhage. Thrombosis and embolism, however, allow some scope for treatment, which should be the same in the two conditions; and as I have argued above that medical treatment in cases of hæmorrhage is useless and cannot avert the fatal result, I advise one line of treatment to be taken in all cases of apoplexy.

From the onset of symptoms in every case, a careful stimulant line of treatment must be adopted, and all depletive measures that may be calculated to lower the blood pressure and diminish the force of the cardiac action should be scrupulously avoided. It has been pointed out in the preceding passages how much the local and general symptoms of apoplexy are the result of cerebral ischæmia produced by the raised intracranial pressure, either from hæmorrhage or from œdema, and how nature attempts to combat this ischæmia by a reflex raising of the blood pressure, to keep the cerebral circulation going, and that how when the intracranial pressure exceeds that of the mean intracranial venous pressure, death must at once result from stoppage of the cerebral circulation. As Thomas truly emphasises, "How can the lowering of arterial blood pressure possibly help such conditions?" Absolute rest is, in the first place, essential when prodromal symptoms appear, and at the onset of an attack diffusible stimulants in the form of alcohol and liquid food; the heart's action may be improved by strychnine, while restlessness may be combated with bromides. If the patient is conscious, he should make as little effort as possible. His head and shoulders should be raised, special care being taken that the neck is not bent, and that nothing shall interfere with the return of blood from the head. If there is unconsciousness with stertor, the head and shoulders should be turned upon one side, so that the tongue should not fall back and impede respiration. If there be much cyanosis from impeded respiration, as is often seen in plethoric subjects, it is advisable to withdraw blood by venesection, for such relief of embarrassment acts as a stimulant to the circulation. Purgation should be avoided, and the bowel relieved at intervals by enemata. Stimulating food in a liquid form should be administered with stimulants at regular intervals; and if there is any difficulty in swallowing, the food should be administered with the nasal tube. The bladder should be carefully watched from the first, lest retention should occur, and the catheter passed when necessary. Lumbar puncture should, when necessary, be performed for diagnostic purposes, and it frequently gives relief from symptoms due to the high intracranial pressure. I have many times seen consciousness return within a few minutes of lumbar puncture, when much fluid can be withdrawn. It is advisable to withdraw all the fluid which will run out at a rate above the normal. Bed-sores and

hypostatic bronchitis must be avoided by the usual measures. In the cases that survive the first few days, passive movements should be used daily to all the joints of the affected side in hemiplegic cases, for this will obviate the painful rest adhesions which form in the joints of the paralysed limbs, and especially in the shoulder joint, and subsequently cause so much pain and misery to the patient. With the return of the power of voluntary movement, active exercises take the most important place in treatment. The final state of hand-and-finger movements depends not alone on the severity of the damage done to the brain, but in part upon the thought given to devising active exercises for it and the assiduity with which the patient can be persuaded to employ them. To avoid fatigue it is best to ordain a given daily period of some minutes to systematic exercise. A rubber sponge of appropriate size, fixed in the palm by a strip of webbing passing round the hand, will limit the passive flexion of the fingers, and will provide a resilient resistance against which the patient may move his paretic digits. Massage is an adjuvant, but never a substitute, for active exercises in the patient who can undertake them. Electrical stimulation of the muscles is absolutely contra-indicated. It has no other effect than to aggravate the spasticity that is so serious a hindrance to free movement. A hemiplegic patient after apoplexy, should be got upon his legs and encouraged to make attempts to walk as early as ever the returning power allows any possibility of the attempt.

HYPERTENSIVE ENCEPHALOPATHY

In the preceding section on the differential diagnosis of apoplexy, mention was made of the sudden and transient cerebral symptoms associated with essential hypertension, and some further reference to them is necessary. It is known that the subjects of this variety of hypertension may ultimately succumb to cerebral hæmorrhage, but it should also be borne in mind that they are subject from time to time to what are known as "hypertensive crises." The patient is the possessor of a persistently high blood pressure. The attack is precipitated by a further rise in this, and develops with intense headache, sickness and sometimes drowsiness or even semi-coma. Examination will reveal the presence of hypertensive retinitis in most cases, but in a proportion there is a definite papilloedema with retinal hæmorrhages and exudate. Accompanying these symptoms there may be hemiparesis, hemianopia, focal or generalised fits, or other indications of local cerebral lesion. The crisis is brief, lasting from a few hours to several days, and usually ends in recovery, but recurrence is likely, and finally many subjects develop cerebral atheroma and succumb to cerebral hæmorrhage. Intervals of several months may intervene between succeeding crises.

The presence of papilloedema is taken to indicate that cerebral oedema is complicating the situation. The transient nature of the crisis, and particularly the rapid appearance and disappearance of such symptoms as hemiparesis, exclude the possibility of arterial thrombosis or other material lesion of the kind, and spasm of the arteries has been invoked to account for the symptoms. There is of course no conclusive evidence that this occurs. Yet while the cerebral arteries are not under the same measure of

vasomotor control as arteries elsewhere in the body, it is known that some such control exists, and it may be that in arterial hypertension more intense spasm is possible than in healthy arteries. At least, it may be said that no hypothesis better founded or more in harmony with the facts of clinical observation has been formulated.

Differential Diagnosis.—As has been indicated, the transitory character of the symptoms exclude gross vascular lesions such as thrombosis, and the same may be said of intracranial tumour and lead encephalopathy. Yet it may be admitted that while it is present the hypertensive crisis shows many points of resemblance to the last two named conditions, especially when papilloedema is found. Plumbism in children and young persons not uncommonly develops with headache, vomiting, convulsions and focal signs, and the development of an intense papilloedema, sometimes also with high blood pressure and albuminuria, and search for other indications of lead poisoning and careful history-taking are necessary to exclude this condition. In intracranial tumour, the systolic blood pressure is rarely above normal limits, the history is longer and the condition progressive. Uræmia can usually be excluded, since in essential hypertension the blood urea is within normal limits, and the only abnormality in the urine may be a trace of albumin.

Treatment.—Venesection is indicated as the first step, and when there is papilloedema or other signs of cerebral oedema (convulsions, high cerebrospinal fluid pressure) lumbar puncture and the withdrawal of cerebrospinal fluid, and also the intravenous or intramuscular administration of hypertonic solutions are necessary. As a measure of urgency from 50 to 70 c.cm. of a 50 per cent. solution of dextrose may be given intravenously. For less urgent cases and as a measure that can be repeated for the relief of headache, six ounces of a 20 per cent. solution of magnesium sulphate may be given per rectum at 6 hourly or less frequent intervals. The convulsions may be treated by rectal administration of paraldehyde (240 to 360 minims in water), or by the hypodermic injection of 3 grains of soluble phenobarbitone in solution.

The subsequent management of the case is that of the underlying essential hypertension.

SINUS THROMBOSIS

Thrombosis of the cerebral sinuses may occur rarely as a primary condition, or it may be secondary to infective processes spreading to the sinuses from contiguous infected regions.

Ætiology.—Primary thrombosis is a rare condition. It is said to affect the superior longitudinal sinus most commonly. It is more common in the first year of life than at any other period, when it may follow diarrhoea, bronchitis or the conditions of exhaustion met with in tuberculous disease, and in congenital syphilis, and it may follow acute diseases such as measles diphtheria, etc. It may also occur at any age, up to advanced old age, in the terminal stages of cancer, phthisis and other chronic diseases.

The essential cause of secondary thrombosis is the advent of micro-organisms to the sinuses. The infection is often a mixed one, but the common organisms present are streptococcus, pneumococcus and *Bacillus*

coli. The sinus may become infected as a part of a general pyæmia, or infection may spread directly through its wall from a focus of local disease, most commonly from an extradural abscess. In most cases, however, the sinus becomes infected from a local spreading septic thrombosis of the veins which open into the sinus, from an infected spot at a distance. Thrombosis of sinuses may also occur from injury, as by bullet wounds and fractures of the skull, and may also result from surgical procedures in the region of the sinuses.

Pathology.—The affected sinus is bulged and distended, and feels to the touch, as if it were injected with a solid mass. In the infective forms, the clot may very quickly break down into pus, and general pyæmia result. When the superior longitudinal sinus is thrombosed, there is marked congestion of the convolutions of the convexity of the brain, often with cord-like clot-distended veins. There is bloody serum in the sub-arachnoid space contaminating the cerebro-spinal fluid withdrawn by lumbar puncture. Later, there is extensive bilateral softening of the cerebral hemispheres, most marked in the paracentral and surrounding convolutions. The cavernous and lateral sinuses do not drain the brain directly, and blocking of one of them does not cause so much cerebral disturbance, on account of the presence of alternative paths for the blood. Thrombosis of the cavernous sinus, however, may extend to the ophthalmic veins and cause blindness with an anæmic and infarcted condition of the retina. The nerves which lie in its outer wall, namely, the third, the fourth, the ophthalmic division of the fifth and the sixth nerves, may be paralysed.

Symptoms.—The clinical aspect of this condition is made up of three groups of symptoms—(1) the general signs of some bodily condition likely to be associated with thrombosis, such as marasmus, pyæmia, local cranial injury or septic disease of cranial bones and neighbouring tissues; (2) general signs of intracranial disturbance, which will depend upon how much the cerebral circulation is upset by the blocking, and which will be severe in cases where the superior longitudinal or the straight sinus is affected, and perhaps altogether absent where the cavernous sinus or the lateral sinus is affected; and (3) local signs of blocking of an individual sinus.

The general signs depend upon congestion, cedema, meningeal exudation and increased intracranial pressure. Headache, drowsiness, deepening into coma, and vomiting are common, while delirium and convulsions may occur. Papilloedema is not infrequent, while head retraction and rigidity of the neck, trismus, strabismus, inequality of the pupils, nystagmus, and irregularity of pulse and respiration may occur. In infective thrombosis, high pyrexia and rigors are the rule.

LOCAL SIGNS.—*Superior longitudinal sinus.*—The general signs are severe and convulsion is common, and bilateral hemiparesis or paralysis is likely to develop. There may be cyanosis and cedema of the forehead. The angular parietal and temporal veins may be distended, and in rare cases thrombosed.

Lateral sinus.—The clot may extend into the jugular vein and cause pain and stiffness on that side of the neck, and occasionally the thrombosed jugular vein may be felt beneath the anterior border of the sterno-mastoid as a tender solid cord. There may be tenderness and swelling over the region of the mastoid emissary vein.

Cavernous sinus.—There is œdema of the orbit, with proptosis and œdema of the conjunctiva, forehead and face. Amblyopia or blindness is the rule. Ophthalmoscopic examination reveals swelling of the disk with multiple hæmorrhages. Paralysis of the ocular muscles and anæsthesia of the eye on the same side may also occur.

Diagnosis.—If local signs, which give conclusive external evidence of sinus thrombosis, are absent, it may be very difficult to distinguish this condition from meningitis, abscess, encephalitis or other intracranial lesions. The septic forms of meningitis should be distinguished by the polymorpho-nuclear leucocytosis in the cerebro-spinal fluid. It must be remembered, that in the primary forms of sinus thrombosis in children, a copious lymphocytosis is met with, which may cause confusion with tuberculous meningitis. Abscess and sinus thrombosis often exist together.

Prognosis.—This disease is, as a rule, rapidly fatal from ever-increasing intracranial pressure; but some subjects, both in the non-infective and in the infective forms, survive. This is especially the case when the thrombosis is confined to one cavernous sinus. Cases of infective thrombosis of the lateral sinus following middle-ear disease have often been saved by timely surgical interference with ligature of the jugular vein and of the lateral sinus on either side of the thrombosed area, and with incision and turning out of the clot.

Treatment.—Beyond vigorous prophylactic measures against the causes of this condition and the palliative treatment of symptoms, surgical measures in cases of local infective origin alone are of avail. Further, in dealing with injuries of the skull in the region of the superior longitudinal sinus, trephining and exploration should be undertaken with a clear understanding of possible thrombosis of the sinus, and its appalling results.

APHASIA AND OTHER DEFECTS OF SPEECH

APHASIA

GENERAL CONSIDERATIONS.—The function of speech, which is the highest and most recently evolved human function has as its anatomical substratum a region of the cerebral convolutions situated in the left hemisphere and having its centre a little behind the middle of the first and second temporal convolutions. It is limited above by the posterior limb of the Sylvian fissure, occupies probably the tip and the whole external convexity of the left temporal lobe, and spreads backwards into the supramarginal and angular gyri, while it extends forwards over all the convolutions of the insula and possibly to the posterior ends of the second and third frontal gyri of the left side.

This "speech region of the brain" comprises not only the cortex but also the subcortical white matter which carries the paths of communication between the speech region and other parts of the brain. Posteriorly it receives an important white tract from the visual region of the cortex. An interruption of this tract results in the condition known as "pure word-blindness," or inability to appreciate written speech. Upon its deep aspect

the speech region of the convolutions receives the temporal projection of fibres conveying the auditory impressions, and destruction of this system by a lesion undercutting the convolutions in the centre of the temporal lobe produces "word-deafness," or inability to appreciate spoken language. In this same region another set of afferents impinges upon the speech area which convey the muscular sense impressions and other sensory impressions which are produced in the movements of articulation and which are the only guidance which the "deaf mute" has in the knowledge of correct execution in his articulation.

A lesion deep in the temporal lobe which interrupts both the foregoing paths, isolates the speech region from any appreciation of correct execution, with the result that spoken language becomes unshapen and degenerates into a voluble jargon, "jargon aphasia."

In the anterior half of the speech area a tract of white fibres gathers by degrees, and passing forward constitutes the bulk of the "temporal isthmus," which joins the temporal lappet to the insula, and runs beneath the insula to the region beneath the first and second frontal convolutions, from whence it is connected with the pyramidal path of the left side, and by way of the corpus callosum with the pyramidal path of the right side. This is the executive outgoing path for speech movements and a complete lesion of this path, as by a limited subcortical lesion underlying the posterior end of the left third pre-frontal gyrus and anterior part of the insula, will result in complete inability to exteriorise either spoken or written speech—"pure aphasia" and "pure agraphia."

In the speech area of the brain thus limited, little or nothing is known of any localisation of function. It is generally held that there is a gradual passing over from receptive function (appreciation of spoken and written language) in the posterior regions, to executive function (exteriorisation of spoken and written language) in the anterior regions.

Inasmuch as the phenomena of "word-blindness" and "word-deafness," as well as executive "aphasia" and "agraphia" result from lesion of the speech area, these seem to result from lesion of the tracts concerned and not from interference with the function of the cortex. These phenomena are of common occurrence in connection with lesions of the speech region and led to the formulation of localised areas of the cerebral cortex with specific functions in regard to speech. Thus, Broca's centre in the cortex of the posterior part of the left third prefrontal convolution was the motor centre for spoken language, while Exner's centre in a similar position in the second left prefrontal gyrus was the motor centre for written language. The "auditory word-centre" in which auditory memories of words were stored was in the cortex of the first and second temporal gyri, and the "visual word-centre" in which visual memories for words were impressed was in the cortex of the angular gyrus. These various centres were connected together by to-and-fro paths which could be separately affected by a lesion, and the attempt was made to explain the multitudinous and varied phenomena which occur in lesions of the speech region by damage to one or other of these hypothetical word-centres or to their connecting paths. The result was highly unsatisfactory, for the cases generally refused to correspond with the theories clinically, and practically never corresponded pathologically. Dejerine, by his discovery that a subcortical lesion in the right place could produce "pure

aphasia and agraphia" at the front end of the speech area and "pure word-blindness" at the posterior end, the cortex being intact, went far to make untenable the theories of narrow localisation of function within the speech centre. Subsequently the work of Pierre Marie, Head and others has placed modern conceptions of speech function upon a less artificial basis.

The speech function seems to be concerned with the left hemisphere of the brain alone in right-handed persons, and this is explained by the major potential of the left hemisphere for receptivity and education associated with the major use of the right hand through the countless ages of humanity. Left-handedness is usually associated with a transfer of the speech function to the right hemisphere, but there are exceptions to this rule.

The possibility of the transference of the speech function from the left to the right hemisphere is great during childhood, to the extent that no lesion of the speech region of the left hemisphere, however extensive, causes lasting loss of speech in a child under the age of six years, provided sufficient intelligence remain. After this age the possibility of such compensation by the right hemisphere for lesions in the left hemisphere seems gradually to diminish and to occur but little after adult life is reached, but even in adult life remarkable exceptions to this rule are seen.

The descending paths from the brain by which speech is executed are the pyramidal paths. The speech area of the cortex seems to command both right and left pyramidal systems equally, so that no lesion of one pyramidal system is ever productive of speech defects, either aphasic or dysarthric.

From this it follows that aphasia, word-blindness, word-deafness, amnesia, etc., only result from lesions of the convolutions and of the white matter closely underlying the convolutions, and never from lesions of the deeper parts of the corona radiata and capsules.

When, however, both pyramidal systems are involved, as, for example, by bilateral lesions of the brain, or by lesions of the brain stem which involve both pyramidal systems where these are contiguous, or by neuronie degeneration of the pyramidal systems in general, then defects of articulation arise comparable to the spastic paralysis of hemiplegia. These do not concern the pattern of speech, but solely the articulation, which becomes slow, clumsy, and slurring and indistinct, from weakness of movement, stiffness of the muscles concerned, and inability finely to adjust the stop positions at which the consonants are made. This condition, which is known as "spastic dysarthria," is commonly met with in double hemiplegia, in lesions of the brain stem involving both pyramidal tracts, and also in the tonic form of progressive muscular atrophy. In older writings it is often referred to under the most inappropriate name of "pseudo-bulbar paralysis."

When the lower motor neurons subserving the speech mechanism are bilaterally affected, a very similar dysarthria results, from the weakness and inaccuracy of the movements thus entailed, which is known as "flaccid or atrophic dysarthria," and which is met with in lesions of the medulla oblongata of all kinds, in progressive muscular atrophy, and in peripheral neuritis.

LESIONS RESPONSIBLE FOR APHASIA.—By far the most common cause of aphasia, in all its degrees and varieties, is vascular disease, usually thrombosis, less commonly embolism, and only in the rarest cases hæmorrhage—

for the reason that the two former lesions often affect the vascular supply of the superficial parts of the hemisphere, whereas hæmorrhage is generally situated deeply; moreover, cases of cerebral hæmorrhage rarely survive the occurrence for more than a few hours. Cerebral tumour is the usual lesion causing aphasia of gradual onset, and is perhaps the only known cause of isolated "word-deafness" and of "vulgar jargon aphasia," for it is the only conceivable lesion which can undercut and, therefore, isolate the temporal convolutions without otherwise interfering with their function.

PHYSIOLOGICAL CONSIDERATIONS.—Within a short time after birth the child begins to recognise the nature and uses of some of the objects in the world around it, and to express its simple conscious process by gestures, and it early appreciates the "gesture language" of those around it. The "mimesis," or gesture language, thus early impressed and expressed, remains throughout life the most stable, the least vulnerable, and the longest lasting of the methods of receiving and communicating ideas. Long before it is able to utter any articulate sound, the infant learns to connect certain sounds which it hears with certain objects and with certain events, and the memories of these auditory patterns first implanted serve by far the most important function in the processes and expressions of thought throughout life. Whereas we rely upon our visual memories for our remembrance and intelligence in general matters almost exclusively, yet as regards speech we rely upon auditory memories to a very large extent, and of course those who have never learned to read do so exclusively. The process of recall, both in silent thought and in speaking, is the revival of auditory patterns. We are, therefore, strong "visuals" as regards general memory, but strong "auditives" as regards speech memory, and the relative strength of the two functions varies somewhat in individuals, according to personal idiosyncrasy and to education, and this individual variation is sometimes apparent in the phenomena of aphasia. From the original connection with hearing, the memories of speech patterns come to be located in that part of the brain associated with the auditory function—in and around the temporal lobe. Later, guided by the auditory memories, the child begins to express himself in articulate speech and he does so by the revival of auditory memories.

All living motion is sensory-originated, sense-guided and sense-governed, and has a motor process of itself has no proved conscious concomitant. Our consciousness is that of the sensations which accompany the movement, or which result from the movement. The knowledge of correct execution so gained fortifies and increases the functional stability of the speech area, and is of immense importance in the speech function. If it be absent owing to a lesion isolating the speech area on the incoming side, speech degenerates into a jargon and soon becomes impossible; just as in tabes the walking becomes irregular from loss of the muscular sense conveyed in the posterior columns, and ultimately standing becomes impossible.

When at a considerably later age the child learns to read and to write, certain visual patterns (letters, words, sentences) become connected with certain objects and ideas, and become linked on to the already well-established auditory memories of speech. The meaning of the visual symbols is learned by the child from the meaning of the word or pattern spoken, which he already knows well, and the already developed auditory speech

function serves as the instructor of the visual speech function, and throughout life remains the more potent, more dominant and less vulnerable function of the two.

Later still, in learning to write, the child relies upon his visual memories, and as his knowledge of correct execution in writing is largely visual and only in minor degree common sensory from the movements of the hand in writing. It follows that the function of exteriorising speech by writing becomes intimately connected with and a part of the visual speech function, and is usually depressed or lost with the visual speech function as the result of disease. It will thus be seen that there are not separate regions of the speech area in which the auditory memories of language and the execution of spoken speech on the one hand, and the visual memories of language and the execution of written language on the other hand, are represented, but that there are four functions intimately coupled in pairs, which have their seat in the same anatomical substratum.

It is a general principle that when the speech area is damaged the speech function becomes depressed as a whole, with the result that function is lost in order of its depth of impression.

Symptoms.—Small lesions of the convolutions seem to produce no defects at all, and this is perhaps true of all the regions of the cortex of the brain. There can be, therefore, no narrow localisation of function, and there must be capacity for compensation for such small lesions in the surrounding undamaged cortex. With larger lesions of the cortex, and in proportion to their extent, mutilation of the patterns of speech, slowness of utterance, inability to find the words (inability to recall), especially nominals, and above all isolated nominals, and finally confusion of speech intelligence occur, in that order.

In the mutilated speech of the aphasic may be sometimes noticed stammering. This condition is at once distinguishable from true jargon aphasia, since the former is slow and halting whereas the latter is facile and voluble. Misplacement of words and the use of wrong words is common and is called "paraphasia." A tendency to repeat a word once pronounced is sometimes present and bears the name "echolalia." The same faults occur also in writing, as faulty spelling, misplacement of letters and words, wrong words, "paragraphia" and "echographia." Much defect of general intelligence always accompanies severe damage to the speech area, and this will be readily understood from the very large rôle which speech patterns play in the working of thought. Difficulty in the recall of words and speech patterns, which has been termed "verbal amnesia" or "nominal deficiency," is a characteristic feature of lesion of the speech area. This difficulty is greatest with spontaneous revival than with recall, which is "kicked up" by direct sensory stimulation. For example, an aphasic person who is unable spontaneously to utter a word, may repeat the word at once when it is spoken to him, when he sees it in writing, or when the corresponding object is shown to him. It is important in this connection to bear in mind that we do not speak in the letters of the alphabet, nor in the words of our dictionary, but in a running pattern of sound. The pattern or context provides the meaning, while the individual words are negligible and have no meaning. The power of the pattern in aiding revival is very great both from sequence rhythm and musical quality. As examples, an aphasic who

has no utterance spontaneously is told to count with his interlocutor. The interlocutor begins counting, the aphasic joins in. The interlocutor then stops, but the aphasic continues counting, carried by the sequence rhythm.

The confusional defects of speech function are met with in extensive damage to the speech area, and are usual as immediate and transient phenomena in all suddenly occurring lesions of the speech area. There is general mental dullness, with varying degrees, usually severe, of depression of speech function, and much confusion, both on the acceptive and expressive side, when any of these functions remain, and the results of the examination of the speech faculty are apt to vary very much from moment to moment, for attention is very difficult to hold and the patient is easily fatigued and bored. Severe degrees of this form of defect may be associated with inability to recognise objects—"object-blindness," and with loss of ability to convey ideas by gesture—"amimia."

Prognosis.—In attempting to estimate the degree of recovery which is likely to occur in cases of aphasia, it is necessary first to bear in mind that sudden cerebral injury is apt at first, by the process which has here been described as functional depression or "diaschisis," to cause very wide loss of function, though the lesion may not be very extensive. A total aphasia, for example, is often the immediate result of a lesion of moderate size. Such phenomena last usually not longer than a week, and until they have passed off it is impossible to make a definite statement, either as to the extent of the lesion or the likely degree of recovery. Speech may be regained by two entirely separate processes—either by recovery of function in partly damaged and functionally depressed areas, or by compensatory activity in the potential speech area of the undamaged hemisphere. The possible recovery of function will depend upon the nature of the lesion and upon its extent. It will be greater when a lesion may be judged to be one of pressure rather than of actual destruction, if such pressure be removable, as in subdural hæmorrhage, abscess and gumma, and least when widely spread arterial disease and a failing heart suggest that the lesion is a thrombosis, and when an irremovable tumour is present. The greater the extent of the lesion if it be presumably from vascular occlusion, as judged by the associated signs, paralysis, anæsthesia and hemianopia, the less is the chance for functional restitution, as there is then little hope of any useful restoration of the circulation through collateral vessels. In children under the age of six years, unilateral lesions produce no permanent speech defects, provided sufficient intelligence remains, but even to this rule some important striking exceptions have been recorded. When adult life is reached, transference seems to occur but little, yet in a few recorded instances destruction of the posterior half of the speech area has been followed by an almost complete restoration of speech function.

Treatment.—A careful and patient system of re-education in speech, such as is used in teaching mentally deficient children, is often of great value in all forms of speech defect. From the amount of labour that the teacher has to expend for very little progress made, this treatment is not often given a fair trial. A fair degree of intelligence must be present, and care must be taken that the lessons are not prolonged to the production of the boredom, with accompanying inattention and confusion, which occurs so readily in aphasic patients. The utterance of a simple vowel sound should

first be taught, then that of the several vowel sounds, and afterwards that of consonants and their combinations, and the patient should be directed while learning to watch the movements of the lips, etc., of the teacher. The simultaneous presentation of an object with its spoken and written name is often helpful in stimulating the remains of speech function into activity. An intelligent patient soon comes to recognise under such tuition that he has no paralysis of the articulatory mechanism.

TESTAMENTARY CAPACITY.—No rule can be laid down as to the capacity of a person suffering from aphasic speech defects to exercise civil rights and to make a will, and each case must be judged upon its own merits. The first and all-important consideration is the degree of intelligence, and when this is good it is essential for such capacity that there should be some mode of cognition and of expression left. Pure word-blindness and the extremely rare condition of pure word-deafness do not interfere with the exercise of civil rights, for the patient can understand what he hears in the first case, and what he reads in the second, and in both conditions can express himself both in speech and writing. In cases of pure aphasia and pure agraphia there is complete civil capacity, but when, as usually happens, the two conditions co-exist, though intelligence and the receptive side of speech may be but little impaired, yet the expressive side of speech is reduced to gesture, and extreme difficulty may be met in ascertaining the patient's wishes. Auditory amnesia, and combined auditory and visual amnesia, and confusional defects, except in the slightest forms, interfere seriously with testamentary capacity and with capacity for exercising civil rights. In such cases there is great loss both on the acceptive and on the expressive sides of speech, with confusion of memory and impairment of intelligence. Most satisfactory results have, however, many times been brought about in apparently hopeless cases by careful, sympathetic and repeated procedures, in which the properties to be bequeathed and the likely legatees are assembled before the patient, thus allowing the testator to match the gift with the recipient. The proceedings should be conducted in the presence and under the direction of a physician thoroughly conversant with the subject of aphasia. All concerned should bear two points in mind, the one being that the wishes of the legator must be paramount, and the other that an obviously just will is most difficult to upset in a court of law.

THE METHOD OF EXAMINATION of patients suffering with speech defects should be in accordance with some definite scheme so drawn up as to test each function of the complex physiological process of speech.

The following scheme is convenient: (1) Is the patient right- or left-handed, and, if the latter, did he write with the right hand? (2) What was the state of education as regards reading, writing and foreign tongues? (3) Does he understand the nature and uses of objects, and can he understand pantomime and gesture, or express his wants thereby? (4) Is he deaf? If so, to what extent, and on one or both sides? (5) Can he recognise ordinary sounds and noises? (6) Can he comprehend language spoken? If so, does he at once attempt to answer a question? (7) Is spontaneous speech good? If not, to what extent and in what manner is it impaired? Does he make use of wrong words, recurring utterances, or jargon? (8) Can he repeat words uttered in his hearing? (9) Is the sight good or bad, is there hemianopia, or papillœdema? (10) Does he recognise written or printed

speech and obey a written command? If not, does he recognise single words, letters, or numerals? (11) Can he write spontaneously? What mistakes occur in writing? Is there paraphria? Can he read his own writing some time after he has written it? (12) Can he copy written words, or from print into writing? Can he write numerals or perform simple mathematical calculations? (13) Can he read aloud? (14) Can he name at sight words, letters, numerals and common objects? (15) Can he write from dictation? (16) Can he match an object with its name, spoken or written, when a series of objects and names are simultaneously presented? (17) Any other tests, emotional, rhythmical, or musical, which may raise the physiological level of the speech centres. (18) Any other means of proving in what way he can receive and express ideas.

OTHER DEFECTS OF SPEECH

1. STAMMERING OR STUTTERING.—A spasmodic defect of articulation leading to a sudden check in the utterance or words, or to a rapid repetition of the consonantal sounds in connection with which the difficulty arises. To the trouble with articulation are often added spasmodic movements of the face and head, or indeed of any part of the anatomy.

Except in the rarest instances this condition is not associated with any structural changes in the nervous system nor in the organs of articulation, but it has been observed as the end-result of a lesion of the speech area. The disorder seems to consist in a lowering of the functional stability of the executive speech mechanism by a physiological embarrassment in consciousness. It is begotten of shyness and self-consciousness. It is never congenital, nor met with in early infancy, but arises at the age when shyness and self-consciousness trouble youth most. It is infinitely commoner in boys than in girls, for the latter are much less liable to self-consciousness. The stammerer never stammers in the speech of thought, nor when talking aloud to himself alone, nor at any time when singing, for in the two former cases the embarrassment of self-consciousness is absent, and in the last case the element of rhythm and music greatly increases the stability and confidence of the function. It not uncommonly appears in conditions of debility, and especially after measles and diphtheria. It has a frequent origin in sudden fright. It is the historical utterance of fright and of those who find themselves suddenly "*in flagrante delicto*." In rebellious cases the element of self-consciousness seems to disappear, while the stammer persists as an ineradicable habit, and it is remarkable how many sufferers of this class have strong aspirations towards public speaking.

In articulate speech three muscular mechanisms are concerned—(1) the respiratory mechanism for supplying the blast of air, (2) the larynx for producing the voice, and (3) the muscles of the lips, tongue, jaw, and palate for articulation. For distinct speech there must be absolute co-ordination of these mechanisms one with another. Consonants are in nearly all cases the source of the difficulty in stammering, and while these are all buccal sounds, yet some begin with a laryngeal sound, while others are purely buccal. The former are termed "*voiced consonants*," and are B, W, V, Zh, Z, Th (as in "*thus*"), D, L, R, G, Y; and the latter "*voiceless consonants*,"

and are P, F, Th (as in "thin"), S, Sh, T, K; while N, M, and Ng terminal, are "voiced nasal resonants." If one articulates these consonants it becomes at once clear that it is the presence of the initial laryngeal element or "voicing" which makes the difference between B, V, Z, D, G, and P, F, S, T, K, respectively.

A careful attention to the manner in which the letter sounds are produced is absolutely essential in the investigation and treatment of stammering. The difficulty occurs most commonly with the explosive consonants, P, B, T, D, G, K, and nearly always where these occur as initial letters—that is, in starting the articulatory mechanism; and to avoid this difficulty which arises after every pause, most stammerers speak in a rapid monotonous fashion. The fault chiefly lies in the direction of energy to articulation rather than to phonation. The patient held up by his stammer usually remains silent, but occasionally, having produced the first sound, he continues to repeat it—the reduplication stammer which has been the origin for the names "stammer" or "stutter" by which the malady is known.

Often the patient uses a trick or contortion to prevent the stutter or to relieve the feeling of nervous tension and embarrassment in consciousness which the defect causes, and these tend to become engrafted on him, as (1) associated sounds—whooping, grunting, crowing, etc.; (2) habit spasms—contortions of the face, limbs, or body, which sometimes take a complicated form and exactly resemble the co-ordinated forms of tic.

Prognosis.—The majority of the cases tend to a spontaneous cure, and recovery is hastened in all cases by systematic treatment. In every class of case the results of treatment may come slowly at first, but perseverance will in almost every case bring success.

Treatment.—Attention should be paid to conditions of general health, and to the mental well-being and satisfaction of the child, with plenty of scope for pleasure and satisfying occupation.

It is well for the patient to speak, read, or recite in a large room alone, loudly, slowly and distinctly. The following system for such exercises is useful: (1) The chest must be kept well filled with air. This most important point is often most difficult to the patient. (2) He must speak slowly, with a full resonant voice. (3) When he comes to the word on which he tends to stutter, he should raise his voice and direct his energies to vocalisation, and not to articulation. If the difficulty be over a voiced consonant, he must be directed to voice it firmly. If the consonant over which he stumbles be a voiceless one, attention must be directed to the vocalisation of the subsequent vowel sound; for instance, in "pat" he must attempt to vocalise the "at," and he will find little difficulty in prefixing "p" as the syllable is uttered. (4) Gymnastic and singing exercises are valuable additions to treatment. Should associated movements be present, the speaking exercises may be carried on in front of a mirror, so that the patient may see these himself and endeavour to suppress them.

The development of confidence and self-reliance is everything in the treatment of stammering. The skilled teacher first gains the liking, respect, and submission of his patient. He then assures him that his defect will disappear, and that he can cure himself, and demonstrates to him by correcting the faults that he can speak normally.

2. **LALLING.**—A defect due to want of precision in the action of the oral

articulatory mechanism. It characterises the speech of many children before the art of articulation is completely learnt. It is only a persistent condition in some cases of defective intelligence.

3. LISPING.—A defect due to the indistinct enunciation of certain consonants, or to the substitution of wrong consonants. It usually occurs in connection with the sounds of Th, R, and S, which change to V, L, and Th respectively. The condition, which is almost usual in infants learning to speak, is due to faulty articulation, and may become a habit, in which case the subject has probably a bad “car” for sound. Defective conformity of the mouth may cause it; for example, a “tongue-tied” person can never pronounce the English R correctly.

4. IDIOGLOSSIA.—A condition in which from the first moments of learning to speak, a child uses wrong consonants, or rather he tends to substitute three or four consonants for the whole series. Very slight degrees of idioglossia are common in little children, whose early speech is intelligible only to their nannies. In marked cases the child comes to speak a language entirely its own.

The course of time and education removes the defects of lisping, lalling, and idioglossia, and the prognosis in all these conditions is invariably good.

Any deformity of the articulatory organs should be remedied if possible.

JAMES COLLIER.

Revised by F. M. R. WALSH.

APRAXIA

Definition.—A disorder of cerebral function, characterised by inability to perform certain familiar purposive movements, in the absence of motor and sensory paralysis and ataxia (Kinnier Wilson). This disorder does not depend upon defective perception (agnosia) nor upon general reduction of intelligence.

Ætiology.—Apraxia may result from both general and local diseases of the brain. It may be met with in general paralysis of the insane, in cerebral sclerosis and in several forms of dementia, and in paralytic chorea. It occurs in its purest form from local lesions of the brain, and may then be confined to one region of the body. It may result from lesions of the posterior part of the prefrontal area of the left side, the so-called “motor or verbal” aphasia and agraphia being good examples of apraxia of speech, and lesions in this region may also cause apraxia of the limbs on one or both sides. Lesions of the anterior half of the corpus callosum have been associated with conspicuous apraxia, as have also bilateral lesions in the posterior parts of the hemispheres. In the latter cases, the apraxia is likely to be associated with some degree of lack of recognition of an object, and of its uses (agnosia), and this causes apraxia from a loss of correct comprehension of the act required. Apraxia is sometimes met with in cases of hemiplegia in which, notwithstanding the complete recovery of motor and sensory paralysis, the performance of familiar acts—from the highest skilled movements, such as the fingering of the pianoforte or of the violin, or the use of his tools by a craftsman, to the simplest act—may be no longer possible. The features of the condition may be well demonstrated by the consideration of left-sided

hemipraxis. There is neither loss of power nor loss of sensibility in the left upper extremity. When such a patient is asked to perform some familiar act with the right hand, he at once does so correctly, but when ordered to perform the same act with the left hand he is unable to do so. Either he makes aimless wandering movements with the left hand, or he may succeed in making movements somewhat resembling those required of him, with much slowness and clumsiness. Sometimes he may perform some act which is entirely different from that required of him, and this phenomenon is called *parapraxis*. When the apraxia is partial, the patient may be able to perform some acts and not others, his inability usually, but not always, increasing with the complexity of the act required. Or he may be able sometimes to perform an act in which he commonly fails. Not infrequently such a patient, wearied with the unsuccessful attempts of his left hand, will abruptly perform the act correctly with his right hand, to get rid of it. And he will define his defect by saying, "I know quite well what you want me to do, but I cannot do it." Spontaneous volitional movement is similarly affected, and this leads invariably to a marked loss of initiative in the use of the affected limb—the patient will not try to use it. The apraxic patient is often to an astonishing degree unaware of his disability, and frequently becomes conscious of it for the first time when it is pointed out to him by another person.

Diagnosis.—Apraxia may be confused with astereognosis, with agnosia and with cortical ataxia. A correct conception of the nature of the two former conditions will exclude the possibility of error. In cortical ataxia the patient obeys the word of command at once and succeeds more or less with the act required, the defect being clumsiness of execution. The clinical examination of patients for apraxia must include—(1) the general psychological condition as regards attention, memory and reasoning; (2) an inspection of sensory appreciation for defects of simple perception in the regions of smell, sight, hearing, taste, cutaneous sensibility and muscular sense; defects of recognition of sensory impressions in these regions (agnosia); defects of memory; and (3) an examination of executive power for any defects in the movements determined by visual, auditory, tactile and kinæsthetic stimuli. What response does the patient make to objects held in front of him or to gestures made to him? Can he imitate movements? Can he when requested make simple and purposive movements, with and without the objects in his hands? When given an object, how does he hold it and use it?

AGNOSIA

In certain conditions of cerebral disease, it is found that each and all of the sensory organs, when called into play, may fail to arouse an intelligent perception of the object exciting them. This inability to recognise the import of a sensory stimulus is called *agnosia*. Those patients who present apraxia and agnosia, often show other interesting phenomena which are of importance; these are (1) inattention, (2) defective capacity for retaining recent impressions, (3) lack of initiative, and (4) perseveration. Perseveration consists in the repetition of an already executed movement when and only when the patient desires to make a fresh movement.

CEREBRAL DIPLEGIA

Synonyms.—Congenital Spastic Paralysis ; Lobar Atrophic Sclerosis.

Definition.—A series of clinical conditions, dependent upon lack of, or imperfect development, or degeneration of the nerve cells of the cerebral cortex, basal ganglia or cerebellum. This agenesis of nerve cells may affect those cells of the pyramidal system which are the latest to develop before birth, namely those for the supply of the lower extremities and the resulting clinical condition is cerebral spastic paraplegia or Little's disease, or all the cells of the pyramidal system may be affected, producing generalised spastic rigidity. Again, the higher regions of the cortex may be affected, and the result is congenital idiocy. Similar affections of the cells of the basal ganglia result in congenital bilateral athetosis, and congenital chorea. When the cerebellum is involved, congenital cerebellar ataxy results. Further, there may be any combination of the above conditions.

Ætiology.—The malady may be apparent at the time of birth, as the child may be born with contractures present. More often, the signs of deficient or perverse movement, or of mental deficiency, appear during the first year of life, as the signs of cerebral activity commence to be exteriorised. In other and rarer cases, the degeneration of the nerve cells seems to be truly post-natal in onset, as in amaurotic family idiocy. In most cases no heredity can be traced, but sometimes several children of the same mother may be affected, and direct heredity has been known. Amaurotic family idiocy is always familial, and is almost, but not quite, limited to the Hebrew race.

Abnormalities of birth are frequent. Premature, or precipitate birth, prolonged birth from uterine inertia rather than from dystocia, and asphyxia neonatorum are all common. The child is frequently the first born of its mother.

Collier has expressed the probable pathogenesis of cerebral diplegia as follows : "If we regard the brain from the time of its earliest stages of development as a field sown with seeds (neuroblasts), which germinate at different periods of foetal life, and the germination is not even complete at the time of birth, the germination of all the elements in due time and their complete development being necessary for the formation of the perfect brain, then we may liken the cause of diplegia to some baneful influence, such as a frost, which acting at a particular time, may spare those seedlings which are well developed and able to withstand it, and those seeds as yet not germinated, but which causes havoc among the tender germinating seedlings, either to their death or severe maiming. In some cases, as, for example, in Little's disease, the neuroblasts thus affected may, after a period of retarded development, ultimately become strong plants and complete their development. It is of interest that in the highest degrees of cerebral agenesis—anencephaly, pituitary abnormalities seem to be constant."

Pathology.—The essential histology of the affected regions is that of non-development, paucity in numbers and degeneration of the nerve cells, with corresponding absence, poor development, degeneration or a combination of these states, of the tracts which spring therefrom. The pyramidal tract, for example, may be found absent throughout, or it may reach to the medulla,

or to the cervical region only, and so show at what period development was arrested. The changes in the nerve cells are followed by secondary gliosis. The final result is termed atrophic sclerosis. More often certain regions are profoundly affected, while others escape relatively or completely; but the distribution is always symmetrical upon the two hemispheres. The convolutions are unduly hard to the touch, and their surfaces often present a worm-eaten and faceted appearance. This irregular form of the convolutions, with wide, separating sulci, gives the brain a characteristic appearance, like that of a walnut kernel.

Symptoms.—The clinical picture of the several forms of cerebral diplegia presents a combination in varying degrees of certain characteristic symptoms, always bilaterally distributed, though sometimes more severe on one side than on the other. These symptoms are: muscular rigidity, paresis, perverse movements, contractures and increased deep reflexes. Mental deficiency, optic atrophy and ataxy are other important symptoms. The signs of the disease become obvious during the first year of life or soon after. In severe cases, soon after birth, the nurse, in washing the child, is the first to notice the stiffness of the limbs, or the regular assumption of a curious bodily attitude. Otherwise, the abnormalities may not be obtrusive, until the child should sit up or learn to get about, when weakness, rigidity, perverse movements and pes cavus may call attention, or backwardness in learning to walk and to talk, and mental deficiency may first suggest that there is something wrong with the child. The following are the common types of the disease, but it must be remembered that any combination of, or transition between, the types may be met with. Cerebro-macular degeneration has certain peculiar features which necessitate a separate description for this malady:

1. *Generalised rigidity; general congenital spastic paralysis.*—There is extensive defect of the pyramidal system. The rigidity and weakness affect the whole of the musculature.

2. *Paraplegic rigidity; congenital spastic paraplegia; Little's disease.*—The pyramidal deficiency is confined to that supplying the lower part of the trunk and lower limbs.

3. *Congenital bilateral athetosis and congenital chorea.*—The agenesis affects the cells of the basal ganglia, with the appearance of irregularity of movement, and of spontaneous involuntary movements, which may be of an athetotic, choreic or irregular type. A certain variable degree of general rigidity is present in these cases.

4. *Congenital cerebellar ataxy.*—The agenesis affects the cerebellum with the appearance of cerebellar ataxy. In this type, the limbs are flaccid, and in mixed cerebral and cerebellar types there is a tendency to hypotonicity of the muscles, instead of rigidity.

5. *Congenital idiocy; restless idiocy.*—The agenesis affects those parts of the brain concerned with the higher functions. These children are emotionless, restless and unteachable. The skull often shows frontal or occipital microcephaly.

6. *Microcephalic idiocy*—where the agenesis is of the whole brain and the skull very small.

7. *Cerebro-macular degeneration.*

PARESIS AND RIGIDITY.—Except in severe cases, in which the weakness

amounts to complete paralysis, there is more rigidity than weakness, and it is often astonishing that there should be so much power in the presence of such a degree of rigidity. The lower extremities are generally the most affected, the upper to a less degree, and the facial region still less. Movement is slow and clumsy and resembles that of the tardigrade animals, and spontaneous involuntary movements are often present in the limbs. Contractures accompany the rigidity, and if walking is possible the gait is digitigrade from contraction of the calf muscles, the knees are flexed from contracture of the hamstrings, the thighs are rotated inwards, and the knees pressed together, rubbing against one another. More severe adductor spasm gives rise to the cross-legged progression. The rigidity and contractures, when severe, may give rise to peculiar attitudes and deformities. A mask-like expression of face, with wide palpebral apertures and large open mouth, is not infrequent. Slobbering is very common. The head may be rigidly retracted, but more commonly the chin is pressed down upon the chest. The spinal column generally shows some deformity in the way of kyphosis, lordosis or scoliosis, and pes cavus or equino-varus is the rule.

PERVERSE MOVEMENTS.—Under this heading must be grouped the very constant maladroitness of voluntary movement, the facial over-action and grimacing in speech and in mimetic expression, choreic movements, athetotic movements and intention tremor. Common sensation and the muscular sense are unimpaired. The sphincters are unaffected. The deep reflexes are increased, but are often difficult to obtain when rigidity is very marked. The trunk reflexes are often absent, the plantar reflexes usually are extensor in type. Since the growth of the skull follows and conforms with that of the brain, cranial abnormalities are common. There may be microcephaly, asymmetry and flattening in the region of the central convolutions, or a furrow corresponding with the interhemispheric fissure, or frontal or occipital smallness and flattening. Every degree of mental reduction may be met with, from precocity and slight mental dullness to complete amentia. But this by no means corresponds with the severity of the bodily symptoms, for the mental defect is often most severe when the bodily symptoms are slight, and conversely. In some cases, very high intelligence persists, when there is utter uselessness of the limbs, and when speech is hardly intelligible. Primary optic atrophy occurs in a small number of cases. Inequality of the pupils and slowness of light reaction are not uncommon. Nystagmus is often met with. Convergent strabismus occurs in about one-third of the cases. Convulsive attacks are of common occurrence, and in about one-eighth of the cases epilepsy becomes established.

Diagnosis.—When the symptoms are well marked, the diagnosis presents little difficulty, since the disease dates mostly from birth, or is discovered during the first year of life. Paraplegic rigidity may possibly be confused with other forms of paraplegia, and, especially, with that resulting from spinal caries. Certain cases of pontine tumour may closely resemble generalised rigidity. The occurrence of such conditions during the first two years of life is, however, very rare.

Prognosis.—In many cases of generalised rigidity, and in all cases of paraplegic rigidity, there is a tendency to slow amelioration of the rigidity, an increase of voluntary power and control of the muscles in the course of time, especially under the influence of careful training, and in paraplegic

rigidity, if the mental acuity be not seriously impaired, laborious treatment may result in an almost normal condition of the limbs by the age of puberty. On the other hand, some cases of generalised rigidity become progressively worse, and succumb, usually before the end of the fourth year. Bilateral athetosis and choreic diplegia, as a rule, follow a very slowly progressive course, without tendency to a fatal result. Paraplegic rigidity apart, a great many of the cases of all forms of diplegia succumb before the sixth year, and in those who survive this age, the tenure of life is short, few reaching far into the third decade of life.

Treatment.—In those cases with a marked degree of mental impairment, and in those which show a course of progressive degeneration, no treatment is of avail. In slighter cases of generalised rigidity, and in paraplegic rigidity, treatment is to be directed to the prevention of the rigidity, to regaining of voluntary control, and the improvement of mental acuity. There is, perhaps, no disease which demands greater patience and persistency in carrying out of suitable treatment, and there are few diseases in which more brilliant results may be produced from apparently hopeless cases by pertinacity in treatment. It is in the early years, when treatment is for the most neglected, that good results are more quickly and readily obtained. From the first, regular massage and passive movements should be employed. Voluntary movement should be encouraged, as far as possible, and as power and movement increase, gymnastic exercises of every kind should be employed. Rigid apparatus for prevention of deformity and to reduce contracture is harmful, for it increases the weight of the limb, and interferes with movement, which is the remedy with which paralysis is to be combated. Tenotomy is of great service in the relief of deformity and contracture, and should be soon followed by passive movements. It should never be performed, unless a fair degree of voluntary power is present. Many of the patients seem to improve more rapidly if thyroid be administered in moderate daily doses.

CEREBRO-MACULAR DEGENERATION: AMAUROTIC FAMILY IDIOCY

1. WARREN TAY-SACHS' DISEASE: THE INFANTILE FORM

Definition.—A family disease of infancy occurring chiefly, but not entirely, in the Hebrew race, affecting children during the first year of life, who are apparently quite healthy when born, and characterised by—(1) progressive mental impairment, ending in absolute idiocy; (2) progressive paralysis of the whole body; (3) progressive diminution in sight, ending in absolute blindness. Pathognomonic retinal changes are constantly present, consisting of a large and conspicuous "cherry-red spot" in the region of the macula, and, in addition, optic atrophy occurs later and (4) a fatal termination in the marasmic state before the age of 2 years.

Ætiology.—Nothing is known of the ætiology of the disease apart from its familial and racial incidence. The tendency to the disease is unquestionably congenitally installed.

Pathology.—This is very striking. It consists of a progressive degeneration of the nerve cells from the highest to the lowest, and ultimately there may be no normal cells remaining anywhere in the nervous system.

The degeneration takes the form of swelling of the cell protoplasm, and of the dendrites with chromatolysis, swelling of the hyaloplasm and destruction of the cell fibrils, followed by disappearance of the nucleus, and finally by absorption of the remains of the cell. Every cell of the central nervous system both of the brain, spinal cord and spinal ganglia is in the end similarly affected.

Symptoms.—There are few diseases in which the *clinical manifestations* are so perfectly uniform as in this malady. The children have all been born at full term, and in perfect health. They thrive well during the first 3 to 6 months of life, when they gradually become listless and apathetic, cease to take interest in the surroundings, and begin to show signs of the visual failure which ends in blindness. Later, the child is unable to sit up, or to hold up its head. The limbs, which may be slightly spastic at first, become flaccid and motionless. There is a gradual increase of all these signs. The mental defect becomes more and more noticeable, the paralysis more extreme, complete blindness follows, and the patient sinks into a condition of marasmus, in which he dies. Convulsions, nystagmus and strabismus are sometimes present.

The retinal changes are pathognomonic and are due to a degeneration and disappearance of the nerve cells of the retina and their processes, which constitute the fibres of the optic nerve. This change is most intense in the region of the fovea centralis, where the retina thins and disappears over a circular area, exposing the vascular choroid. This gives rise to the characteristic appearance, on ophthalmoscopic examination, of a cherry-red spot in the region of the macula. This spot is actually a hole in the retina exposing the choroid. The optic disk shows progressive atrophy.

Diagnosis.—Distinction has to be made between this and other forms of progressive diplegia. The symptoms are so distinct that a physician, who is acquainted with the disease, and able to recognise the retinal picture, can hardly fail to make the correct diagnosis.

Treatment.—No treatment is of any avail.

2. OTHER FORMS OF CEREBRO-MACULAR DEGENERATION

In addition to the classical infantile form described in the preceding article, two other forms are well known in which the pathological changes are similar but much less severe than in the Waren Tay-Sachs' disease, and there is a similar familial incidence, but the incidence of the malady occurs later in life and the course is less rapid and the result far less serious. The later the onset in life the slighter and less progressive are the symptoms. The cherry-red spot at the macula, so constant in the infantile form, does not occur in the later forms. The characteristic retinal change is a disturbance of the retinal pigment commencing in the macular region, rather like retinitis pigmentosa, accompanied by honeycomb changes at the macula and sometimes by optic atrophy. The *juvenile* form occurs in later childhood and is characterised by the association of the retinal changes and visual defect with some degree of mental deterioration. The *adult* form is the least progressive of any, and the clinical manifestations are the visual defect and retinal changes in the absence of mental deterioration.

INFANTILE HEMIPLEGIA

While in childhood hemiplegia of slow onset is due to the same causes as in adults, cerebral tumour being the common cause and chorea not an infrequent one, yet the majority of the cases of infantile hemiplegia of rapid onset are examples of diseases peculiar to children, to which no comparable disease occurs in adults, and to such cases the term "infantile hemiplegia" is restricted. These conditions are due to gross organic lesions of the brain, and for this reason must be strictly separated from the cerebral diplegias which are the result of cell lesions and not of gross lesions.

Ætiology.—In two-thirds of all the cases, the onset occurs within the first three years of life. The malady becomes increasingly rare as childhood advances. A few of the cases are of prenatal origin, and some of these have been proved to have been due to injury to the fœtal brain from a blow upon the mother's abdomen, while others are due to syphilitic fœtal vascular disease. In a third class of mysterious origin, mothers have given birth to several hemiplegic children, examples of which we have recorded. Some of these children are born with definite hemiplegia and contractures. Again, a very few cases are due to obstetrical events during birth, by which the cerebrum is injured. Acute infective diseases play a very important rôle in the causation of the disease, for about one-third of all the cases develop the malady during the course of a known infection. By far the most important of such fevers are measles and scarlet fever, but hemiplegia may occur in the course of pertussis, small-pox, röteln, diphtheria, dysentery, pneumonia, typhus, typhoid, mumps, malaria, chorea and endocarditis. While there can be no doubt that primary vascular lesions are responsible for a few of the cases in which this condition complicates the specific fevers, whooping-cough, for example, may cause cerebral hæmorrhage; marasmic conditions in any fever may cause thrombosis of cortical veins, and chorea and endocarditis may cause embolism, yet it is certain that in the majority of cases, an inflammatory focal lesion of the brain or encephalitis is the pathological lesion. In cases which arise with no definite ætiological connection, it seems clear that a primary encephalitis is responsible, but there is no evidence at present as to its causal factors.

Pathology.—The following lesions are met with, either alone or combined in order of frequency: (1) Atrophic sclerosis; (2) cyst formation; (3) shrunken patches resembling wet wash leather, with some degree of atrophic sclerosis in their vicinity, and (4) porencephaly. Of these, the atrophic conditions seem to be the results of encephalitis, which may also cause some cyst formation; the cystic conditions may result from the above, or from hæmorrhage or thrombosis, and porencephaly is certainly due to embolism.

Symptoms.—The onset is rapid, and in two-thirds of all the cases the disease is ushered in by convulsions, which may be unilateral, but are more frequently general, and are frequently repeated during a period of from a few hours to 24 hours, after which the patient sinks into a subconscious state, from which he gradually emerges in the course of a few days, to show the signs of some cerebral defects, usually hemiplegia, sometimes hemianopia, or aphasia, or any other sign of local cerebral or cerebellar lesion. Pyrexia

often accompanies the convulsion, and vomiting is common. The onset may be without convulsions or loss of consciousness.

The relation of the onset of the paralysis to the convulsion varies. It may reach its height immediately after the initial convulsion, or slight hemiparesis may occur which deepens after each subsequent convulsion. Sometimes the early convulsions leave no paralysis, but this appears towards the end of the first week, either suddenly with fresh convulsion, or gradually, as the patient recovers from the comatose state. The paralysis at its onset is flaccid, and involves the whole of one side of the body to a greater or smaller extent. An initial monoplegia is of extreme rarity. The paralysis may not reach the greatest intensity until the end of the second week. Subsequently it lessens, in some cases disappearing completely in from a few weeks to 3 months; in others, it may show no signs of improvement. The limbs, at first flaccid, subsequently become spastic and develop contractures. In the course of years there may be great arrest of growth on the affected side, and this is not in relation with the degree of paralysis, but apparently depends upon the degree of destruction which has occurred in the parietal lobule. Post-hemiplegic spontaneous movements of an athetoid, choreic or irregular kind are common, and are attributable to lesions in the corpus striatum and subthalamic grey matter, for which regions encephalitis shows an especial predilection. Epileptic fits recur at varying intervals in about half of all cases of infantile hemiplegia. These always commence upon the affected side and are sometimes confined to it. Mental deficiency is met with in all degrees, in relation to the position and extent of the cerebral cortex which is involved in the lesion.

Diagnosis.—The nature of the malady at the onset, with convulsions, may be possibly suggested by prodromal pyrexia, by the severity and long duration of the convulsions, and by the prolonged subconscious state that often follows. Convulsions occurring several days after the onset of specific fevers should strongly suggest the diagnosis. When the signs of hemiplegia or of other local cerebral lesions appear, the diagnosis presents no difficulty.

Course and Prognosis.—In a very small proportion of the cases the patient does not survive the initial manifestations of the disease, and dies in convulsions. Apart from this event, infantile hemiplegia has little tendency to destroy life. The initial flaccid hemiplegia tends to improve and gives place to a slowly improving spastic hemiplegia, which, with the return of some power, shows perversity of movement, stiffness and slowness, ataxy, athetosis and choreic movements or tremors according to the position of the lesion. The spontaneous movements appear within a year of the onset. Slow improvement may go on for years, but cases with much mental reduction or when recurring epilepsy is frequent, improve but little.

Treatment.—We know of no measures that avail to prevent the occurrence or lessen the severity of the cerebral destruction which occurs from encephalitis. Too often the damage to the brain has happened as soon as a diagnosis is possible. When the paralysis has developed, treatment is to be directed to the prevention of rigidity and contractures by regular passive movements, to regaining voluntary control by encouragement and patient exercises, and to the improvement of mental acuity. Where there is much contracture and deformity, tenotomies are of great service, provided there be some voluntary power in the muscles, the tendons of which are

to be divided. Recurring convulsions should be treated as idiopathic epilepsy.

SYPHILIS OF THE NERVOUS SYSTEM

Of the many problems presented by syphilis of the nervous system one may be chosen for special reference, namely, the alleged dual nature of nervous syphilis and of the causative organism. It has been maintained that there are two distinct pathological forms of nervous syphilis, namely, primary *parenchymatous* syphilis, as exemplified in tabes and general paralysis, where the initial lesion is held to be in the nerve elements themselves, and primary *meningo-vascular* syphilis, in which the initial lesion is in the blood vessels and meninges. But the dual nature of syphilis implied by these distinctions is subversive of pathological principles, for the initial and fundamental lesion of syphilis, wherever found and at all stages, is a lymphangitis or an arteritis, and very strong evidence would be needed to enforce the conviction that the reactions of nervous tissues to the presence of the spirochæte differs essentially from that of all other tissues of the body. Nor is this view supported by the morbid anatomy of the diseases concerned, for in every case of tabes and general paralysis, vascular and meningeal lesions can be found after death. Moreover, in the vast majority the increased number of cells in the cerebro-spinal fluid shows that the meninges are attacked even in the earliest stages.

For these reasons the trend of opinion is to deny the existence of parenchymatous syphilis as an initial lesion of syphilis in the nervous system, and to hold that the vessels and meninges are first injured in all forms of syphilis of the nervous system.

The contention that the organism of syphilis exists in two forms next demands consideration. Of all syphilitics the proportion in whom the nervous system is attacked is small. To explain this low incidence it has been assumed that neuro-syphilis results from infection by a biological variant of the *Spirochæta pallida* with special affinities for nervous tissues—the “neuro-tropic variety,” while other forms of syphilis follow infection by the “dermo-tropic variety.” At first sight this attractive conjecture seems to be supported by numerous clinical observations. These are, that in some instances several persons infected from the same source have later developed syphilitic nervous diseases; that the superficial manifestations of syphilis are often mild or absent in those who ultimately develop neuro-syphilis, and that secondary and tertiary syphilis, outside the nervous system of these patients, is rare; also that in Oriental countries where syphilis is common, some forms of neuro-syphilis, namely, tabes and general paralysis, are seldom seen.

But it has never been shown that the persons who formed the source of infection for several cases of nervous syphilis have themselves developed this disease; it can no longer be held that tabetics and paralytics are free from secondary and tertiary lesions—witness the frequency of aortitis in these cases; finally, Europeans who contract syphilis in the East are just as likely to suffer from tabes and general paralysis as if they had contracted

it at home. It cannot be denied that the secondary phenomena are often so slight as to pass unnoticed by patients who later become tabetics or paralytics, or that patients with severe integumental lesions rarely develop tabes or general paralysis. These facts, however, together with a large amount of additional evidence, suggest that the ultimate result of infection depends rather on the individual attacked than on any peculiarity of the infecting agent, and they lend no support to the notion of the duality of the syphilitic organisms.

For the purpose of description it is still convenient to describe syphilitic diseases of the nervous system under two headings; Interstitial or meningo-vascular syphilis and parenchymatous syphilis. To avoid misunderstanding it must be emphasised that these are merely clinical aspects of one disease—neuro-syphilis, and that in both forms the primary lesion is in the vessels and meninges.

THE BLOOD AND CEREBRO-SPINAL FLUID IN SYPHILIS OF THE NERVOUS SYSTEM

A normal fluid may be regarded as one with a pressure equal to 150 to 180 mm. of water, a cell count not exceeding 5 per c.mm., an albumin content of from 0.025 to 0.05 per cent., and negative Wassermann, globulin and Lange's gold tests.

In *secondary syphilis*, without nervous symptoms, changes are found in the fluid in 80 per cent. of the cases. The infection of the nervous system occurs early in the second stage of the disease and it is a common happening. In many of the cases this infection dies out in the course of time either spontaneously or as the result of treatment and the cerebro-spinal fluid reverts to a normal condition. In other cases the infection remains and the W.R. continues positive in the cerebro-spinal fluid, and it is exclusively in this class of patient that tabes, general paralysis and the other degenerative maladies of the nervous system which are due to syphilis arise. Increased pressure, lymphocytosis, excess of albumin or a positive Wassermann reaction are found with a frequency which diminishes in this order. If the blood and fluid are both normal at the end of a year, neuro-syphilis is not likely to arise.

In *latent syphilis*, without nervous symptoms, the blood is positive in about 70 per cent., but changes in the fluid are found in 20 per cent. only. A fluid which is normal in the latent stage almost never becomes pathological later; if it deviates from the normal it is highly probable that neuro-syphilis will develop later.

In *early cerebro-spinal syphilis* the blood is usually positive, but it is sometimes negative when treatment has been thorough or recent. This indicates that an examination of the fluid is essential when the blood is negative in a case where the diagnosis is doubtful. Cells and albumin are usually greatly increased, the gold test is usually positive, and the Wassermann reaction is positive in almost every case when 1 c.c. of fluid is used in making the test. The blood and fluid often become normal after the first course of treatment, but a relapse occurs frequently and long before the recurrence of symptoms. Vigorous mercurial treatment and 4 or 6 full courses of arsphenamine at intervals of 3 months usually render the blood and fluid persistently negative. If both are normal a year after the fourth course, recurrence is unlikely.

In *cerebro-spinal syphilis* of longer duration, the reaction in the blood is

almost always positive, and the fluid is seldom normal in active cases. A positive reaction in the fluid probably indicates an active process, even in the absence of recent symptoms. A normal fluid indicates a healed process. The inference is that all cases with a positive fluid and a history of cerebro-spinal syphilis should be treated energetically whether fresh symptoms are present or not. The treatment outlined above under early cerebral syphilis usually renders the fluid negative and the indications for prognosis are the same.

In *tabes* Wassermann's test is positive in the blood in about 70 per cent. of cases; it is often negative in the fluid when the test is done by the original method with 0.2 c.c. of fluid, but with 1 c.c. it is positive in almost 100 per cent. An increase in the number of lymphocytes in the cerebro-spinal fluid as well as an increase in the total albumin with a relative increase of globulin is almost constant. In some cases in which the disease had remained stationary for a long time the cerebro-spinal fluid was normal in every respect.

Lange's colloidal gold test gives useful information when the diagnosis lies between *tabes* and general paralysis, for the latter gives a typical reaction, but the test is of no value by itself in distinguishing *tabes* from other diseases of the nervous system (see page 1558).

Treatment by mercury and intravenous injections of neoarsphenamine sometimes renders the fluid normal. It is stated that in such instances relapse is less likely to occur than in other cases, but no exact correlation has been shown to exist between the clinical course of the disease and the presence or absence of changes in the blood and fluid.

General paralysis of the insane.—The Wassermann reaction is strongly positive in the blood and in the fluid practically in 100 per cent. of cases, and an increase of cells and a positive globulin reaction are almost constant. Lange's gold test gives the characteristic parietic curve.

Treatment by mercury and arsphenamine may produce slight modification in the reactions, but it has very little effect in checking the progress of the disease.

THE ESSENTIAL LESION OF SYPHILIS

Every lesion in syphilis commences with the collection of spirochaetes in the lymphatic spaces surrounding small arteries. This is followed by an inflammatory reaction with oedema and exudation of many lymphocytes and plasma cells around the small vessels, and the "cuffing" or "muffing" of these vessels with such cells is characteristic. These cells may wander freely into the nervous tissue away from the vessels and may form clumps, often containing giant cells, and these are miliary gummata. Such a perivascular lymphocytic exudation is typical of syphilis, poliomyelitis, tuberculosis and lethargic encephalitis, but the distinction can be made by the nature of certain histological elements present. Syphilis is distinguished by the presence of numerous plasma cells among the lymphocytes, poliomyelitis by the large admixture of polymorphs, tubercle by the absence of plasma cells and the presence of Koch's bacilli, and lethargic encephalitis by the absence of any elements except the lymphocytes. The initial periarteriolitis of syphilis is often followed by invasion of the whole vessel wall (panarteritis), and often proliferative endarteritis which may give rise to thrombosis is the most conspicuous feature in the panarteritis. Later, the wall of the vessel may scar and may develop patchy calcareous deposit. The lymphocyte

deposit goes into fibrosis or increases to gumma formation, and there is neuroglial felting. Further thrombosis of the vessel may cause the softening and infarct conditions which necessarily follow vascular obstruction, and local necrosis results. The hyperproteinia and pleocytosis of the cerebro-spinal fluid are expressions of this essential lesion upon the surfaces and spaces of the nervous system, and the meningeal scarring and adhesion are its results.

So far the pathology of nervous syphilis is simple, but the so-called "parenchymatous" or degenerative lesions which are apt to be widespread and progressive are as yet inexplicable. They are commonly found in the absence of any findable spirochæte, or of any sufficient inflammatory lesions in their locality, and have been observed also by Carey Coombs in the heart muscle and the aorta, and may be progressive when all signs of active syphilis such as the W.R., hyperproteinia and pleocytosis have died out finally. These degenerative lesions may not be improved or stayed in their progress by any form of treatment. The slowly oncoming progressive and unarrestable optic atrophy and the systemic lesions of the spinal cord in tabes are good examples of the degenerative lesions, and in them spirochætes have rarely or never been found, while the inflammatory lesions are absent or minimal and cannot be the factors of so wide a destruction. The nearest locus where spirochætes are commonly found in tabes is the bronchial glands.

CEREBRO-SPINAL SYPHILIS

Ætiology.—Cerebro-spinal syphilis (excluding tabes and general paralysis) occurs in about 4 per cent. of all persons who acquire syphilis. The onset of symptoms is commonest from 1 to 5 years after infection, but it may be as early as 2 or 3 months, or as late as 30 or 40 years. The brain is affected more often than the spinal cord, and usually when the main symptoms point to the latter some signs will be found to show that the brain is also attacked.

1. CEREBRAL SYPHILIS

Pathology.—The disease may begin in the meninges, in the blood vessels or in the bones of the skull. In cases where the main incidence falls upon the *blood vessels*, the arteries at the base of the brain forming the circle of Willis or arising from it, together with their branches, are most often attacked. To the naked eye they show irregularities in size, due to thickening of their walls in circumscribed areas. Proliferation of the intima with a round-celled infiltration of the outer coats—*endarteritis obliterans*—is the characteristic microscopical change. The same changes occur in smaller arteries within the brain or on its surface, and these vessels, as well as those at the base, may be compressed or invaded by disease beginning in the meninges. In each case their lumen is narrowed or obliterated, thrombosis occurs readily, and softening may result in parts cut off from their blood supply. Obliterative changes also occur in the veins and lymphatics, and lead to further impairment of the nutrition of the brain.

The commonest form of *meningeal syphilis* is a diffuse gummatous leptomeningitis at the base of the brain. On the convex surface of the brain

it begins most often over the frontal and parietal lobes. The meninges may be affected alone, but more often the vessels are also diseased. In severe cases a gelatinous exudate fills the sub-arachnoid space and extends along the vessels and nerves. Later, the newly-formed tissue organises, and forms sclerosed masses of thickened adherent membranes containing numerous small gummata.

Gumma of the brain is rare. When present it arises from the meninges, and may be multiple. The convexity of the hemisphere in the motor region is the site of election. A gumma may spread so as to involve the overlying bone.

Many important results of cerebral syphilis are due to impaired nutrition of parts not directly affected. Thus softening, hæmorrhage, cyst-formation, atrophy of cells and tract degenerations may occur, and non-syphilitic diseases may be simulated.

Symptoms.—The main incidence of the disease may fall upon vessels or membranes, the lesions may be diffuse or circumscribed, any portion of the brain or any cranial nerve may be affected alone, and every combination of lesions and, therefore, of symptoms, is possible. In many cases their multiplicity, their presence in unusual combinations, and the changes in their intensity and distribution from time to time give a clue to their nature.

Headache is a common prodromal symptom. It is often severe, and is usually worse at night. Sustained mental and physical effort becomes difficult, the memory is impaired and the character changes. Irritability, intense excitement or delirium may follow, but more often the patient becomes lethargic. At this stage paralysis and localising signs may be absent. In many the pupils are unequal or irregular, or they contract sluggishly to light, and in some the optic disks show blurring of the edges and other signs of early papilloedema.

At any time, with or without prodromata, more definite signs of vascular disease or of paralysis of one or more of the cranial nerves may appear. *Arterial thrombosis* is usually preceded by prodromal symptoms, but it may come on in one apparently well. Its seat of election is the middle cerebral artery or its branches, and weakness of one arm or of one side of the body with or without aphasia is a very common early symptom. The weakness is often slight and transient. If hemiplegia occurs, it takes several hours or a day or two to develop. It is more often a paresis than a paralysis, and consciousness is usually retained. Sometimes the affected limbs are rigid and tremulous. The symptoms of thrombosis in other arteries are given on p. 1559.

In *vertical meningitis*, headache is usually severe, and the skull is often tender over the affected part. It frequently attacks the motor areas, and convulsions are common. They may be confined to one limb or to one side of the body, consciousness being retained, but more often they become generalised and consciousness is lost. In more chronic cases mental symptoms may predominate, the patient becoming slowly demented.

With *basal meningitis* severe deep-seated headache is almost always present. Later, a characteristic lethargy with severe impairment of the mental functions may appear. Whilst in this stuporose state the patient can usually be roused, when he answers questions in a sleepy fashion and obeys simple commands, but his memory is bad, and he is unable to give an

adequate account of himself. Variations in the degree of the stupor are a striking feature. In some cases profound torpor may persist for several weeks.

Soon after the onset, signs of implication of the cranial nerves appear. Any one of them may be affected alone, but, as a rule, several are attacked, where they lie close together after their exit from the brain, or as they leave the skull through the foramina. Ocular symptoms are rarely absent. Inequality in size, or irregularity in the outline of the pupils, may be the only sign, but diminution of the light reflex, ptosis, squint, diplopia and weakness of the movements of the eyeball are frequent, and papilloedema is common. It is characteristic of the cranial nerve palsies in syphilitic basal meningitis that the symptoms often show great variations in degree and distribution at different times.

The symptoms produced by large *gummata* are those of any cerebral tumour—headache, vomiting and papilloedema.

Diagnosis.—The aphorism, probably justified 25 years ago, that syphilis should be thought of in every case of nervous disease that is not quite clearly non-syphilitic in origin, has long outlived its usefulness as a generalisation. But it still lingers and is responsible for the practice, far too general, of throwing the burden of neurological diagnosis upon the shoulders of the clinical pathologist to make blood and cerebro-spinal fluid examinations, as a substitute for that careful history taking and clinical examination that would usually suffice for a complete diagnosis without these accessory investigations. Neuro-syphilis should not be guessed at in a haphazard fashion, but the attempt should be made by the clinical methods mentioned above to assess the pathological nature of the lesion or disease process in any case of nervous disease, and to use serological methods to confirm clinical diagnosis, or to clear up uncertainty when this still obtains after full clinical consideration. A history of syphilis or signs of the disease elsewhere are of first importance. Obstinate headache alone should arouse suspicion, and when signs of vascular or cranial nerve troubles follow a prodromal period of headache and mental impairment, syphilis is the most likely cause.

Jacksonian epilepsy or fits of any kind in patients without an epileptic history are suggestive of syphilitic meningitis or gumma, even though these are not the commonest causes of these symptoms. As a rule, these convulsions are readily distinguished from those of idiopathic epilepsy, by their partial distribution, by the weakness which remains in the parts which were convulsed, and by the presence of papilloedema, of cranial nerve palsies, or of other signs of organic disease which are found in the intervals between the fits. Cerebral new-growths produce similar symptoms, but this diagnosis should not be finally accepted until syphilis has been excluded. In all cases an examination of the blood and *cerebro-spinal fluid* is essential (see p. 1629) because a positive Wasserman or Kahn reaction in the blood may be taken to indicate no more than that the subject has at some time had syphilis. It does not usually mean that the tumour is syphilitic in nature. It is on the whole less rare to find a glioma in a syphilitic subject than to find a gumma of the brain. Only the presence of the changes characteristic of neuro-syphilis in the cerebro-spinal fluid, as well as in the blood, can justify the diagnosis of cerebral gumma.

Prognosis.—The outlook in cases with mild symptoms is good if the treatment is efficient. In severe cases of syphilitic arteritis it is grave. The vessels most often invaded are large end-arteries, and the softening which results when their lumen is occluded is not amenable to anti-syphilitic remedies. Relapses are common, and patients apparently cured are often seized in a few months with fresh cerebral troubles. The lesions in the meningeal and gummatus forms of the disease are mainly cortical, and large areas of softening are not produced. The prognosis is much better in this form, and complete cure is almost the rule when treatment is begun early. It is often impossible, however, to decide from the clinical signs whether the vessels or the meninges have suffered the more. In the absence of this knowledge the prognosis depends on the results obtained by the use of appropriate remedies.

Treatment.—See p. 1636.

2. SPINAL SYPHILIS

Pathology.—The disease may begin in the bony or membranous coverings of the cord, in the blood vessels on its surface, or in the interstitial tissues within its substance, and spreading from one to the other, usually attacks several of these structures in various combinations. As in cerebral syphilis, many of the changes in the cord are secondary to disease in the vessels or meninges, and appear in parts not directly attacked by specific processes. The commonest form of spinal syphilis is *meningo-myelitis*. The meninges are thickened and adherent, while the spinal and meningeal arteries and veins show obliterative changes, and are surrounded or infiltrated by small round cells. The same changes are apparent in and around the vessels and pia septa within the cord. In severe cases, the membranes are united to form a thick fibrous sheath around the cord, and the outlines of the nervous structures as seen in transverse section are almost obliterated by the presence of numerous small gummata or of myriads of small round cells. The nervous elements are compressed by the cell infiltration, or undergo softening or necrosis as a result of obliteration of the blood vessels. Meningo-myelitis is usually confined to a narrow area in the dorsal region, and is often associated with a more extensive meningitis.

In some cases with severe arterial changes, extensive softening results from thrombosis or hæmorrhage and produces severe paralysis of sudden onset—*acute syphilitic myelitis*.

Large gummata are rare. They occur in the cord, or on its surface, and produce the signs of compression.

In another rare form in which the membranes alone are affected—*pachymeningitis* and *leptomeningitis*—the dura and pia-arachnoid unite to form a thick fibro-gummatous sheath around the cord to which they become adherent.

Syphilitic disease of the vertebral column is very rare. It produces changes which resemble those of tuberculous spinal caries, with osteitis and periostitis, and the formation of granulation tissue and gummata on the outer surface of the dura. Necrosis of the bones may lead to deformity of the spine. It is seen most often in the cervical region, where it begins in the spine, or spreads from a syphilitic ulcer in the throat.

Symptoms.—In *meningo-myelitis*, pain in the back, tenderness of the spine, and radiating pains or a feeling of constriction in the limb or around the trunk, are often present in the premonitory stage. After these have lasted several days or weeks, cord symptoms appear. They may come on rapidly, or very slowly. Often the first complaint is of numbness or tingling in the lower limbs, or of weakness or stiffness after exertion. Several attacks of temporary weakness may precede severe paralysis, and in different cases every degree is seen, from slight stiffness to complete paraplegia. When the paralysis comes on slowly the lower limbs become spastic, the knee- and ankle-jerks are exaggerated, and the plantar response is “extensor.” In severe acute cases the limbs are flaccid and the tendon reflexes are at first diminished or lost, spasticity and increased reflexes developing later. In both forms the abdominal reflexes are diminished or lost below the level of the lesion. In most cases bladder control is impaired, and in some this is the first symptom. Sensory troubles may be slight when the paralysis is severe. Numbness and tingling are common, and some objective loss can usually be detected, the temperature sense, especially for cold, and the sense of vibration being most often at fault.

In one group of cases—*Erb's syphilitic spinal paralysis*—spastic weakness develops slowly in the lower limbs, without meningeal symptoms. The bladder is usually affected, and sensory loss is slight. In this form the spinal disease appears later after infection than is usual in other forms of *meningo-myelitis*.

Pachymeningitis (diffuse gumma of the theca) as an isolated disease is usually found in the cervical region. The earliest symptom is pain in the neck, radiating down the upper limbs and between the shoulders. After a time, usually several months, weakness, wasting and loss of sensation appear in the arms. Still later, spastic paraplegia may develop from compression of the cord. When the lumbar region is attacked, the same symptoms appear in the lower limbs.

In *syphilitic caries* tenderness over the diseased bones, pain on movement of the spine, and radiating pain in the distribution of the sensory roots at the level of the lesion, are the chief symptoms. When the cord is compressed, power and sensation are diminished in the parts below.

Diagnosis.—Spinal syphilis appears in many clinical forms and often resembles other diseases. Hence it must be considered in every case of spinal disease without an obvious cause. The diagnosis may be founded on—(1) a history of syphilitic infection; (2) the presence of syphilitic lesions in other parts of the body; (3) Wassermann's reaction in the blood or pathological changes in the *cerebro-spinal fluid* (see pp. 1629, 1630); (4) signs of associated cerebral syphilis; (5) rapid improvement under treatment by specific remedies.

Prognosis.—The outlook for recovery of power is good when the meninges only are attacked, but bad when the symptoms are due to softening, hæmorrhage, atrophy of motor cells, or tract degenerations, for these are secondary changes and are not influenced by anti-syphilitic remedies. In a given case, however, it is not possible to assess accurately the amount of damage sustained by different structures, and the prognosis is always doubtful. Complete recovery occurs in about one-third of the cases of slight or moderately severe *meningo-myelitis*. The majority make a partial recovery, and are able to

walk fairly well, in spite of the weakness and stiffness which remain. In cases with a sudden onset of severe paralysis, the prognosis is very bad, and death from bedsores or bladder and kidney infection is the usual result. On the other hand, the outlook for recovery of power is better when slight troubles come on rapidly than when they develop extremely slowly, as in Erb's form of the disease, in which many years pass before the paralysis becomes severe; for in the first case the lesions are interstitial and they respond well to treatment, whereas parenchymatous degeneration of the nervous elements themselves is present in the latter, and treatment is of less avail. The prognosis in early cases is determined by the effects of anti-syphilitic treatment. In cases of long standing, the most that can be expected is that appropriate treatment will arrest the course of the disease.

Treatment of Cerebro-spinal Syphilis.—As soon as the diagnosis is made, vigorous anti-syphilitic treatment should begin. The drugs used are mercury, bismuth, the arsphenamine derivatives and potassium iodide. In the opinion of some authorities mercury still holds pride of place in the treatment of neurosyphilis, especially when administered by inunction. Treatment will vary according to the acuteness and nature of the lesion present.

In *acute syphilitic meningitis*, unless the condition is grave, it is best to begin with mercurial inunction (60 grains of the ung. hydrarg., B.P., well rubbed in) for the first week, and then to give a course of neoarsphenamine (0.6 g.), at five or six day intervals, until 6 or 8 injections have been given. Simultaneously with this, intramuscular injections of a bismuth preparation (salicylate, or iodobismuthate of quinine, each dose containing 3 grains of bismuth) are employed. Subsequently, courses of bismuth or mercury and of arsenic may be given alternately until examination of the cerebro-spinal fluid shows cessation of the active disease.

In *chronic syphilitic meningitis* treatment may be started with inunctions of mercury or injections of bismuth, followed by a course of arsenical injections. A full course of mercurial inunctions consists of sixty inunctions if signs of mercurialism do not develop. Potassium iodide in large doses may also be given. Treatment must at the same time be correlated from time to time with cerebro-spinal fluid examinations.

In *vascular syphilis* also treatment is best begun with either mercury or bismuth, and arsenic not given for the first two weeks. Iodides should also be administered.

Massage and passive movements should be carried out daily, when the limbs are weak. When sensory loss is present, careful nursing is necessary to prevent the formation of bedsores; and when control of the bladder is defective, the usual precautions must be taken for the prevention of infection of the urinary tract.

GENERAL PARALYSIS OF THE INSANE

Synonym.—Dementia Paralytica.

Ætiology.—As in tabes, the essential factor in the causation of general paralysis is previous syphilitic infection. Males are affected much more frequently than females. The onset is commonest between the ages of 30 and 50 years, from 10 to 20 years after infection. As a result of congenital

syphilis or of early innocent infection, it may appear in childhood, youth or even adult life. It has been estimated that about 5 per cent. of *syphilitics* develop general paralysis, but the incidence in those who have been well treated in the early stages is not more than 1 per cent. The incidence varies in different races, and seems to be greater with increasing civilisation. It is more apt to occur in town and city dwellers, and in those who have led a strenuous intellectual or business life.

Pathology.—The skull-cap is thickened, especially in its anterior part, its density is increased, and the diploë is obliterated. The dura mater is thickened and adherent to the skull, and may show the changes of *pachymeningitis hæmorrhagica*. The arachnoid is tough and thick, and white lines are seen between the sulci and along the vessels. The pia is thickened, its meshes are distended by pale yellow fluid, and on attempting to strip it off portions of the cortex are torn away. The amount of cerebrospinal fluid is increased. The brain looks wasted and shrunken, and its weight is abnormally low. The sulci are wide and the convolutions are narrow. The ventricles are dilated, and their ependymal lining presents a granular or a frosted appearance. On section the grey matter of the cortex is seen to be thinner than normal—decortication. On histological examination gross changes are found in the membranes, in the blood vessels, in the neuroglia, and in the true nervous elements. The earliest changes are found in the cortical vessels and membranes. Nuclear proliferation occurs in the walls of the smallest pial vessels and in the perivascular lymphatics. There is overgrowth of the endothelial cells in the capillary walls, and around them lie peculiar cells—the so-called plasma cells. Small lymphocyte-like cells, mast cells, and many others of doubtful nature appear in and around the perivascular channels, and by blocking them interfere with the nutrition of the cortex. The larger vessels also show proliferation of the endothelium, degeneration of the muscular coat and perivascular infiltration. The spirochætes are readily demonstrated in the brain substance.

The fibres of the neuroglia proliferate, its cells multiply, and some assume an abnormal size or shape. These changes in the interstitial tissues are followed by degeneration of the cortical cells and atrophy of their processes. The changes are most marked in the cortical cells and association fibres of the anterior part of the cerebrum, but similar degenerations are found in the basal ganglia, the cerebellum, the brain stem and the spinal cord.

Symptoms.—The disease is characterised by progressive deterioration of the mental and physical powers.

MENTAL SYMPTOMS.—The most recent acquisitions are usually lost first. Hence the earliest sign of mental failure will differ according to the intellectual and emotional make-up of the individual attacked. Memory, judgment and reasoning are impaired from the first, the æsthetic, moral and intellectual attributes alter early, and changes occur in the domains of conduct and emotion which astonish or distress the patient's friends. To one who sees the patient for the first time the defects may not be apparent, but those who know him will speak of the *changes* in his intellectual capacity, character or behaviour. Cheerfulness has given place to depression or irritability. the quiet and unassuming man has become passionate and boastful, the good father has turned against his family, promises are no longer kept, a good

business is neglected, money is spent unwisely, high artistic skill is lost, the moral code is transgressed, and so on. There is no end to the variety of the early symptoms, but in each case they represent a change for the worse.

In the classical form of the disease, elation and expansive delusions concerning health, wealth, social position or physical and athletic powers are prominent, but in a larger number of cases the patients are depressed in the early stages, and the delusions when they appear may be melancholic or hypochondriacal. Unlike the paranoiac, who may refuse to disclose his delusions or who may reason skilfully from his false premises, the parietic reveals his delusions readily, and can be made to betray their falseness by his own words. To the direct question regarding his occupation the Emperor, the possessor of untold wealth, the world's greatest general, will reply unconcernedly that he is a boot-black, or the champion athlete of the universe will give answers showing a complete absence of familiarity with any branch of sport.

In the course of time dementia increases, memory is abolished, delusions are forgotten, emotion disappears, and in a year or two the patient is unable to move from his bed where he lies speechless, paralysed and incontinent. The various concomitants of the delusions are described in the following paragraphs.

The most constant and most characteristic signs are changes in the pupils, tremors of the face, tongue and hands, and disorders of speech. Changes in the pupils occur early, indeed they are often present as signs of past syphilis before symptoms of general paralysis appear. Inequality in size, irregularity in outline, and the complete or incomplete Argyll Robertson phenomenon, are very common pupillary signs. Primary optic atrophy is frequent, but except in tabetic cases it is rarely complete. Paralysis of the external ocular muscles sometimes occurs as a result of associated tabes or cerebral syphilis. Tremor is often an early sign. In the face and hands, though often present when the parts are at rest, it is best seen in speaking or when movements, such as showing the teeth and holding the arms outstretched, are carried out to order. The typical tongue tremor is a backward and forward "trombone" movement of the organ, when the attempt is made to protrude it. Speech is often affected early. At first it is merely hesitant. Later it becomes blurred, syllables are omitted, interpolated or slurred, and the voice becomes feeble and lacks intonation. As the memory fails, confusion arises in the construction of long sentences, proper names are forgotten, the choice of adjectives and verbs becomes more and more limited, and the vocabulary diminishes until only interjections are left. Written language suffers in the same way, and may show defects of execution and of ideation before spoken speech is noticeably altered. At the onset voluntary power in the muscles is usually maintained, but undue fatigue after moderate exertion is a common early symptom. As the disease progresses, weakness appears in the lower limbs and soon affects all the muscles. Some of the signs of injury to the pyramidal tracts, such as increased tendon reflexes, diminution or loss of the skin reflexes, and Babinski's plantar response, are found sooner or later in almost every case. In a small number the tendon reflexes are abolished and other signs of tabes, such as sensory disturbances, are present. Retention of urine or incontinence sometimes

occur in the early stages. Towards the end, control of the bladder and rectum is always lost.

Epileptiform seizures of various kinds are common. They may be the first obtrusive symptom, and may occur at any time in the course of the disease. The attacks may have all the aspects of idiopathic epilepsy, or they may be local and of the nature of Jacksonian fits. Attacks resembling *petit mal* have also been observed. In the so-called "congestive" apoplectiform attacks, paralysis of one limb or of one side of the body comes on suddenly with or without convulsions and passes off in a few days or weeks. The patient may become comatose and breathe stertorously, or he may be merely somnolent or confused.

Insomnia is frequent in the prodromal period, but in the early stages sleep is often excessive. Later, sleeplessness and motor restlessness are often troublesome symptoms.

CLINICAL TYPES.—*Exalted or expansive form.*—This form includes the cases in which elation, euphoria and grandiose ideas are prominent.

Demented forms.—The patients often seek advice of their own accord, complaining of diminished mental and physical power or failing memory. Mental deterioration runs its course without marked depression or exaltation.

Depressed form.—This common form is characterised by melancholic and hypochondriacal delusions. Some have delusions of persecution. Very often the patient exaggerates his afflictions to a degree not seen in other forms of insanity, melancholic megalomania. Remissions are common.

Maniacal form.—The features of this form are attacks of acute maniacal excitement, which may resemble acute delirious mania. Remissions are common, and apparent complete recovery may be made; but the attacks recur, each one leaving the patient more demented.

Many other varieties have been described, the stuporose, the convulsive, the tabo-paretic, and so on. The characters of these types are sufficiently indicated by the names.

Diagnosis.—It is upon the detection of character alterations in the patient that the early diagnosis of general paralysis commonly depends. As has been said, these alterations vary from subject to subject, but constant components are impairment of judgment, defects of memory, and signs of impaired emotional control. Emotional tone may be one of depression, or one of exaltation, and while the latter is the more striking it is probable that the former is the more frequently encountered. It is the patient's family and his fellow-workers who first notice the insidiously developing change, and their evidence may be essential to early diagnosis. It is, however, important to remember that an altered emotional tone is invariably accompanied by some indications of mental deterioration, and this latter component serves in the differentiation of the psychoneuroses from general paralysis. Commonly the patient himself has little or no insight into his altered state, and may express his subjective sense of well-being and of intellectual acuity in glowing terms that arouse a suspicion of the true state of affairs in the trained observer.

Accompanying these essential alterations are a number of somatic physical signs, such as a slurring articulation, some tremor of the lips and tongue, perhaps some tremor of the hands. Pupillary anomalies are almost constant,

but fall short of those seen in *tabes dorsalis*. Thus, myosis is uncommon, but irregularity of outline, inequality and sluggishness of the light reaction are usual. There may or may not be changes in the tendon reflexes and plantar responses, but diagnosis in the initial stages of the malady has often to be made in the absence of many of these somatic signs. In doubtful cases confirmation must be sought, and can be obtained from the examination of the cerebrospinal fluid and from the Wassermann reaction (see pp. 1629, 1630).

Course and Prognosis.—After an insidious onset, the disease progresses steadily and usually ends fatally in about 3 years. Acute forms may run their course in a few weeks. When convulsions are frequent, death usually results in about 6 months. When periods of extreme restlessness and excitement alternate with depression, one year is the average duration. In simple demented and depressed forms, the duration is usually about 3 years. The course is most prolonged in those who have attacks of wild excitement or mania, as remissions are very common in this form. During the remissions, patients may be able to return to work, and 10 years may elapse between the first attack and the fatal termination. The course is often prolonged in women, in congenital cases, and in cases of tabo-paresis.

Treatment.—As some cases of cerebral syphilis simulate general paralysis, in every instance where the latter disease is suspected rigorous anti-syphilitic treatment should be tried in the hope that the patient is suffering from the more curable condition.

Malaria therapy in general paralysis.—Encouraging results have been obtained by infecting paralytics with benign tertian malaria. Blood is obtained from a patient suffering from malaria (not necessarily during a rigor), and is injected intramuscularly, or infected mosquitoes may be applied to the skin in a wide-mouthed jar, the orifice of which is covered with muslin. The incubation period may be as long as a fortnight or more after subcutaneous injection, but failure to infect with 5 c.c. of blood is rare. If the blood has to be transported it should be received in a sterile test-tube containing glass beads; after defibrination it is transferred to another sterile tube and packed in ice, when it will remain active for 6 hours or more. The recipient is allowed to have six, ten, or more rigors, the number depending on his general condition during the treatment; the infection can be cut short at any moment with quinine; relapses rarely occur in this experimental malaria. In favourable cases a remission occurs, improvement continuing for several months; the end result of the treatment cannot be assessed until 6 months or more have elapsed. When the patient has recovered from the debilitating effects of the malarial infection, anti-syphilitic treatment should be initiated. For this purpose tryparsamide has been successfully employed, after an initial injection of 1 g., a course of weekly injections of 2 g. may be carried out—15 injections being given. This drug has been known to cause optic atrophy and therefore the visual function must be carefully watched during its administration.

Malarial therapy is not without risk and should be employed only by those experienced in its use, and under institutional conditions where adequate nursing is obtainable. Malarial therapy owes such efficacy as it possesses to the severe and recurrent pyrexia it produces, which is very destructive of spirochaetes in the brain. Recently attempts have been made to bring

about a more readily regulated hyper-pyrexia by mechanical means, such as prolonged retention in electrically heated cabinets. This procedure, not less than malarial therapy, calls for considerable experience and the requisite apparatus, and even on these terms it cannot be said to be free from danger.

TABES DORSALIS

Synonym.—Locomotor Ataxia.

Ætiology.—Tabes is more frequent in men than in women (10 to 1), and begins most often between the ages of 30 and 45. The essential factor in its causation is previous syphilitic infection. The interval between infection and the onset of symptoms varies from 2 to 20 years; commonly from 5 to 10 years. As a result of congenital syphilis, or of infection in infancy, it sometimes begins in childhood, youth, or early adult life—infantile or juvenile tabes. Occasionally husband and wife are both affected—conjugal tabes.

Pathology.—Despite what has been said earlier (p. 1630) of the essential lesion in syphilis of the nervous system, it has to be admitted that all we know with certainty of the lesion in tabes is that there is degeneration of exogenous fibres in the posterior columns of the cord. At various times it has been suggested that the primary lesion is a form of local meningitis of the posterior roots proximal to the posterior root ganglion. Recently, Richter has described a peculiar type of granulation tissue in this situation, and he believes that this “strangles” the entering nerve fibres, and thus leads to their secondary degeneration. But Stern has shown that this tissue is present in the normal subject, and therefore the hypothesis of a primary local meningeal lesion remains unsubstantiated. It is known that toxins may ascend afferent nerve fibres from a peripheral focus of infection, and that degeneration of exogenous nerve fibres *within* the cord may result, the peripheral portions of the nerve fibre showing no lesion. It has been suggested that the toxins of syphilis may thus ascend from a peripheral focus, and that the essential and primary tabetic cord lesion is thus a degeneration of nerve fibres within the cord. Some of the degenerated fibres end around cells in the grey matter soon after they enter the cord, while all the fibres with a long intraspinal course enter the posterior columns, and ascend in them to the nuclei of Goll and Burdach in the medulla. As a secondary change the neuroglia around the degenerated fibres increases in amount and density. Hence the characteristic feature in sections of the cord in tabes is sclerosis of the posterior columns. The sclerosis usually appears earliest in the postero-lateral columns of the lower lumbar and upper sacral regions. In the dorsal and cervical cord it is confined at first to the postero-internal columns, which contain the degenerated fibres from the lumbar and sacral regions, but in advanced cases when the dorsal and cervical sensory roots are also affected the posterior columns are sclerosed throughout.

In advanced cases the endogenous tracts of the posterior columns show degeneration, and in some the afferent tracts in the lateral columns are also affected. In sections stained by the Weigert-Pal method the diseased areas are paler than the rest of the white matter. By the Marchi method parts containing recently degenerated fibres show numerous black dots, which represent fatty material in the degenerating myelin sheaths.

In cases of long standing, atrophy of the entire sensory root with degeneration in the peripheral parts of the sensory nerves, and atrophy of cells in the ganglia are frequent findings. The ocular palsies of tabes are probably mainly due to gummatous meningitis, but it is probable that in the case of the third nerve there may be a degeneration of the nerve cells in the nucleus. Tabetic optic atrophy is also the result of a combined interstitial gummatous inflammation and primary degeneration of optic nerve fibres.

Symptoms.—The inadequacy of current descriptions of the clinical manifestations of tabes is shown by failure on the part of those who depend upon them for guidance to diagnose the disease before it has reached an advanced stage. So long as tabes is described as a disease characterised by severe lightning pains, absent knee-jerks and Argyll Robertson pupils; so long as the diagnosis is withheld until these symptoms are found together; so long as patients without ataxy are stated to be in the *early* or *preataxic* state—just so long will valuable years be wasted, as they are at present, before patients receive treatment at a time when it might be expected to arrest the course of the disease. It is true that these important symptoms appear ultimately in a very large proportion of the cases, and they are often present when the patient is seen for the first time. It is equally true, however, that many of these patients have complained of symptoms which, if they had been appreciated by the physician, would have betrayed the disease many years before, and that throughout these years they have presented physical signs which, although the knee-jerks and pupillary reactions were still present, would have made the diagnosis of tabes certain. In the following paragraphs stress will be laid on the signs that appear early and allow the diagnosis to be made at the onset of the disease. Chief amongst these early symptoms are disturbances pointing to interference with the functions of the posterior nerve roots.

SENSORY DISTURBANCES.—*Subjective.*—Following a general law the first manifestations of altered function are subjective—the patient complains of sensory troubles before any changes can be discovered by objective examination. The most important of these subjective troubles in tabes are the so-called lightning pains. These pains merit the closest attention. They are rarely absent, they often precede other symptoms by 5 or 10 or more years, and most important of all they possess peculiar features which render them pathognomonic of tabes and allow the diagnosis to be made in a syphilitic on their presence alone. Although they are rarely absent careful interrogation may be needed to disclose them. To the question, "Have you had any pains?" the patient may answer "No." If then he is asked if he has rheumatism, he will often answer "Yes," and proceed to give an account of characteristic tabetic pains of several years' duration. In other cases the patient mentions his pains, but their significance escapes notice because it is thought that they are too slight for tabetic pains. It must be made clear at once therefore that the peculiarity of the pains in tabes does not lie in their severity, for they vary from a trifling sensation of discomfort to almost intolerable agony, but in their distribution, in their direction of propagation and especially in their arrangement in time.

As a rule, they come on in attacks, in which single momentary pains are repeated at intervals of a few seconds or minutes for several hours, the whole bout lasting several days or weeks. Between the attacks there may

be long intervals of complete freedom from pain. The pains are felt most often in the lower limbs, but any part may be affected. They may be referred to the skin, to the muscles or to the bones. They are very common in the bony prominences around the knee and on the foot. The direction of radiation varies. In some the pain seems to shoot up or down a limb, but in a larger number it seems to strike the limb vertically as if a sharp object were piercing it from without. Some patients experience both kinds of pains. The onset of each pain is always sudden. If it is severe the patient may cry out, and if it overtakes him whilst walking he is forced to stop and he may fall. The duration of each pain is usually momentary, but sometimes it lasts a second or two and fades away gradually. During a given bout the pains usually recur in the same place each time for several hours on end, and then appear in another part, say on the following day. In a few cases, however, they confine themselves to two or three points, now appearing in one and now in the other. In a still smaller number the site varies from moment to moment, so that the patient never knows where the next one will strike him. In one group the pains are repeated very rapidly in one place for a few seconds, and then after an interval in another, so that the timing recalls the sound of a machine-gun firing short bursts—tap, tap, tap, pause, tap, tap, tap, pause, and so on with a longer interval now and then during which the gun is trained on a new objective.

After a bout the skin is often tender, and ecchymoses may appear over parts in which the pains were felt, though this is exceptional. Cold, changes in the weather, anxiety and especially over-exertion make the pains worse. They are often more severe for a day or two after treatment by intravenous or intrathecal injection of specific remedies. Other pains with characters which are not peculiar to tabes are common. They are described as aching, burning or gnawing pains. Like the lightning pains, they alter with changes in the weather and are usually attributed to rheumatism. Other common subjective sensory symptoms are "pins and needles" in the extremities, a feeling of walking on a soft substance, and of constriction around the trunk or limbs. More important than these, because it often appears very early, is hyperæsthesia of the trunk, especially in its lower part. Light touches or applications of water at certain temperatures are almost unbearable.

Objective sensory disturbances.—Signs of damage to the posterior nerve roots appear in the earliest stages of the disease, and are demonstrable in many cases long before the classical signs appear. The detection of this early sensory loss is of great importance, for its distribution is pathognomonic. The parts in which sensation is first impaired are—(1) a band on the chest and along the inner border of the arms; (2) the feet; (3) around the anus; (4) on the nose.

As the disease advances, sensory loss extends upwards from the feet, downwards from the chest, and outwards from the nose and anus in concentric circles. Ultimately these areas coalesce, and in the later stages sensation is diminished all over the body. All forms of skin sensation are not equally affected. Sometimes the defect is first discovered on testing with light tactile stimuli, but more often pain and temperature are first impaired.

The senses of deep pain and of position and passive movement, as well as the vibration sense, are often diminished in the legs in the early stages. In advanced cases these defects are present in all the limbs.

In cervical tabes sensory disturbances occur first, and are most severe in the arms.

In severe cases sensation of all kinds may be almost completely abolished. No cutaneous stimuli are felt and the deep structures are insensitive to pain. To this is added loss of the sense of position, not only in the limbs but also in the trunk, so that the patient is unaware of their position when his eyes are closed. If he sits up with the arms outstretched, on closing his eyes the arms "wander," the fingers execute slow "piano-playing" movements, and the body sways. In extreme cases the patient falls on his side as soon as the eyes are closed.

MUSCULAR HYPOTONIA AND THE TENDON REFLEXES.—Loss of muscle tone occurs in lesions of various parts of the nervous system, and is not necessarily accompanied by changes in the reflexes in the limbs, but when it results from interruption of the spinal reflex arc the two signs are found together. Hence in tabes, where the afferent limb of the reflex arc is the first structure affected, hypotonia and diminution of the tendon reflexes are characteristic signs. The decrease in the tone of the muscles is often well marked when lightning pains are the only symptom of tabes, and loss of skin sensation the only other sign. It is shown by flaccidity of the muscles, and by an abnormal range of active and passive movement of the limbs.

The leg can often be raised to an angle of 100° from the horizontal, with the knee extended, whereas a normal person cannot raise it more than 60° , and excessive range of dorsiflexion of the foot is often a striking sign. In extreme cases the legs can be made to encircle the neck, the body can be flexed so that the head touches the bed between the knees, and the patient is able to imitate the tricks of the "double-jointed" man.

The knee-jerks are very often absent when the patient is first examined, and in the later stages they are almost always lost, but compared with the signs already mentioned this one is of late onset, and may be missing even in the ataxic stage. The position formerly held by the knee-jerks in the symptomatology of tabes should be given to the tendo Achillis (ankle) jerks. Loss of the ankle-jerks is indeed an early sign in tabes, for it often precedes loss of the knee-jerks by many years. The tendon reflexes in the upper limbs are lost early in cervical tabes, and are frequently absent in cases of the ordinary type.

The skin reflexes are often exaggerated to a degree rarely met with in other diseases. This is best seen on the abdomen, and is usually associated with hyperæsthesia to touch and temperature. Later, when the tactile sense is lost, the skin reflexes are often diminished. The plantar reflex is usually normal. It is sometimes absent when sensory loss on the soles is severe, and in cases where sclerosis of the pyramidal tracts exists as a complication of tabes the response is "extensor."

ATAXIA.—The fibres conveying those afferent impressions which are essential for the proper execution of voluntary movements, are more resistant than those with other functions, and inco-ordination, though extremely characteristic of tabes, is usually a late symptom, or it may be absent throughout the whole course of the disease. Its onset is marked by unsteadiness in walking and difficulty in maintaining the balance of the body. These troubles are first noticed when co-ordinated movements are performed without the aid of vision. As the defect increases unsteadiness appears even

with visual guidance. To maintain their balance the patients walk on a wide base with the eyes directed to the ground. At a later stage some raise the feet too high, throw them too far forward and bring them down forcibly, the whole sole striking the ground at once—stamping gait. Others reel from side to side like drunken men. Still later the support of one or two walking-sticks is required, and ultimately walking becomes impossible. The inco-ordination is not only present in walking, but can be seen in all voluntary movements, *e.g.* in the heel to knee test. The same defects occur in the upper limbs. At first there is merely clumsiness in performing fine movements such as picking up small objects and in adjusting the dress. In the end the ataxia may become so great that the patient is unable to feed himself.

By appropriate tests inco-ordination can usually be disclosed before the patient has noticed it. Some of the tests are: standing with the heels and toes together, standing on one foot, walking backwards, rising quickly from a stooping position and turning quickly in walking. In each instance the unsteadiness is greatest when the eyes are closed and when the feet are bare.

SPRINTER TROUBLES.—These are the result of the lowering of pain sensibility in the bladder which is the afferent element in the reflex of micturition. An increased distension of the bladder becomes essential before the act can be started, and this fails before the bladder is completely emptied, and residual urine is present in slowly increasing quantity. Though this causes little or no inconvenience to the patient it often leads to cystitis and renal complications. Difficulty in starting micturition and nocturnal incontinence are the common complaints. Complete retention and paralytic incontinence are rare, and when retention occurs it has in our experience been due almost invariably to enlargement of the prostate, the removal of which has been well borne and has given complete relief. Sexual desire and power are usually lost early in the course of the disease.

OCULAR SYMPTOMS.—Changes in the reaction of the pupils and in their size and form are very frequent and are of great importance for diagnosis. The chief of these is the Argyll Robertson phenomenon, in which the pupil contracts on accommodation but not when exposed to light. This sign appears in both eyes in 70 per cent. of cases, and is one of the earliest to appear. It is sometimes found in one eye with a normal or diminished reflex in the other. It may be present in an incomplete form, the contraction to light being slight and sluggish when the reaction to accommodation is brisk, or the pupils may contract when first exposed to the light only to dilate again. Occasionally the pupils are fixed and do not react to either stimulus. In rare cases the reaction to accommodation is lost while the light reflex persists.

The size of the pupils varies greatly in different cases. Most often they are small, but pupils of moderate size are common, and sometimes they are widely dilated, though this is very exceptional and usually associated with optic atrophy. It is not unusual to see pupils which, when contracted on accommodation, are no larger than the head of a pin, but the "pin-point" pupils are extremely rare. Inequality of the pupils or irregularity in their outline is present in most cases. It is said that the pupils are sometimes normal in every respect even in the advanced stages of the disease. On the other hand, in old tabetics in whom the disease has been present for

very many years the pupils may be found wholly inactive both to light and on convergence.

External ocular muscles.—In the early stages transient palsies of the muscles of the eyeball often cause ptosis, diplopia or squint, lasting a few days. Permanent paralysis may come on at any time, but is most frequent in the later stages. A persistent drooping of the eyelids—tabetic ptosis—is a common sign. This is attributable to a lesion of sympathetic fibres. The patient tries to overcome the defect by contracting the frontalis muscles, and the wrinkling of the forehead with slight drooping of the lids gives the patient an expression—the tabetic facies—by which the disease may be recognised at a glance.

Optic atrophy.—Defective vision from atrophy of the optic nerve is often the symptom for which the patient first seeks relief. It occurs in about one case in ten, and almost without exception ends in complete blindness. The loss usually begins in the periphery of the visual field, and is often unnoticed until central vision begins to fail. Occasionally central vision fails early. At first one eye suffers more than the other, but ultimately, after a period which averages 5 years, all vision is lost in both. Patients sometimes relate that their blindness came on suddenly, or in a few hours or days. In these cases optic atrophy has been present for a long time, but the fibres subserving central vision have escaped until the last. On the other hand, vision may fail very slowly, with periods of arrest or apparent improvement, and total blindness is sometimes delayed for 10 or 15 years. The atrophy is primary, that is, it is not preceded by papilloedema. Pallor appears first on the temporal side, whence it spreads over the whole disk. The edges of the disk are sharply defined and the lamina cribrosa is visible as slightly darker spots, so that the disk stands out clear and bright, like a full moon. When optic atrophy is the first symptom, it is often impossible to detect any incoordination in the lower limbs, and ataxia may be long delayed. The knee-jerks are often brisk, but some of the early signs—lightning pains, sensory disturbances or loss of one or both ankle-jerks—are almost always present, and the Argyll Robertson pupil is a constant accompanying sign. In a number of the patients with optic atrophy the signs of general paralysis are added to those of tabes (tabo-paresis), and the course of their illness is that of the more serious disease.

OTHER CRANIAL NERVES.—The senses of smell and taste are sometimes lost. Vertigo, tinnitus and nerve deafness are common. Lightning pains are often severe in the distribution of the trigeminal nerve, and loss of sensation on the nose, especially to pain, is one of the earliest and most frequent signs. Paralysis of the vocal cords, though rarely sought for, is present in many cases.

VISCERAL CRISES.—There are two varieties of visceral crises which are associated with disturbance of the parasympathetic and with the sympathetic innervation respectively. The former, which is confined to the vagus distribution, consists of spontaneous sensory irritation and its reflex results which is never painful since the vagus contains no pain-conducting elements. The examples are the laryngeal crisis and the gastric crisis, which comprises painless vomiting. The latter belongs to the sympathetic distribution, always involves severe pain, and is made up of the painful gastric crises, and the rectal and vesical crises.

Gastric crises.—The organ most subject to crises is the stomach. Attacks of severe abdominal pain with repeated vomiting come on suddenly. They last a few days, or a week or two, and are often repeated every few weeks for long periods. Sometimes pain or vomiting alone is present. There is always complete anorexia. The patient looks very ill during the attack, but it is never fatal. They often occur before other symptoms of tabes appear, and are often mistaken for acute obstruction, and other conditions requiring urgent surgical treatment, but careful examination will rarely fail to reveal indubitable signs of tabes. If attention were paid to the ankle-jerks, and to sensory disturbances, instead of to the knee-jerks, unnecessary operations would be less frequent.

Next to the stomach, crises are most frequent in the larynx (*laryngeal crises*). In the commonest form there is spasm of the larynx, with noisy breathing, cough and dyspnoea. Sometimes the attacks resemble whooping-cough or laryngismus stridulus. They are much shorter than gastric crises, rarely lasting more than an hour. Death in an attack is extremely rare.

Attacks of extremely painful and prolonged tenesmus (rectal crises) are not uncommon. Attacks of frequent painful micturition (vesical crises) and of pain like renal colic (renal crises) are rare.

Cardiac, nasal, bronchial, intestinal and other crises have been described.

VASOMOTOR AND TROPHIC DISTURBANCES.—The most important of these are changes in the joints and perforating ulcers. Rarer forms are local sweating, loss of hair, nails or teeth, attacks of herpes, hæmorrhages into the skin, necrosis, rarefaction and spontaneous fracture of bones, excessive callus formation and spontaneous rupture of tendons.

Charcot's joint disease.—Arthropathies may develop at any stage of the disease. Occasionally the patient seeks advice for the first time with this complaint. The first sign is usually rapid swelling in and around a joint, with effusion and œdema. The effusion, in slight cases, subsides slowly and the joint recovers, but more often the enlargement is followed by destruction of the cartilages, wasting of the ends of the bones, peri-articular new-bone formation and destruction of the ligaments. The joint becomes disorganised, the range of movement is increased, and crepitations of startling coarseness are heard and felt when the part is handled. The characteristic feature is the complete absence of pain. Dislocations occur readily, especially at the hip. The diseased joint sometimes becomes infected. This is commonest in the foot. The joints most often attacked are, in order of frequency: knee, hip, shoulder, elbow, ankle, small joints of the hands and feet, the spine.

Perforating ulcers are commonest on the sole of the foot. Patches of hard thickened skin are frequently seen on the soles of the feet. Sometimes blisters form beneath this thick epidermis, and on bursting leave an indolent sore. Once formed the ulcer is very indolent. It is usually painless.

Complications.—Tabes is frequently complicated by other syphilitic affections of the nervous system, of which the commonest and most important is general paralysis of the insane. Some tabetics develop general paralysis, and many paretics present some of the signs of tabes. Indeed, these conditions are merely different aspects of the same disease, and are named according to the predominant features. Sometimes it is difficult to decide the category of given cases, and the name *tabo-paresis* is used to describe them. At the same time it may be observed that *tabo-paresis*

runs a much slower course than uncomplicated general paralysis, and apart from any question of treatment the survival period is longer in the former than in the latter. Occasionally the pyramidal tracts degenerate and signs of spastic paraplegia are added to those of tabes. Atrophy of the anterior nerve roots with consequent wasting of the corresponding muscles is a fairly common complication. Outside the nervous system the commonest complications are aortitis, aortic regurgitation and aneurysm.

Diagnosis.—Most tabetics come under observation for the first time when one of the many symptoms of the disease begins to cause serious trouble. The obtrusive symptom may be: lightning pains, failing vision from optic atrophy, double vision from paresis of ocular muscles, attacks of vomiting, tenesmus, unsteadiness in walking, painless joint disease, impotence, troubles with micturition, or some other less common complaint. In these the diagnosis rarely causes difficulty. A history of characteristic pains, or evidence of syphilis in the past, justifies the diagnosis of tabes on the symptoms alone. In almost all of these cases, moreover, unequivocal signs will be found which make the diagnosis certain. Two signs—the Argyll Robertson pupil and absence of the ankle-jerks or knee-jerks—are of supreme importance, for although one is often lacking, the absence of both in the kind of case we are discussing is rare. To one or both of these several of the following confirmatory signs are usually added: inequality or irregularity of the pupils, diminished sensibility to pinprick of the skin on the nose, on the chest and feet, absence of pain on compressing the calf muscles, loss of vibration sense in the feet, muscular hypotonia, defective sense of position in the limbs and unsteadiness when the eyes are closed.

When the symptoms and signs are slight and few, or when suspicious signs are found during a routine examination, the diagnosis is sometimes difficult, and may require for its elucidation a careful inquiry into the history, an examination of the blood and cerebro-spinal fluid, and a meticulous investigation of the nervous system. These cases are discussed in the following paragraphs.

THE DIAGNOSIS OF EARLY TABES.—1. Since Westphal, some 60 years ago, described loss of the knee-jerks as an early sign of tabes, and established the existence of the pre-ataxic stage, the profession, apart from neurologists, has altered its views but little, and still hesitates to diagnose tabes while the knee-jerks are present. Consequently mistakes in diagnosis are common. The diagnosis can and should be made when lightning pains are the only symptom. Pains with the characters already described occur in no other disease, and their presence calls for a careful investigation for evidence of past syphilis. In this first stage of tabes the diagnosis is founded on (1) characteristic pains; (2) evidence of syphilis in the past, obtained from the history or by examination of the blood and cerebro-spinal fluid (see pp. 1629, 1630).

2. Only rarely need the diagnosis be made on these grounds alone, for in almost every patient with lightning pains careful examination will reveal confirmatory signs. The most important of these are sensory disturbances and alteration in the pupils. Hyperæsthesia to touch and temperature on the lower part of the trunk is very common, although few patients mention it until their memory is refreshed by careful interrogation. In a patient who has had syphilis and suffers from lightning pains, a clear demonstration

of sensory impairment confined to the characteristic areas makes the diagnosis still more certain. Other signs to which a high value may be given are absence of pain when the calf muscles are compressed, loss of the vibration sense in the feet, and muscular hypotonia. Irregularities in the outline of the pupils without an obvious explanation, or pupils which react well to accommodation but sluggishly to light, are very strong evidence of past syphilis and should be duly appraised. This may be called the second stage of tabes in which the diagnosis is founded on (1) evidence of syphilis; (2) tabetic pains; (3) sensory disturbances with a characteristic distribution.

3. If to these sensory disturbances there is added an Argyll Robertson pupil, or if one ankle-jerk or knee-jerk is absent or definitely diminished when compared with its fellow, the diagnosis is established beyond doubt. This may be called the stage of the fully developed disease. The diagnosis rests on—(1) evidence of syphilis; (2) lightning pains; (3) characteristic sensory signs; (4) the Argyll Robertson pupil in one or both eyes; (5) absence of one or both ankle- or knee-jerks, or a definite diminution in one of them.

Lightning pains probably indicate that the disease is active. In the absence of pains tabes would still be suggested by the combination of an Argyll Robertson pupil with an absent ankle or knee-jerk, or by the combination of one or both of these signs with characteristic sensory loss. In such cases, however, it would be impossible to say whether the patient was suffering from tabes which was likely to progress, or whether the disease had been arrested in its earliest stages.

DIFFERENTIAL DIAGNOSIS.—*Peripheral neuritis.*—The signs common to both diseases are loss of reflexes, hypotonia, inco-ordination and sensory loss. Wasting, loss of power and tenderness of the calf muscles distinguish peripheral neuritis. A complete history and examination will usually reveal the cause of the neuritis, or disclose certain signs of tabes.

Friedreich's disease.—Loss of tendon reflexes and inco-ordination occur in both diseases, but the age of the patient, the family history, the speech defects, nystagmus and the deformities of the feet and spine make the diagnosis easy. Juvenile tabes is sometimes mistaken for Friedreich's disease.

Course and Prognosis.—In most instances the disease is well established before some serious symptom brings the patient under observation. For this reason it is usually impossible to determine the sequence and duration of the signs that are found, but if the onset of lightning pains and of ataxia are taken as landmarks, an idea of the extreme variability of the course of tabes in different cases will be obtained. In many patients the disease remains stationary in the earliest stage and causes no disability. In a large number inco-ordination appears after a pre-ataxic stage of 10 or 20 years. Some become ataxic within five years of the onset of pains, a few within a year. Once ataxy appears, its rate of increase varies within wide limits. It may be so rapid that walking becomes impossible in a few weeks; it often increases very slowly, and only interferes seriously with walking after several years, and in a large number periods of increase in the ataxy alternate with long periods in which it is stationary or undergoes temporary amelioration.

The course of the other symptoms is equally variable. In general, irritative phenomena—pains and crises—tend to diminish, while the signs of destruction of sensory nerves—diminished sensation, hypotonia, etc.—

increase. Ocular palsies are frequently of short duration, and bladder and rectal symptoms are often temporary. It is impossible to foretell how any given case will progress, but there seems to be some connection between the period which has elapsed since syphilis was contracted and the rate of evolution of the disease—the longer this period the more benign the course. If the symptoms have increased slowly in the past, the future course is likely to be slow, whereas cases of rapid onset often progress rapidly. When optic atrophy occurs, blindness results almost invariably, and a proportion of these cases develop general paralysis of the insane.

The prognosis as to life is variable. Most tabetics die of intercurrent maladies or of some cardio-vascular complication, but life is constantly menaced by cystitis and ascending infection of the urinary tract. It should be remembered that in many cases of tabes, the malady undergoes arrest, and the patient may never become ataxic or grossly disabled. Such arrest may be found in persons who have at no time had any anti-syphilitic treatment. Conversely, tabetics who have been rigorously treated in this way may become progressively disabled. On the whole the prognosis as to both working capacity and life is best in those cases where the bladder can be kept free from infection.

Treatment.—This falls under three heads: treatment by anti-syphilitic remedies, general treatment, and treatment of individual symptoms.

ANTI-SYPHILITIC TREATMENT.—Mercury and bismuth are the most valuable drugs. Several courses of daily inunctions should be given at intervals, until 60 inunctions have been applied. This may be supplemented by injections of arsphenamine. Between the courses, which may be repeated at 6-monthly intervals, mercury should be taken by the mouth in a pill or mixture. Most observers agree that all forms of intraspinal therapy are useless, and many will doubt whether any form of anti-syphilitic medication can be conclusively shown to influence the course of the malady. Nevertheless, patients should be given the benefit of such doubt on this point as there is, and it is this attitude, rather than one of unreasoning optimism, that really determines the adoption of anti-syphilitic treatment in tabes dorsalis. It should be remembered that long continued and repeated treatment of this order is apt to evoke an unfortunate psychological reaction in the subject of such a chronic malady as tabes, and to produce in him a syphilophobia that is not less distressing than syphilis. In few circumstances can it be more essential to remember that one is treating a sick man and not merely a disease.

GENERAL TREATMENT.—In early cases the patient should be encouraged to continue at his work and avocations, so far as this is consistent with the avoidance of undue mental or physical stress. Strict moderation in the use of alcohol and tobacco should be enjoined. The diet should be generous, and efforts should be made to prevent the rapid loss of weight which is a feature of many cases. Strict attention to the bowels is necessary. In many tabetics the normal call to stool is not felt, and if regular efforts to open the bowels are not made, stasis develops readily. This should be treated by enemata or by glycerin suppositories. Purgatives should be used with discretion. They are of little use in stasis, and should not be given if there is any tendency to rectal incontinence, as this is always worst when the motions are soft. A change from purgatives to enemata or suppositories

will often relieve this distressing symptom. The bladder should be emptied at regular intervals, regardless of the call, which is apt to be less insistent than in normal persons. In general, rest in bed is to be deprecated. In some instances, however, where ataxy develops rapidly, it is advantageous, provided that daily treatment by massage and exercises is instituted at once.

TREATMENT OF SYMPTOMS.—Pains.—Of the many drugs that have been tried for the relief of pains the following either in single or in various combinations have been found useful: aspirin, phenacetin, phenazone, amidopyrine, cannabis indica, colchicum, ammonium chloride and sodium salicylate. After one has lost its effect another will often give some relief. Morphine is the only drug that is certain in its action, but it cannot be allowed except on isolated occasions, when for some special purpose it is essential that the patient should be free from pain for a few hours. In no disease is the morphine habit more rapidly acquired or more difficult to break. External applications rarely do any good. Chloroform on lint sometimes gives relief. Hot baths, hot applications to the limbs and blisters to the spine are worthy of trial.

The clothing should be warm, and sudden changes of temperature should be avoided. Residence in a warm country is an advantage. Attention to small details, such as the avoidance of constipation and abstinence from alcohol, often has a favourable effect.

Crises.—Gastric crises, like the pains, are very resistant to treatment. Chlorbutol in cachets containing 10 grains is often useful. It may be given twice or at most thrice in 24 hours. The effect of the drug should be watched carefully, as it sometimes produces alarming depression of the heart and respirations. When chlorbutol fails cerium oxalate and tincture of iodine should be tried. The use of morphine is not justified. Rectal crises are sometimes relieved by small doses of grey powder with opium or pulv. ipecac. et opii. The lower bowel should be emptied daily by enemata. In mild cases with morning diarrhoea an enema or a suppository should be used before the first evacuation. Thereafter the patient should try to resist the desire to defæcate, which soon passes away, and with a little training this troublesome symptom can usually be overcome. Laryngeal crises though very alarming are practically never fatal. They are usually relieved at once by an inhalation of nitrite of amyl.

Bladder disturbances.—When there is any difficulty in passing water a mixture containing 5 minims of liq. strychninæ thrice daily will be found useful. When the bladder is imperfectly emptied the use of the catheter should not be delayed. Only too often neglect of this matter leads to death from pyelo-nephritis. It is well to remember that serious infections may run a painless course. Their presence must be sought for even when pain is absent. This entails an examination of the urine from time to time for evidence of inflammation in the urinary tract. If pus-cells are present in the urine, urotropine and acid sodium phosphate should be given by the mouth. If this does not remove them, the bladder should be irrigated daily until the urine becomes normal. True incontinence of urine is often diminished by 5 minim doses of tincture of belladonna thrice daily, or by the use of the following pill: R Ergotin (Bonjean) gr. 1, Ext. Belladonn. gr. 1-4. Ft. Pil. Sig. i t.d.s.p.c.

Ataxia.—Just as a normal person by practice and effort can learn to perform feats of balance and muscular co-ordination which are impossible for one untrained, so the tabetic by concentrating his attention on his movements can be taught to make greater use of his remaining powers. The results of appropriate re-educative treatment are often astonishing. It is no uncommon thing to see patients who had been confined to bed for months able to get about freely again. Permanency of the result is often a gratifying feature.

As long as the patient is able to get about the necessary re-education can be acquired, if he is taught to pay particular attention to each movement of his limbs, and to attempt to carry it out accurately. In more severe cases, and when the patient is confined to bed, re-education should be given along the lines devised by Fraenkel. Constant supervision is necessary at first, and the treatment should begin in an institution, or under the supervision of a skilled attendant.

No remedy is of avail in checking the progress of optic atrophy.

The condition of the feet often requires attention. Corns should not be cut. Perforating ulcers should be curetted and dressed with a paste of iodine and starch. A cradle should be placed over the feet to prevent deformities, and over-extension of the knee-joint should be prevented by wearing a suitable splint.

Charcot's joints.—As soon as this condition is discovered, the patient should be put to rest, the joint immobilised, and those measures used which tend to relieve the œdema and the effusion into the joint; and if occasion demand, the joint should be aspirated. When the joint becomes dry it should be rested for a long period. For example, the patient with a Charcot's foot should use a peg stump for six months, when the condition will be found to have healed. The knee is a difficult joint to support, and the best treatment is excision of the joint, with the production of a stiff knee.

CONGENITAL SYPHILIS OF THE NERVOUS SYSTEM

Affections of the nervous system are much less frequent in congenital syphilis than in the acquired disease. Viewed broadly, the pathological changes and the clinical manifestations are the same in both. Regarding the first, meningitis, endarteritis and gummata are common to both forms; but while *central softening* from arterial disease is characteristic of acquired syphilis, *cortical cell atrophy and subsequent sclerosis* are prominent features in congenital cases. As for the symptoms, mental defects, with convulsions and spastic weakness of the limbs, are typical of congenital syphilis in contrast to the hemiplegias and monoplegias, with or without convulsions, which occur in the acquired form. It is noteworthy that the combination of obvious visceral and integumental lesions, with parenchymatous degeneration of the nervous tissue, is very common in the congenital, but not in the acquired disease.

Symptoms.—Many syphilitic infants suffer from *convulsions* during the first two years of life and in many cases these are given as the cause of death. In those who survive, fits may continue or they may begin again towards the end of childhood. The latter is more common. The fits in some

cases have all the aspects of idiopathic epilepsy, and may continue throughout life without the addition of any symptoms suggestive of local brain disease. In another group, convulsions are followed by symptoms of *hemiplegia* or of *spastic diplegia*. The same defects may appear apart from convulsions.

Mental impairment is one of the common features of the disease. Idiocy is rare. More often the defect is first noticed between the ages of 5 and 15 years. The child may merely cease to learn, and retain any acquirements he possesses, or he may lose his memory and become slowly demented.

Vision is often defective as a sequel of atrophy of the optic nerve or of choroido-retinitis, and bilateral deafness is not uncommon. Affections of the remaining cranial nerves are rare.

Juvenile general paralysis appears most often between the ages of 10 and 17 years. It has been seen as early as the eighth, and as late as the thirtieth year. In some cases it results from congenital syphilis, in others from syphilis acquired in infancy or in childhood. The physical signs are the same as in the adult form. The mental symptoms, as might be expected, differ from those in adults, when mental decay sets in before the appearance of the instincts and passions which form the content of the delusions in older patients. A boy of 12, for example, is not likely to have delusions regarding his wealth or his intellectual capacity or his sexual powers, although he may well have grandiose ideas concerning his physical strength. Optic atrophy is very common in juvenile cases, and as in adults, signs of tabes are present in many cases.

Juvenile tabes presents the same features as in adults. It is important to remember that in rare instances, tabes in an adult owes its origin to congenital syphilis or to syphilis acquired in infancy.

The diagnosis of congenital syphilis of the nervous system rarely causes any difficulty, as the patients almost invariably present some of the stigmata of their malady.

Treatment by mercury should be carried out perseveringly. The results are disappointing.

DISSEMINATED SCLEROSIS

Synonyms.—Multiple Sclerosis ; Insular Sclerosis.

Ætiology.—Disseminated sclerosis disputes with neurosyphilis and intracranial new-growths for primacy as the commonest organic nervous disease in these islands and throughout Europe. It is said to be less frequently observed in North America.

Cases have been recorded in which the disease was noticed after acute illnesses, such as scarlet fever, influenza and rheumatism ; but it is probable that these simply made more prominent a condition already present. Febrile illnesses are usually followed by increase in the symptoms, and many patients with disseminated sclerosis relate that they became much worse after an attack of influenza. In the great majority of the cases there is nothing in the family or personal history to which the disease can be attributed. In one instance, confirmed by examination after death, it attacked a mother and her child, and a few similar cases, as well as the affection of several members of a family, or of a household, have been recorded.

The onset is most frequent between the ages of 16 and 30, the sexes being affected equally. It is rare for the disease to begin after the age of 55.

The cause is still wholly unknown. Weston Hurst has recently expressed the considered view that there is no sure evidence that any of the demyelinating diseases of the nervous system are directly due to the action of a filtrable virus. The signs of inflammatory reaction in this disease are compatible with the view that it is infective in origin, but it may be added that it behaves like no known infective disease.

Pathology.—The disease has been described by Nageotte and Riche as “an affection constituted by multiple inflammatory foci, varying greatly in size and number, disseminated irregularly throughout the length of the cerebro-spinal axis. The chief features of these foci are (i) their sharp outline, (ii) their irregular and capricious shape, (iii) the fact that they do not interrupt the axis cylinders, which are only demyelinated and deformed as they traverse the focus. Hence the absence of Wallerian degeneration. The abundance of neuroglia in the foci justifies the name sclerosis which has been given to the process.”

These foci are visible on naked eye examination, the fresh ones as greyish translucent foci, the older ones as greyish or pinkish shrunken areas. Grey and white matter are both affected, the foci having some predilection for the walls of the ventricles. The foci bear no necessary relation to blood vessels.

Under the microscope the older patches are found to contain proliferated neuroglia and nerve fibres which have lost their myelin sheaths. The axis cylinders in the sclerosed areas escape destruction for a long time. For this reason secondary degenerations do not occur in the spinal tracts, and sections of the cord between lesions at different levels present normal appearances. Ganglion cells are also spared; hence wasting of the muscles supplied by the affected segments is not a feature of the disease. In recent patches, œdema is present with infiltration by small lymphocyte-like cells, plasma cells and compound granular corpuscles around the blood vessels, especially in the adventitial sheath of the veins. It is highly probable that these inflammatory changes represent the initial lesion, and that the alterations in the nerves and in the neuroglia are secondary to them.

Symptoms.—In the early stages the axis-cylinders in the diseased areas are not interrupted completely, but suffer partial and temporary impairment, which alters in intensity with the severity of vascular and other inflammatory changes in the tissues around them. Moreover, as the inflammation subsides in one patch a new one develops and produces a different set of symptoms. Hence it is not surprising that the earliest symptoms are often slight and fleeting, or that they may first appear now in one part and now in another. In spite of this, however, certain symptoms and physical signs appear with remarkable regularity* and render disseminated sclerosis, in the more advanced stages at least, one of the most distinctive and most easily recognised diseases of the nervous system.

It is remarkable that though the demyelinating lesions, which are often of considerable size, occur anywhere in the central nervous system and commonly involve the fillet, the lateral fillet, the spinothalamic paths and the peripheral neurones in their intramedullary course and the visual path, yet anything but the most transient loss of function never occurs in connection with these

systems. On the other hand, the phylogenetically newer systems—the pyramidal paths and the proprioceptive system commonly suffer permanent damage. The common transient loss of vision may be determined by an oedematous lesion of the optic nerve as it traverses the optic foramen.

MOTOR SYMPTOMS.—Weakness in the lower limbs is the symptom for which many patients first seek relief. Beginning with a feeling of heaviness or stiffness in one or both limbs, the weakness, which may be limited at first to one group of muscles, increases, in some uniformly, in a larger number with remissions or with periods of apparent recovery, until at last, after a time which varies from a few weeks to many years, it ends in severe spastic paraplegia. The physical signs are those of pyramidal lesions in general—increased tone in the muscles and exaggeration of the tendon reflexes, diminution or loss of the abdominal and cremasteric reflexes, and Babinski's plantar response. They are of extreme importance, for some or all of them may be present when the patient's complaints are still trivial, and they are found so constantly in all stages of the disease that the diagnosis of disseminated sclerosis is rarely made in their absence.

The paralysis can often be distinguished from that of other pyramidal affections by the variations in its severity from time to time, and by the occurrence of remissions or of apparent recovery, the improvement sometimes lasting for weeks or months, and, in rare cases, for many years. In most cases, moreover, examination will reveal some other sign—nystagmus, intention tremor, or pallor of the disk—which betrays the cause of the paralysis. In one large group of cases, however, the symptoms are those of a steadily increasing spastic paraplegia without remissions and without any indication, either in the physical signs or in the history, of extra-pyramidal disease. The gait may be but slightly altered, even when the tendon reflexes are greatly exaggerated and the plantar responses are "extensor." Later, it becomes spastic or spastic and ataxia. Sometimes ataxia makes walking very difficult, when the power in the limbs is only slightly impaired. In the arms there is often loss of power associated with exaggeration of the tendon reflexes. In some cases the arms are affected before the lower limbs, when astereognosis and loss of sense of position from a lesion in the course of the parietal projection produce one of the commonest of the early symptoms—the "useless arm."

TREMOR.—The characteristic tremor in the arms appears on voluntary movement only, and increases in rate and amplitude as the goal is approached. For these reasons it is called intention, volitional, or terminal tremor. It is sought for by causing the patient to touch his nose with the tip of one finger. In its minimal form the tremor appears as two or three jerky movements of the finger just as the goal is attained, or the finger reaches the nose without any abnormal movement and then oscillates, so that it slips away from the nose again or depresses it several times before coming to rest. The tremor may be noticed first in writing or in performing other delicate movements, such as threading a needle. Later, the rate and amplitude of the movements increase, and the tremor, although still greatest at the end, appears almost as soon as a voluntary movement begins. In advanced cases it prevents all useful movements, and the patient is unable to do anything for himself. The arms are affected earliest and most often, but nodding of the head is common, and any part of the body may be affected. Beside

intention tremor, other types of inco-ordination of the limbs are occasionally seen, such as those characteristic of lesions of the optic thalamus or of the mid-brain or of the cerebellum.

SENSORY SYMPTOMS.—*Subjective.*—Numbness and tingling in the extremities and alterations in the sensation of various parts are common complaints. They are often transient, and may be the only symptoms during the premonitory period. Severe pains are rare, but many patients complain of stiffness or of aching in the limbs and in the back. Occasionally intense neuralgic pain of trigeminal nerve distribution is found.

Objective.—Severe sensory loss is not common, but careful examination will often reveal areas of skin in which sensation is impaired. Occasionally the loss is severe, and may show so sharp an upper level as to suggest the presence of a spinal tumour. In many cases the sense of position and passive movements in the limbs is seriously affected, in others loss of vibration sense is the only sensory sign. An isolated loss of the last named, in the legs, is a phenomenon of diagnostic importance. Like the other signs, the sensory disturbances often show considerable variations in extent and degree at different examinations.

OCULAR SYMPTOMS.—Attacks of *double vision* are frequent, and highly characteristic of the disease. Close interrogation, avoiding the leading question if possible, will often elicit an account of these attacks when the patient has not mentioned them at first, either because he has forgotten them, or because it does not occur to him that a symptom so remote or so transient can have any bearing on his present trouble. This diplopia is of the highest importance, because it is often the sole complaint when the patient seeks advice for the first time, and because its presence, or a history thereof, is often the deciding factor in the diagnosis of early cases with spinal symptoms. Double vision in a young person should always arouse the suspicion of disseminated sclerosis, and if it is associated with signs of pyramidal tract disease, the combination makes the diagnosis almost certain.

Strabismus is uncommon. Even when the patient is seen whilst complaining of double vision it is unusual to detect any limitation in the range of the ocular movements.

Ptoxis is rare.

Nystagmus is present in more than half the cases, but not so frequently as an early sign. It is usually fine, rapid and horizontal, appearing only when the eyes are directed to the side. In some cases the eyes oscillate constantly whatever their position. Except in rare cases, there is no apparent movement of objects, even when the oscillations are of wide range.

Visual failure.—Diminution of visual acuity due to lesions in the optic nerves—*retrobulbar neuritis*—occurs sooner or later in nearly every case. It may precede all other symptoms by a period of several years. As in the case of the other symptoms, it is subject to exacerbations and periods of improvement. A young healthy person complains of rapidly increasing mistiness of vision, usually in one eye, sometimes in both or in one after the other, reaching its maximum in a few hours or days; this is often preceded or accompanied by pain about the orbit, which is increased on moving the eye. In the common unilateral case the signs are those of a lesion in one optic nerve; the pupil on the affected side is larger than its fellow; its direct reaction to light is impaired, but it contracts well consensually. Tests with

a small object, preferably coloured, reveal a central scotoma. At the onset the disk is usually normal, but in a few instances the inflammation reaches the nerve head, in which event the disk is blurred and swollen. Later the disk may be pale or normal. Rapid improvement of vision is the rule. Special tests may reveal a persistent slight loss of visual acuity, and a partial central scotoma, or, very rarely, a complete central scotoma. Subsequent acute attacks are common. In some cases the onset of visual failure is gradual. Usually the defect is slight, but it may be serious, although complete blindness never occurs. In these cases the disk is pale, especially in its temporal portion, and the field shows a central scotoma or narrowing at the periphery.

MENTAL SYMPTOMS.—Defective memory and slight impairment of intellectual power are common. Some of the patients are morose and subject to fits of depression, but the majority are surprisingly cheerful, and do not seem to suffer mentally even when their physical state is most pitiable. In many cases there is considerable loss of emotional control, and ready laughter or weeping is fairly common. More often there is merely a tendency to laugh at trivial things.

SPHINCTER DISTURBANCES.—These troubles arise from interference with the long path in the spinal cord by which volitional consent and inhibition are held upon the act of micturition. Therefore, lack of control in the form of hesitancy and precipitancy are common, and retention may occur. In rare cases, control over the rectal sphincter is lost.

OTHER SYMPTOMS.—Deafness, giddiness and tinnitus, sometimes with repeated vomiting, are common. Epileptiform convulsions are rare. In most instances the distribution of the signs will indicate that the lesions are multiple; but sometimes, although the patches are numerous, the signs are those of a single lesion, say of the internal capsule, of the midbrain or of the cerebellum.

CEREBRO-SPINAL FLUID.—The colloidal gold test may give a weak paretic curve in association with a negative Wassermann reaction. In a few cases the number of cells is increased. Otherwise the fluid is usually normal.

Diagnosis.—The combination of spastic weakness of the legs with "Charcot's triad" of symptoms—namely, intention tremor, nystagmus and scanning speech—which is so widely and so erroneously regarded as characteristic of the disease and as necessary to its recognition, is rarely seen except in the later stages of disseminated sclerosis. As this malady usually presents itself to us in its initial stages, when it may and should be diagnosed, it commonly consists in a group of signs of involvement of the pyramidal tracts: namely, increased tendon jerks, Babinski plantar responses, absent abdominal reflexes, a little weakness of dorsiflexion of one or both feet, possibly also some weakness of flexion of the proximal segments of the lower limbs, and usually a degree of impairment, or loss, of vibration sense over the malleoli. In many cases, this is all we can find, but in an otherwise healthy young adult, it is a syndrome more likely to be due to disseminated sclerosis than to any other pathological process.

Perhaps there may be confirmatory signs, such as a little nystagmus, slight intention tremor or sensory ataxy of an arm; it may be pallor of the temporal half of one or of both disks—a pathognomonic sign. If some or all of

these signs have, as it were, been arrived at after such a fluctuating course as we have seen to be so typical of most cases of disseminated sclerosis, then diagnosis can be no longer in doubt, and it is comparatively seldom that pathological examinations of blood or cerebrospinal fluid are really necessary for this end.

When, after some years, the disease is fully developed it still retains its individuality. The patient is commonly euphoric, there is frequently tremor of the head, and sometimes of the whole body, when the patient tries to stand or walk. The arms are unsteady, the legs spastic and weak—sometimes showing a tendency to pass into the condition of “paraplegia in flexion.” There is little sphincter control left, but cutaneous sensibility is commonly almost or quite intact.

At whatever stage disseminated sclerosis comes under observation, a careful inquiry into the history of the illness is important, and to elicit this requires a knowledge of the natural history of this disease as it has been outlined here.

Disseminated sclerosis has to be diagnosed from various diseases, of which we will consider the following :

Hysteria.—The serious mistake of attributing the early symptoms of this relentless disease to hysteria can be avoided by careful examination of the nervous system. Pallor of the disk, absence of the abdominal reflexes, or a distinct difference between them at corresponding points on opposite sides, unequal exaggeration of one or more of the tendon reflexes when compared with their fellows, Babinski's plantar response on one or both sides—any one of these signs alone would render a diagnosis of hysteria untenable.

Compression of the cord.—When the signs in disseminated sclerosis are purely spinal, the diagnosis from *spinal tumour* presents real difficulties. The first may be mistaken for the latter, when the paralysis increases steadily without remissions and is associated with sensory loss extending upwards to a definite level, while the reverse error may be made when the symptoms caused by a tumour are purely motor, or vary in intensity, or are associated with nystagmus.

Friedreich's ataxy.—This may be suggested by the presence of ataxy in a young patient with disseminate sclerosis. The distinction can be made at once, for in the latter disease the tendon reflexes in the lower limbs are exaggerated, whereas they are lost early in Friedreich's disease.

Course and Prognosis.—Despite the remarkable fluctuations which may mark its course, the disease ultimately disables the sufferer and is the cause of his death. Nevertheless, it is important to remember that after the initial outbreak of symptoms, some patients regain normal physical capacity, lose all abnormal physical signs, and lead a normal life for several years. Five, 10 and 15 year periods of this kind are by no means rare, and in general it may be said that the period of evolution of the disease is longer than is generally supposed. On the other hand, a few cases run a rapidly downhill course from the onset. The later in life disseminated sclerosis makes its first appearance, the more benign its course, and sufferers may be found who have reached old age without gross or total disablement. Commonly, after two or three fresh exacerbations with intervening recoveries of greater or less completeness, a slowly increasing permanent disability sets in. It is not

possible to say that those cases which run the longest and less distressing course owe this to treatment, for many untreated cases fare relatively well. But there are certain factors which do appear to influence its course unfavourably in most, though not in all, instances; thus, intercurrent illness, especially if it be febrile, injuries which disable the patient for a short period, all surgical interventions—including the therapeutic interruption of pregnancy which is designed to avert the frequently-seen exacerbations that follow the puerperium—and prolonged or recurrent physical exhaustion.

Treatment.—The behaviour of disseminated sclerosis makes the assessment of any mode of treatment extremely difficult, and a failure to appreciate the wideness of its fluctuations and the length and completeness of some of its remissions is responsible for many therapeutic claims that in the hands of those best acquainted with this malady fail to justify themselves. So far, there is no remedy which exerts any constant or certain influence upon the course of the disease.

Arsenic is the remedy which has the longest vogue, and many believe that it is of value, though the present writer has never been able to satisfy himself that this is the case, and has seen severe exacerbations interrupt the course of a series of injections of arsenical preparations.

Nevertheless, and *faute de mieux*, we may prescribe arsenic either by the mouth, as Fowler's solution, or by intramuscular injection of one of the arsphenamine derivatives. In the former case, some begin with a dose of min. 3, which is increased by one minim per dose on alternate days until min. 8 is taken three times daily. The dose is then reduced again to its original level. If this method is used, it is best to stop all arsenic for a week at the end of each complete curve of dosage. Not every patient can tolerate doses larger than min. 3 or min. 4 three times daily. This method is probably as useful as that of intramuscular injection, but considerations of expediency may dictate the use of the latter method.

A more recent suggestion is that of Brickner, who gives quinine hydrochloride in doses of three to five grains twice daily, continued over a long period. Here, again, intolerance may intervene and prevent this. The present writer has used quinine extensively in this way since its introduction by Brickner, and regards it more highly than arsenic. Other recent forms of medication include liver therapy, pyrexial therapy, protein shock therapy, and vaccine therapy. None of these has justified itself in the writer's experience, and when it is recalled that a febrile illness commonly aggravates the severity of disseminated sclerosis, it is scarcely surprising that pyrexial therapy should sometimes have the same result.

The fact that disseminated sclerosis is sometimes—though not always—adversely affected by a confinement has led to the increasing advocacy of terminating pregnancy at the third month to avert this ill effect. But this procedure is exposed to the same objection as a full-term delivery or any surgical procedure, and sometimes has the same unfortunate influence upon the course of the malady. It is therefore not a therapeutic measure that can be justified by its results. The correct procedure is to take every possible measure to maintain the health and nutrition of the pregnant woman, and to afford her at this time and after the puerperium more than the ordinary amount of rest. This is the rational, if not always the acceptable, line of treatment.

Of great importance is the right ordering of the patient's life, when practicable, and the avoidance of fatigue in the early stages of the disease.

JAMES COLLIER.

W. J. ADIE.

Revised by F. M. R. WALSHE.

ACUTE DISSEMINATED ENCEPHALOMYELITIS

Synonym.—Acute perivascular myelinoclasia (Hurst).

Ætiology.—This is unknown. The malady may follow one of the exanthemata, or may develop without any known exciting factor. A form following vaccination first attracted attention in 1922, and later cases have been recorded after measles, smallpox and varicella. It is doubtful if the meningo-encephalitis of mumps belongs to this category. Whatever be the preceding acute specific fever, or if none has preceded the attack, the symptomatology and pathological lesions are the same. It seems unlikely that a filterable virus is responsible, since no neurotropic virus is known that attacks the white matter. All such viruses so far identified have been polio-clastic, that is, they attack grey matter only.

The relation of this variety of encephalomyelitis to other demyelinating diseases, *e.g.* disseminated sclerosis and neuromyelitis optica, is uncertain. There are no grounds for assuming that the pathological process has a common cause.

In conclusion, it seems that acute disseminated encephalomyelitis may occur apart from any exanthem, and is entitled to recognition as a separate pathological process with its own clinical picture.

Pathology.—The lesions are widespread throughout the central nervous system, and consist in perivascular foci of softening involving the entire nerve fibre, axis cylinder and myelin sheath. The foci tend to coalesce. There is proliferation of the microglia cells, and some lymphocytic reaction with a secondary gliosis. The lesion of the nerve fibres is probably primary in the myelin sheath. The lumbar cord tends to be more severely hit than higher levels.

Symptoms.—This is characteristic and uniform. The onset is relatively sudden, and when an exanthem has been present, the nervous symptoms make their appearance at more or less regular times: thus from 10 to 13 days after vaccination, from 5 to 13 days after the appearance of the rash in smallpox, and at the end of the first week in measles.

In most cases the symptoms are predominantly encephalic, but myelitic forms are also seen. In the former case there is a slight rise of temperature, some unsteadiness of gait if the patient is ambulant. Drowsiness soon develops, and there is headache, stiffness of neck and back, Kernig's sign and tache cerebrale. The abdominal reflexes usually disappear and there is disturbance of sphincter control. The state of the tendon jerks is variable. The drowsiness may deepen into coma. At this stage the limbs become flaccid and the tendon jerks abolished. There may or may not be cranial nerve palsies and some oedema of the optic disks. Death may ensue from bulbar paralysis with hyperpyrexia and a terminal broncho-pneumonia. On the other hand, a dramatic recovery may ensue even when the patient

is in this grave state. With survival there is a gradual return to full consciousness.

In the spinal type unconsciousness, if it occur, is short-lived. A severe paraplegia with incontinence of urine and fæces and sensory loss develop. The paraplegia is usually flaccid with loss of tendon jerks at first. The upper limbs may escape or be less severely affected than the lower. Recovery is the rule, but this may be slow and finally incomplete.

Treatment.—There is no specific treatment and the management of the case should follow the lines applicable to any severe and acute paralysis, with special care to avoid bedsores, infection of the bladder, hypostatic pneumonia and wasting.

NEUROMYELITIS OPTICA

Synonyms.—Diffuse myelitis with optic neuritis ; Devic's Disease.

Definition.—A form of disseminated myelitis, preceded or accompanied by retrobulbar neuritis, with or without papilloedema. It is commonly acute in onset, and may end in death or in arrest with residual disabilities. Recovery is rare. Persons of all ages from adolescence onwards may be affected.

Ætiology.—Nothing whatever is known of its causation, and therefore it has been suggested that the disease is infective. None of the neurotropic viruses is known to produce the demyelination which is the characteristic lesion of the disease, nor is there any evidence that this is bacterial.

There are points of resemblance to disseminated sclerosis on the one hand, and to Schilder's disease on the other, both in the morbid anatomy and symptomatology of the disease, but there are equally significant points of difference, and whether or not the three are ætiologically related cannot be affirmed.

Pathology.—The spinal cord shows either diffuse or multiple disseminated lesions. They may be confined to a few segments of the cord, or may be found from end to end of this structure. The essential feature of the lesions is a demyelination of axis cylinders. There is also round-celled perivascular infiltration, an intense proliferation of microglial cells, and a multiplication of tiny vessels in the affected areas. The optic nerves present the same type of lesion, namely, an intense demyelination of the nerve fibres.

Symptoms.—The blindness which indicates the optic nerve lesion may precede or may follow the appearance of paraplegic symptoms. The latter develop rapidly, and may spread upwards until sensory loss and muscular weakness reach the upper thoracic level. Blindness, with some swelling of the optic disc, and central scotoma may ensue. The patient may become progressively worse and die ; or the paralysis may become stationary and then proceed to complete recovery of both power and of vision ; or the subject may be left with disability of varying severity.

The paraplegia is that characteristic of a diffuse spinal lesion in that there is sensory loss, paralysis, and loss of sphincter control.

Treatment.—No treatment has any clear influence upon the course of

events. Arsenical preparations have been employed—as for disseminated sclerosis. The management of the case is that of any paraplegia.

F. M. R. WALSHIE.

SCHILDER'S DISEASE

Synonym.—Encephalitis periaxialis.

Definition.—A malady characterised anatomically by a progressive and massive demyelination of the white centre of the cerebral hemispheres, proceeding from a single focus or from two symmetrical foci, and producing the clinical picture of progressively increasing failure of cerebral function, local at first, but advancing in terms of the functions of the contiguous regions which are next affected, by the spread of the disease from its starting-point.

Ætiology.—Nothing is known of the essential nature of the disease, nor is it certain that all cases included under this heading form a homogeneous group. Originally regarded as an inflammatory, probably an infective, disease, the increasing evidence of its familial incidence suggests that it may be primarily degenerative. It has also been suggested that those cases in which an inflammatory reaction is present may be infective, and those in which it is absent—as it may be—degenerative. Many of the reported cases have occurred in childhood, even as early as the second year. The latest case was in the fifth decade of life. The sexes are equally affected.

Pathology.—The characteristic lesion consists of: (1) A primary demyelination and, later, destruction of the axis cylinders of the central white substances of the cerebral hemispheres, which till very late spares the subcortical zone of white fibres and the radial cortical fibres, and produces a translucent jelly-like appearance of the oval centres. (2) A very early and perhaps primary overgrowth of the neuroglia, forming a feltwork, which is particularly intense round the vessels. (3) A general infiltration of the white matter of the brain with round cells, all of which are of neuroglial origin, and most of which are engaged in the removal of altered myelin or in the formation of neuroglial fibres.

The process commences most commonly as a symmetrical patch of demyelination, in either occipital white centres, less frequently in both temporal white centres or in both prefrontal white centres, and spreads directly thence until the whole of the oval white centres becomes demyelinated. The corpus callosum is involved, and the demyelination spreads downwards through the crura into the brain stem. Sometimes, especially in the central regions, the disease starts on one side, and, after playing havoc with the white centre of one hemisphere, spreads across the corpus callosum into the other. The resulting picture of a brain, normal on the surface, and on section with apparently normal cortex and intact subcortical white bands, but with the oval centre completely changed and translucent, is peculiar to this disease. Not infrequently other patches of the disease may be scattered throughout the central nervous system. This scattered distribution and the prominence of demyelination bring Schilder's disease very close to disseminated sclerosis, and it has actually been described as "disseminate sclerosis in childhood"; but the massiveness and mode of spread of the lesions, together with their distribution, with predilection for the brain and avoidance of the spinal cord,

its incidence in childhood and its entirely different symptomatology, separate Schilder's disease sharply from disseminated sclerosis. It is largely to Collier that we owe the clinical recognition of this malady.

Symptoms.—The clinical aspect is precisely that which might be expected from a progressive destruction of cerebral function, spreading by contiguity from the initial seat of the disease. In many of the cases blindness—by which is meant blindness without any change in the optic disks and with pupils reacting normally to light—has been the first symptom, and is the result of the symmetrical demyelination of the occipital white matter. As the disease spreads forwards into the temporal regions, bilateral deafness appears; and, later, bilateral ataxy and astereognosis—due to parietal involvement, bilateral spastic paralysis—the result of central involvement, and complete amentia—due to callosal and prefrontal involvement, develop.

In those cases in which the initial seat of the disease is in the temporal, central or frontal regions, the first symptom to appear is obviously determined by the location, and the order of development of symptoms will be changed, but the mode of progress is the same in all. Where the disease starts on one side only, hemianopia or hemiplegia is the first symptom, and these are followed by the train of added signs produced by the extension of the disease into other regions. Complete mindlessness and paralysis always dominate the clinical picture in the end. The disease-process within the brain sometimes causes swelling with increase of intracranial pressure, and signs of the latter may appear in the form of headache, vomiting and papilloedema. Such cases are not common, and most of them have been regarded in life as cases of intracranial tumour. Fits are by no means uncommon. Sometimes they constitute the initial manifestation of the disease, and they may occur at any time during its course, and may be local or general. Fever is usually absent, but there may be irregular pyrexia and some of the more acute cases have been pyrexial throughout. The cerebro-spinal fluid is normal in the majority of the cases, but sometimes there is an increased protein content and a small excess of lymphocytes.

Diagnosis.—The onset with cerebral blindness or with bilateral deafness, followed by signs of progressive cerebral destruction, is so rare in any other disease as at once to suggest the diagnosis of Schilder's disease, indeed no less than two-thirds of the reported cases have shown this picture. When the disease begins unilaterally, and more particularly when headache, vomiting and papilloedema are present, the distinction from intracranial tumour is difficult or even impossible, for in both diseases the local commencement and the progressive destruction occur. In Schilder's disease, however, high-grade papilloedema is not met with, and consecutive optic atrophy does not occur. It should be borne in mind that any locally commencing progressive destruction of the brain may be an example of this malady.

Course and Prognosis.—In most cases Schilder's disease is regularly progressive to a fatal termination. In some, however, periods of standstill have been noted, while in a few others marked improvement for a time has occurred, as the result of administration of mercury, arsenic and iodides. The duration has varied from 7 days to 36 months, with an average of 9 months.

Treatment.—No treatment is at present known that will influence the course of the disease.

THE PRIMARY CEREBELLAR ATROPHIES

In the present state of knowledge a satisfactory description or classification of the primary cerebellar diseases is not possible. They are extremely rare, and in the recorded cases the nature and the incidence of the pathological change varies so from case to case that in all probability what have been described as distinct "types" are in many cases no more than different stages of a single process.

Of all the primary atrophies of the cerebellum it may be said that their ætiology is unknown, but the cause probably endogenous. In some forms there is clear evidence of heredo-familial factors, but not in all. Some of them appear in early infancy, others in later life. The lesion is bilaterally symmetrical, essentially an atrophy, and in the late cases is slowly progressive.

The most useful working classification is as follows :

- (i) Atrophy of the cerebellar cortex.
- (ii) Atrophy of the central white matter of the cerebellum.
- (iii) Atrophy of the spino-cerebellar tracts.

Of these the least rare is Friedreich's ataxy, in which there is also a lesion of the posterior column fibres, but nevertheless this disease is most conveniently dealt with here.

(i) *Atrophy of the cerebellar cortex.*—This may be total, or may be confined to particular cortical regions. It may be found as a post-mortem discovery in idiots dying in infancy, or may develop slowly in middle-aged or elderly persons (Marie's delayed cortical atrophy). The last-named form is most marked on the upper anterior parts of the cerebellum. There is a characteristic loss of Purkinje cells. The clinical picture is that of a slowly developing ataxy of gait, severe disorder of articulation, and later ataxy of the upper limbs. Nystagmus is rarely present.

(ii) *Atrophy of the central white matter of the cerebellum.*—This is commonly known as olivo-ponto-cerebellar atrophy, and is characterised by a severe loss of nerve fibres in the central white matter, with a secondary proliferation of glia fibres and nuclei, the cortex being relatively intact. In addition there is degeneration of the pontine nuclei and their fibres (middle cerebellar peduncles) and of the olives. This also is a slowly progressive lesion of middle-aged and elderly persons, occasionally familial in incidence. It can only with difficulty be distinguished clinically from cortical atrophy, except when it is accompanied by Parkinsonian symptoms or by dementia, as is sometimes the case.

Scherer has found that a similar type of lesion is constantly found in the substantia nigra and corpus striatum in these cases, and when severe is responsible for the Parkinsonism sometimes observed. Similarly, focal atrophy in the cerebral cortex may be present, and may account for the dementia which may be present. Scherer suggests that all these changes have the characters of a focal, premature senile change, and points out that the lesions of Pick's focal atrophy and Huntington's chorea also belong to this category.

(iii) *Atrophy of the spino-cerebellar pathways.*—A rare form is the so-called spino-cerebellar ataxy, which may vary in its clinical expression from a con-

genital tremor, to gross ataxy of all four limbs, accompanied in some cases by optic atrophy, and ocular palsies. The lesion is a degeneration, falling most severely upon the dorsal-spino-cerebellar tract and less severely upon Gowers' tract and upon Clarke's column. It is a malady of children and adolescents.

More familiar is Friedreich's ataxy, which is described below under its own heading.

I. FRIEDREICH'S ATAXY

In addition to the slow, clumsy ataxy, Friedreich's type is characterised by the absence of the knee-jerk and other deep reflexes, and by the presence of the extensor plantar response and of contractures, especially in the form of pes cavus, and by the presence of curvature of the spine in the later stages of the disease.

Ætiology.—The first signs of the disease usually appear in early childhood and before the sixth year; but symptoms may not be evident until a few years later. In a considerable number of cases, however, the onset is delayed until the time of puberty, while in a few examples the onset may be delayed until after the age of thirty years. As a rule the age incidence is approximately the same in each child-rank of the same family; but sometimes the phenomenon of "anticipation" is well marked, the disease appearing at an earlier age in each succeeding generation as a whole, or in successive children of the same parents. The disease is said to be slightly more common in males. Isolated cases in which no heredity can be traced are not rare. Indirect heredity is the most common, for the reason that the subjects of this disease are usually afflicted in childhood and incapacitated by the time adult life is reached, and that they therefore do not procreate. Transmission occurs both through the males and through the females. Direct heredity is, however, by no means so uncommon as has been supposed, and in one family under my observation the disease had been transmitted from father to son for seven generations.

Pathology.—The spinal cord is unusually small, and apparently this smallness may be congenital, and the posterior roots tend to be small, grey and poorly myelinated. The essential change is a primary degeneration of certain neurones in the dorsal column of the spinal cord, of the pyramidal tracts and of the spino-cerebellar tracts, both dorsal and ventral. This degeneration commences first in the periphery of the axon, which slowly dies back towards the nutrient nerve cell, as the branches of an aged tree tend to die back towards the trunk.

The degeneration of the dorsal columns is usually the earliest change, and remains the most prominent feature throughout. The degeneration of the fibres of the pyramidal tract appears later. It has its origin in the ascending frontal convolutions, where atrophy and disappearance of the giant pyramidal cells have been shown.

The spino-cerebellar tracts are constantly degenerated, the direct cerebellar tract being the most seriously involved. The cells of Clarke's column, from which the direct cerebellar tract takes origin, and around which the pyramidal tracts end, degenerate and disappear, as does also the network of collaterals which surrounds these cells. Consequent upon these degenerations, and secondary to them, well-marked neuroglial proliferation or sclerosis

occurs. The cerebellum may be normal, or it may show varying degrees of atrophy of Purkinje's cells, or of any other of its cell elements, and of the tracts connected therewith.

Symptoms.—The onset is always insidious, and physical signs of abnormality usually precede any complaint on the part of the patient or his relatives. The first symptoms generally appear between the sixth and the tenth year of childhood; but if a careful examination be made of the younger members of the families upon which Friedreich's disease is incident, physical signs of the disease, especially the extensor response in the plantar reflex, the retraction of the great toe and some degree of pes cavus may often be found before the sixth year. Not infrequently the onset of symptoms does not occur until puberty, and in some families it is delayed until after the age of 30 years.

Ataxy is always the first sign to appear, and this is shown by an awkwardness of gait and a tendency to stumble and fall readily. Sometimes it is obvious from the history, that the ataxy dates from the earliest years of infancy when it is said that the child was never strong on his legs from the time of learning to walk, and that he could never run properly or join on equal terms with other children at play. As the disease progresses, the gait slowly becomes more irregular and clumsy. The patient walks with his feet upon a broad base, and staggers and reels from side to side; but, notwithstanding this, he keeps a fairly direct line of progression. He takes short steps which are unequal, and which are irregular in relation to the line of progression, and the movement of each foot as it is raised is poorly co-ordinated. There is never the undue excursion and noisy stamping of the feet which are so characteristic of the gait of tabetic patients.

In standing the body oscillates from side to side in slow and clumsy fashion, and coarse tremors of the head and trunk are constant features in advanced cases (titubation). Sometimes Romberg's sign is present; but this is never so well marked as in tabes, and it is frequently absent. The ataxy invades the upper extremities, as a rule, later than the legs. There is first clumsiness with the finer movements, and then little by little with all the movements. It closely resembles the ataxy due to gross disease of the cerebellum, and differs from that which occurs in tabes, and that irregular breaking of a movement towards the end of its accomplishment, which has been long termed "intention tremor," is frequently seen.

Very characteristic of the disease, and highly important in diagnosis, is the occurrence of irregular involuntary movements, which are often described as like those of chorea or of myoclonus. They differ entirely, however, from the movements of chorea, etc., in that they occur only when the limb or some of its segments are unsupported. In advanced cases such movements are constantly seen in the head and neck as nodding movements and tremors, and in the trunk as swaying instability, when the patient is sitting unsupported or standing. Similar ataxy and irregular movements affect the muscles of the eyes, of the face, tongue, larynx, etc., and the respiratory muscles. In the eyes they are seen as fine, regular nystagmus and as coarse, irregular jerkings, chiefly upon lateral deviation. There is no other disease in which ataxy of the facial muscles is so conspicuous for, in engaging the patient in conversation, all the facial muscles may be observed in irregular contraction. The ataxy of these muscles causes an invariable impairment

of articulation, which gradually becomes indistinct, clumsy, drawling and slurred. The syllables tend sometimes to be separated, adding a staccato element. Explosive utterance is almost constant, and from the irregularity of the respiratory movements short inspiratory whoops are not uncommon. Articulation thus closely resembles that of advanced disseminated sclerosis, the cause being identical in the two diseases, namely, interference with the cerebellar co-ordinatory mechanism of speech.

The strength of movements is at first little impaired ; but as the disease advances and the pyramidal degeneration increases, the power is gradually lost in proportion to the degree of the pyramidal degeneration, which varies greatly in different cases. The lower extremities are affected first and most, and later the arms, and in severe cases at a late stage paralysis may be almost universal.

The condition of the muscular tone depends upon the relative degree of degeneration in the posterior roots and in the pyramidal tracts respectively, the former tending to abolish and the latter to increase it. As a rule the influence of the posterior root degeneration is preponderant and, therefore, the limbs are flaccid and hypotonic, but occasionally they are somewhat rigid. Contractures are the rule, but these are confined to the lower extremities. The most constant of these produces the deformity of the feet characteristic of Friedreich's disease, and known as "pes cavus." The great toe is strongly retracted, the tarsus is pulled up, and the metatarsus is dropped and the plantar arch is increased. The outline of the inner border of the foot comes to resemble the letter Z, the tarsus, metatarsus and great toe forming the three limbs of the Z. Sensibility is but little affected ; but in most cases minute examination reveals slight relative loss to touch, pain and temperature, most marked at the periphery of the limbs and diminishing upwards. Similarly there may be slight loss of sense of position in the limbs, with diminution of osseous sensibility to the slowly vibrating tuning-fork.

The ocular movements are almost always intact apart from the already described nystagmus. In rare instances strabismus, diplopia and ptosis have been recorded. The pupils are not affected. Optic atrophy is a rare phenomenon in Friedreich's disease, yet it has been reported in quite a number of otherwise typical cases.

Mental symptoms are usually not conspicuous, but some of the patients are of poor mentality from the first, while others show a tendency to severe mental degeneration in the later stages of the disease. Emotional instability, irritability and outbursts of temper may occur.

Absence of the tendon reflexes is a most characteristic feature, and is often the first objective sign of the disease. When one considers, however, that the absence or presence of the tendon reflexes depends upon the relative degree of affection of the posterior column upon the one hand, and upon the pyramidal degeneration upon the other, it is not surprising to find in cases where there is a major degeneration of the pyramidal tracts, that the knee-jerks may persist or even be brisk into the advanced stages of the disease. The abdominal reflexes gradually disappear. The plantar reflex is invariably an extensor response. The sphincters usually escape. The cerebro-spinal fluid presents no abnormality.

Spinal curvature is very common, and may reach a severe degree. It consists of a scoliosis of the dorsal region, and often with some kyphosis, and

with a compensatory reverse lumbar curve. The cause of this deformity is probably the defect in the postural tone of the muscles, which occurs when the afferents subserving the function of postural tone, and which are contained in the spino-cerebellar tracts, are severed.

Diagnosis.—In uncomplicated cases the diagnosis is a matter of no great difficulty on account of the strikingly distinct nature of the symptoms. Friedrich's disease can hardly be mistaken for tabes, since the history of heredity, the peculiar deformity of the feet and spine, the extensor response, the speech affection and the nature of the ataxy contrast strongly with the loss of pain sensibility and of deep sensibility, the pupillary changes, the sphincter trouble, the abnormal Wassermann reactions and the abnormal cytology of the cerebro-spinal fluid in tabes. The distinction from disseminated sclerosis presents more difficulty; but in this disease the onset never occurs in childhood, there is no heredity, the deep reflexes are never lost, and the spinal deformity does not occur.

Course and Prognosis.—The course of the disease is usually progressive in slow and irregular fashion, and the prognosis is therefore in every case serious; but the average duration of the disease is over 30 years, and in some cases it seems to have no tendency to shorten life. The prognosis is worse and the course more rapid in those patients who have shown disability from the time of learning to walk. In some cases the disease appears to become arrested, as, for example, in one family which came under my observation, twelve members in three generations were affected with typical Friedrich's disease, yet none of them was incapacitated from following a normal life, and those that were deceased had all survived the age of 70 years. Intercurrent maladies, febrile illnesses and debilitating influences generally, may have a strong effect in hastening the advance of the disease, and bringing about a fatal termination. Confinement to bed from any cause whatever has a most derogatory influence upon the ataxy, and upon the capacity for walking. It is therefore of great importance that these patients shall be kept off their legs as little as is possible. Cases in which the ataxy becomes extreme, or in which paralysis from pyramidal degeneration becomes severe, necessarily become bedridden, and in this condition the patients may survive for many years. In other cases rapid increase of the symptoms of degeneration within the nervous system is followed immediately by drowsiness, asthenia and coma, and death occurs in that peculiar toxic state which is commonly the end-result of all degenerative nervous diseases.

Treatment.—No treatment is known which specifically affects the malady. General tonic treatment, and all measures which improve the general health and mental well-being, often have a surprising effect in improving the ataxy. Re-educational training of the limbs and trunk in the form of Fränkel's exercises are most beneficial. Properly designed boots to ensure the most advantageous use of the deformed feet must be provided.

2. FAMILIAL SPASTIC PARALYSIS

This malady is here described with the hereditary ataxies, since it seems to fall naturally into the group of diseases in which primary degeneration of the pyramidal tracts is a usual anatomical feature, and of which a familial and hereditary incidence is the rule. Moreover, among the hereditary

ataxies every grade of transition is seen to the type of pure familial spastic paraplegia. Whilst in the majority of the hereditary ataxies cerebellar, spinal and cerebral lesions coexist, yet there are the purely cerebellar and the purely spinal type; and the purely cerebral type, in the form of familial spastic paralysis, forms a natural end to the series.

Ætiology.—The disease is sometimes hereditary, but is more commonly familial and incident upon several children of the same parents. Sporadic cases are not very rare. The onset is gradual in early life, and usually occurs after the sixth year.

Pathology.—The pathological changes consist in a primary degeneration of the pyramidal neurones which apparently takes place in terms of the length; those supplying the lumbo-sacral region, being lower and longer, are earliest affected; those supplying the brain stem, being shortest, are the last to be affected. Degenerative changes in the neurones of the posterior columns of the spinal cord are often present, showing the transition to the pathological type of the hereditary ataxies.

Symptoms.—The clinical aspect consists in the slow development of spasticity and weakness, first and most in the legs, which gradually increases and progresses to the trunk and upper extremities, and involves the face last and least. The usual signs of pyramidal involvement are present in the loss of abdominal reflexes, increased deep reflexes and extensor type of plantar reflex. The malady is progressive, increasing to complete paralysis, and in its course contractures of the spastic muscles occur, that of the foot and leg producing some degree of pes cavus, while, above this, flexor contracture at hip and knee is met with. Optic atrophy is by no means uncommon. Mental symptoms do not occur in uncomplicated cases, neither is epilepsy observed.

Diagnosis.—This malady is most easily confused with cerebral diplegia; but the latter disease appears much earlier, so soon after birth, in fact, as defective movement in the child can be ascertained. Further, cerebral diplegia is not a progressive disease in the majority of the cases, and it is often associated with mental deficiency and recurring convulsions.

PARALYSIS AGITANS

Synonym.—Parkinson's Disease.

Definition.—A progressive disease of insidious onset and slow course, usually occurring in the second half of life, and characterised by a peculiar stiffness of the muscles, which tends to fix the body in a certain posture, which can be changed less speedily than in health, and which gives rise to a distinctive facial expression, bodily attitude and gait. The stiffness is accompanied by weakness, and often by rhythmic tremors, which have earned for this malady the name "shaking palsy."

Ætiology.—Little is known of the causal factors of this malady. It is essentially a disease of the decline of life, and though in rare instances it is met with as early as the eighteenth year, the maximum incidence is from the fiftieth to the seventieth year. Men suffer twice as frequently as women. Heredity seems to play no part in the causation; but it is remarkable that longevity in one or both parents is common.

Pathology.—No naked-eye changes are to be found other than the vascular and degenerative changes which are common in senile conditions. The facts that tremors and rigidity, almost identical with those of this disease, may be met with in tumours involving the substantia nigra of the crura cerebri—two striking cases with autopsy having been under my own care—and still more importantly, the frequent appearance of a paralysis agitans-like end-result in lethargic encephalitis, where the subthalamic region and substantia nigra are conspicuously picked out by the lesions, make it probable almost to a certainty that the locus morbi of paralysis agitans is the basal ganglia.

Symptoms.—The onset is always insidious, and the muscular rigidity is almost always the first sign to appear. This rigidity affects the face, neck and trunk to a greater extent than the limbs, and when the limbs are affected then the proximal muscles present a greater degree of rigidity than do those of the periphery. The oncoming rigidity of the facial muscles does away with the usual play of the emotional movements in facial expression, and the face assumes a fixed, anxious and mask-like expression, with absence of the usual involuntary nictitation. The voice loses its inflexions, and becomes monotonous, from rigidity of the muscles of larynx, tongue and lips; but there is no other defect of articulation. Very striking is the effect of the rigidity of the muscles of the neck, for the patient carries his head and neck in one piece with his trunk as if he were a statue, never inclining or raising it in the customary expressive manner, and if he turn round to look at anything he tends to move the whole trunk round with the head. In looking sharply to one side the eyes move before the head, whereas, under normal circumstances, the coarse adjustment of this movement is done first by the neck muscles, and the fine adjustment subsequently by the eye muscles. The stiffness of the trunk muscles gives a stooping attitude with the head inclined forwards, while that of the upper extremities causes the shoulders to be rounded, and the arms carried with the elbow semiflexed, and pressed into the sides. The gait is highly characteristic in marked cases since, on account of rigidity of muscles, it is deprived of spring and suppleness; the patient, in the characteristic attitude above described, takes small gliding steps, displacing his centre of gravity as little as possible. If, by any circumstance, such as catching the feet against an unevenness of the ground, or a push, the centre of gravity is much displaced, the patient often has a difficulty in regaining it, and in moving to recover his centre of gravity is unable quite to catch it up, and so continues the movement of necessity until he fall or come in contact with some object by which he can arrest himself and restore his balance. This phenomenon is more often seen in advanced cases, and is known as “propulsion,” “retropulsion” and “lateri-pulsion,” according as the centre of gravity is displaced and the movement occurs in a forward, backward or sideways direction. Festination is the term used for the quickening of the pace sometimes seen in this attempt to overtake the displaced centre of gravity. In the hand the rigidity is greater in the interosseal muscles, and the hand therefore tends to assume the “interosseal position” with the fingers pressed together and the thumb adducted, the metacarpophalangeal joints being flexed, and the interphalangeal joints extended. From this rigidity of the hand the writing becomes small as well as tremulous, and the patient finds it difficult to write in a straight line. Muscular weakness always accompanies the rigidity and the tremors. It is slight until the late

stages of the disease, when it may increase rapidly and render all useful movement impossible. On account of the rigidity and consequent slowness of movement, the sense of weakness which the patient experiences is much greater than the actual weakness as tested by the dynamometer. Tremor is present in the majority of cases. It usually commences in the hand and forearm, and is most conspicuous in this situation; but it may be seen in the face, tongue, jaw, neck and feet, while, in rare cases, it may be universal. The nature of the tremor is peculiar, and is highly characteristic. It is a regular rhythmical contraction of the muscles, alternating in the opposing groups with a frequency of from four to six oscillations per second with a range of from an $\frac{1}{8}$ th to $\frac{3}{4}$ ths of an inch. Its rhythmic nature, its slowness and its course range distinguish it from other varieties of tremor. In the hand the characteristic movement of the tremor is the rolling together of the opposed thumb and fingers, cigarette-rolling, bread-crumbling or drum-tapping movement. There is nearly always in addition a peculiar pronator-supinator tremor. The tremor is increased by excitement and by self-consciousness, and ceases during sleep. A highly characteristic feature of the tremor is in about one-half of the cases is that it continues during repose, and is temporarily arrested by the execution of volitional movement. In the other half of the cases, however, the tremor appears or is increased on voluntary exertion, and tends to be less during repose. There seems to be an antagonism between the tremor and the rigidity, for in cases where the rigidity is very conspicuous the tremor is little marked or absent, and conversely, when tremor is universal or is of early onset, rigidity is a less noticeable feature.

Other symptoms of the disease which are very commonly complained of are—(1) difficulty in turning over in bed, which is the obvious result of the rigidity of the trunk muscles; (2) flexion of the toes into the sole of the foot, so that they are trodden on, from spasm of the plantar muscles; (3) pain of a dull aching character in the trunk and limbs, which is presumably produced by the long-continued traction of the rigid muscles upon their attachments; (4) abnormal sensations of heat and cold; and (5) hypersensitiveness to changes of temperature—the patient cannot bear to be near a fire nor yet in a cold room. Mental symptoms are conspicuous by their absence, except in the last stages of the malady, when profound asthenia overtakes both mind and body. The constant bodily discomfort, restlessness, sensations of fatigue, which the rigidity and the tremors engender, and the consciousness of a malady which is found only too soon to resist every effort to lessen or arrest it, often result in gloomy and lasting mental depression. Objective sensibility is unimpaired. The special senses and the cranial nerves are not affected. The sphincters and the reflexes are normal. Trophic changes in the periphery of the limbs, thinning and glossiness of the skin, with fluted nails and vasomotor disturbance, are common. Bed-sore is commonly met with in the late stages of the malady.

Diagnosis.—There are three points which can be surely relied upon to render the diagnosis of paralysis agitans certain in every case, namely—(1) the aspect of the patient when he is walking, when the fixed mournful expression, the stooping attitude with round shoulders, the elbows pressed into the side, and the hands carried across the abdomen in the interosseal position, the immobility of the head and neck, and the curious gliding gait which cannot fail immediately to arrest the observer's attention; (2) the rhythmic rolling

tremor which is quite unlike any other form of tremor, and which often continues during rest; and (3) the absence of any of the usual signs of organic disease of the central nervous system. Difficulty may perhaps be experienced when the aspect is little marked, and the tremor is confined to some unusual situation, such as the face, tongue or neck; but, if the possibility of tremor in any situation being that of paralysis agitans be borne in mind, its rhythmic rolling nature will give the diagnosis. When paralysis agitans is confined to one side of the body, the appearance of the patient may superficially resemble that of hemiplegia; but in these cases the peculiar aspect of paralysis agitans is marked, and the organic signs of hemiplegia, such as the extensor response in the plantar reflex, the increase in the deep reflexes, and the absence of the abdominal reflex upon the paretic side are not present. In senile tremor the rhythmic rolling quality is absent, and the aspect is not that of paralysis agitans. In post-hemiplegic tremor the organic signs of hemiplegia are present. Toxic tremor is irregular and never rhythmical, and is (mercurial tremor excepted) a fine tremor. The intention tremor of disseminated sclerosis, cerebellar disease and lesions of the red nucleus are so peculiar, and so widely different from the tremor of paralysis agitans, as to render confusion impossible.

The one clinical condition, which may so closely resemble paralysis agitans as to be superficially indistinguishable, is a not uncommon end-result in lethargic encephalitis, where from a lesion in the basal ganglia the same weakness, rigidity and tremors appear as occur in paralysis agitans. The distinction is not difficult, for the onset of lethargic encephalitis is usually acute, and the symptoms are definite. Moreover, the paralysis agitans-like syndrome of lethargic encephalitis sometimes shows a progressive amelioration, whereas paralysis agitans tends to a progressive downward course. The following features present in post-encephalitic Parkinsonism but absent in true paralysis agitans are of value in making a differential diagnosis: (i) a fluttering tremor of the closed eyelids; (ii) tremor of the protruded tongue; (iii) defect of convergence and of accommodation; and (iv) excessive salivation.

Course and Prognosis.—Paralysis agitans often begins in one limb, usually the upper, and spreads thence to the corresponding limb of the opposite, or to the other limb of the same side. In the latter case it has approximately a hemiplegic distribution, and it may remain for years much more evident upon one side of the body. The course is slowly progressive with variable rate. In some cases the malady may remain stationary for years, and this is more often seen in middle-aged subjects, before the disease has reached an incapacitating stage. Such arrest in the early stages is not often seen in young subjects, for in the latter the disease seems to take a more continuously downhill course. Real improvement in the symptoms is never seen. A fatal issue may occur in as short a time as two years; but this is exceptional, since paralysis agitans has little tendency to shorten life. The average duration is from 10 to 15 years, and since the major incidence of the disease is in the sixth decade of life it will be seen that many of the patients are of average longevity. Death may occur from intercurrent maladies, especially from bronchitis; but more commonly, after the lapse of many years, the patient becomes bedridden from increasing weakness and rigidity, and sinks into a condition of sleepy asthenia which is soon terminated by coma. An unduly

high blood-pressure is unusual in the subjects of paralysis agitans, and it is noteworthy that they do not suffer from gross cerebral vascular lesions, such as thrombosis or hæmorrhage.

Treatment.—Paralysis agitans is one of the least tractable of maladies even as regards the relief of symptoms. Hygienic measures and tonic treatment, calculated to lessen the rapidity of the degenerative process, should be employed. Where there is much rigidity, gentle exercise, passive movements and massage are useful. Care should be taken to avoid the falls which the unstable gait is likely to engender, since these are often followed by a marked exacerbation of the symptoms. Pain is best treated with aspirin, and sleeplessness with a mixture of aspirin and small doses of barbitone (grs. 2 and 3). As might be expected, electrical stimulation of the muscles tends to aggravate the tremor, and even in the predominantly rigid cases can do no good. In the latter type of case, some subjective relief may be obtained by the administration of tincture of stramonium or of belladonna (doses of from 5 to 20 minims), or of hyoscine hydrobromide (grs. $\frac{2}{100}$ to $\frac{1}{100}$ by mouth thrice daily in chloroform water). These drugs may render movements freer, but they have no influence upon the tremor. When the patient is bedridden, great care must be taken with the skin, since the immobility of the trunk greatly increases the liability to the formation of bed-sores.

HEPATO-LENTICULAR DEGENERATION

Synonyms.—Progressive Lenticular Degeneration ; Wilson's Disease.

Definition.—A rare progressive disease of the nervous system, often familial, characterised by involuntary movements, rigidity and hypertonicity, with contractures, without signs of pyramidal disease ; and by dysarthria, dysphagia, emotionalism and progressive emaciation. Several closely related clinical forms of the disease bear distinctive names : *tetanoid chorea* (Gowers), *pseudosclerosis* (Westphal), *progressive lenticular degeneration* (Wilson), and *torsion spasm*, and *dystonia musculorum deformans* (Thomalla). Cirrhosis of the liver occurs in all forms. The Kayser-Fleischer zone of corneal pigmentation occurs in the first three forms, but has not yet been recorded in torsion spasm. The most constant nervous lesions are found in the corpus striatum.

Ætiology.—The disease often occurs in children of the same parents, but there is no evidence that it is congenital or hereditary. The age of onset has been as early as 7 years and as late as 26 years. The primary and essential lesion is in the liver ; its cause is unknown. Syphilis is not a factor.

Pathology.—A multilobular cirrhosis, with "hobnail" liver, is always found after death. There is good evidence that the cirrhosis is not slowly progressive, but is the result of a number of attacks of acute hepatitis. The hepatitis has caused death in some members of affected families before nervous symptoms appeared. The nervous lesions are purely degenerative. In Wilson's case they were almost confined to the lenticular nucleus, especially the putamen. Every degree of degeneration was seen, from discoloration and sponginess of the nucleus in rapidly fatal cases, to shrinkage and atrophy, and even to complete disintegration and excavation of the ganglion. Later observers have described lesions in many other parts of the nervous system. The lesions are often most intense in the corpus striatum, but the noxious

agent has no strictly selective action on any one anatomical group of ganglion cells, or on any limited area of the nervous system.

Symptoms.—In many cases there are no symptoms of disorder of the liver during life. In other cases an account is obtained of symptoms referable to acute hepatitis before the onset of nervous symptoms—attacks of diarrhœa and vomiting, pyrexia, jaundice, migrainous headaches, hæmatemesis and sometimes definite ascites.

The first nervous signs to appear is usually involuntary movement of the extremities, which may be of several kinds. In progressive lenticular degeneration, rhythmical tremors, increasing on voluntary movement, furnish the most common symptom. This is followed by rigidity of the face, the muscles of the neck, and later of the trunk, which rigidity increases steadily until the patient becomes helpless. The rigidity of the face and neck muscles gives rise to a peculiar expressionless appearance. Still later, extensive contractures, usually in the flexed position, in the upper and lower extremities, follow; but sometimes there is extensor contracture of the latter. During sleep the tremors cease, but the contractures do not relax. Dysarthria, of a sturring type, results from affection of the muscles of speech, and may end in complete anarthria. Progressive muscular weakness and general emaciation follow; and the patient becomes emotional, facile, docile and childish. There is no fibrillation or localised amyotrophy. The optic disks and pupillary reactions are normal. There is an absence of nystagmus, cerebellar symptoms, and impairment of sensation. The reflexes are not altered, as in the case in pyramidal disease.

Prognosis.—The disease always ends fatally in a few months or years; the average duration is about 4 years.

Treatment.—None is known to have any effect upon the course of the disease.

JAMES COLLIER.

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Revised by F. M. R. WALSHIE.

KERNICTERUS

Definition and Ætiology.—A yellow pigmentation of certain of the basal ganglia, associated clinically with motor disorders of the type known as extra-pyramidal, and found as a rare phenomenon in children who, normal at birth, develop jaundice within the first three days of life.

In neonatal jaundice the brain may be diffusely pigmented, or more rarely the pigmentation may be confined to the putamen, subthalamic and dentate nuclei, the cornu Ammonis and fascia dentata. To the latter variety of jaundice of the brain the name “Kernicterus” has been given by Schmorl. The nerve cells in the affected masses of grey matter show evidence of destruction and degeneration, while the nerve fibres are demyelinated.

Symptoms.—The child is healthy at birth, but within a few days develops intense jaundice, usually the form known as icterus gravis neonatorum, though kernicterus has been found in association with septic jaundice. The onset of jaundice is followed within 24 hours by tonic and clonic movements, muscular rigidity and opisthotonos, alternating with periods of flaccidity.

If the child survive, within a few weeks involuntary movements of choreo-athetoid form develop. Emotional instability and mental retardation appear as the child grows older.

Prognosis.—The cases so far identified and on record are too few to allow of any generalisation as to the expectation of life of the subjects of this malady, but in two cases recently reported by Greenfield, one patient died at the age of three months, the second at nine years.

Diagnosis.—Athetosis and comparable forms of involuntary movement are not rarely seen in children, and are in the majority of instances not associated with kernicterus. Yet when a case of such motor disorder is seen in a child in respect of whom there is a history of neonatal jaundice, the possibility of this disease should be borne in mind. Again, the development of marked symptoms of organic nervous disease immediately after the appearance of severe jaundice in a newly-born infant should lead to a consideration of this condition as the probable pathological basis.

Treatment.—There is no evidence that the condition is susceptible to any mode of treatment.

F. M. R. WALSH.

EPILEPSY

Synonym.—Idiopathic Epilepsy.

Definition.—A condition characterised by suddenly occurring disturbances of cerebral function, prone to occur over long periods of time or even through life. Of the intimate nature of the disturbance all we know with certainty is that a series of characteristic changes in the normal electrical activity of the cortical nerve cells accompanies it. The clinical forms of the disturbance suggest that this involves both loss and release of function in various regions of the brain.

Ætiology and Pathology.—Few problems in medicine have been more obscure, or more provocative of speculation and of controversy than the nature of epilepsy. It has been widely assumed in the past that there is an idiopathic epilepsy, a definite and often heritable disease, with an individuality and natural history of its own, and one of which the characteristic and often the sole expression is the fit.

On the other hand, it has also long been realised that fits clinically indistinguishable from those of idiopathic epilepsy may occur in the course of many and diverse affections of the brain, as, for example, in certain inflammations, degenerations, intoxications and also in association with new growths. Such fits have been placed in a category of symptomatic epilepsy, the relation of which to the idiopathic variety never admitted of clear formulation.

These facts have led some writers to maintain that epilepsy is no more than a symptom, one expression of many and diverse neuro-pathological processes, and because of this manifold causation they prefer to speak of "the epilepsies." Against this view it may be urged that not one of these pathological processes is necessarily, or even commonly, productive of fits and may occur without their development. Such processes cannot therefore be wholly or even primarily responsible for the fits that may, upon occasion,

accompany them. As the late Kinnier Wilson, a strong supporter of the purely symptomatic nature of epilepsy pointed out, "The fit, the fundamental symptom of epilepsy, has no histopathology." In this statement we see that the fit has become a symptom of a symptom. Again, he stated, "The number of epilepsies for which no cause other than that of inherent irritability can be discovered is being steadily reduced." This pronouncement speaks in favour of a unity rather than of a diversity in epilepsy.

It is probable, therefore, and the evidence about to be stated supports the suggestion, that the fit, and the tendency to its recurrence that we call epilepsy, owe their production to some abnormal quality of structure or of function in the brain, and that the many pathological processes that may give rise to fits—as incidental but not essential phenomena—are no more than secondary and contributory factors capable of fit production in the susceptible brain alone. Yet even if there be but a single essential factor common to all fits, it still remains practically useful, if not necessary, to speak of an idiopathic and a symptomatic epilepsy. For purposes of diagnosis, prognosis and treatment the distinction is clearly important. We must separate the two, for example, when we proceed to generalise as to the heritable qualities of epilepsy, and the treatment of a case of uræmia with fits is clearly widely different from that of a case of idiopathic epilepsy. This distinction still allows us to suppose that in idiopathic epilepsy the "fit threshold" of brain, as Lennox expresses it, is so low that the ordinary activities of life and very minor fluctuations in bodily health are sufficient to fire off a fit; while in what we call symptomatic epilepsy, the threshold is higher and is only crossed when some gross superadded disturbance of the brain—of wholly independent origin—is superadded.

The general point of view thus summarised finds a considerable measure of support in the recent researches of Lennox, Gibbs and others upon this subject. They have employed an instrument known as the electro-encephalograph which promises to do for the brain what the string galvanometer of Einthoven has done for the heart. It was originally observed by Berger (1929) that regular oscillations of potentials of a rhythm of 10 per second could be detected if electrodes applied to the scalp were led off to a recording instrument. This work has since been confirmed and very considerably amplified by Adrian in this country and many other observers. It is known that these oscillations, or, as they are called, brain potentials, are due to the electrical activity of cortical nerve cells. The various rhythms, and their sources in the cortex, that come within the normal range of variations in the healthy subject have been studied, and it has subsequently been found that in the epileptic subject these rhythms undergo characteristic variations during the course of the fit. These observations may be summarised as follows (Lennox): Every fit is accompanied by a disturbance of the normal electrical activity of the brain. The three main types of fit have distinct forms of altered rhythm, the distinguishing feature being the frequency of the waves. In the major fit the waves are abnormally fast; in the minor fit they are alternately fast and slow; while in the so-called psychical fit the waves are slow. The pattern for each patient is apt to show individual peculiarities which repeat themselves in successive fits.

The electro-encephalograph also records fits which are clinically invisible ("subclinical") and abnormal variations of rhythms in epileptic subjects may

be found during the intervals between fits. These electrical changes are found alike in "idiopathic" and in "symptomatic" fits, and afford further evidence in favour of a unity in epilepsy rather than of a number of "epilepsies."

Lennox suggests that epilepsy may, therefore, be regarded as expressing a defect in the rhythm-regulating mechanisms in the brain, or in other words as a "paroxysmal cerebral dysrhythmia." He also suggests that the cause of epilepsy is never single, that there is a fundamental cause; namely, an inherent instability of the brain, and a secondary or contributing cause. Of such there are probably very many, and it is the combination of the two that evokes the fit. It should be borne in mind, however, that we can say no more with certainty than that these variations in cortical electrical rhythm accompany the fit. We may not in the present state of knowledge say that they cause it, and the search for the factor or factors productive of the electrical disturbance remains for the future.

This brings us to a consideration of the many factors that in the past have been held responsible for the production of fits. These do not demand or deserve any detailed discussion, for they are predominantly speculative and have always lacked confirmation, either pathological or biochemical. Disturbances of metabolism, of endocrine function, and of acid-base equilibrium have all been invoked. Yet research has failed to find any constant or characteristic disorder of protein, fat or carbohydrate metabolism, or of endocrine function of any kind, nor have studies of the blood in respect of its cellular or chemical composition been other than negative. The uncertain influence of metabolic and endocrine disorders, when these are manifestly present, in the production of fits tend to strengthen the growing view that none of these factors plays any but a secondary and contributory rôle in the malady. There are, on the other hand, certain well-known clinical features of epilepsy that have in the past been deemed to point to the importance of some metabolic variations in exciting fits; thus, the increased incidence of fits in women at the time of the katamenia, and their common cessation during pregnancy have been noted in this connection.

The features of the fit suggest that the functions of the affected regions of the brain may be disordered in several ways, by suppression, as, for example, in the blunting or loss of consciousness which is probably the most constant and invariable feature of epileptic fits of all kinds, and by release, as in the hallucinations (visual, auditory and gustatory, etc.), the delirium and automatism that are sometimes seen. The actual convulsive moments of the generalised or of the Jacksonian fit can hardly be regarded as release symptoms but point from their form and character to direct stimulation of cortical cells.

Morbid Anatomy.—As has already been stated epilepsy has no histopathology, though in the brains of chronic epileptics various minor changes have been noted. Much of the material from which information on this point has been gathered is useless, since the subjects have been aments or demented, and the relation of the changes found to the occurrence of fits during life is uncertain. Among the changes thus found are smallness of the brain, meningeal thickening, convolitional atrophy and fibrosis of the Pacchionian bodies. But none of these is invariably present, and we say that there are no certainly essential macroscopic changes in the epileptic brain. Histological examination has proved equally inconclusive. Sclerosis of the cornu Ammonis,

consisting of degenerative changes in nerves, cells and gliosis, have long since been reported, but of their significance nothing can be said. In short, as has been already stated, "there is no histopathology of the fit." Examination of the endocrine organs has also given no significant results.

Heredity.—In the past great stress has been laid upon the alleged heritable qualities of idiopathic epilepsy, and they have been made the basis of sweeping prohibitions in the matter of the marriage and child-beggetting and -bearing of epileptic subjects. Nevertheless, it seems that direct transmission of epilepsy from parent to child is exceptional, and this has certainly been the experience of the present writer in dealing with the abundant material that presents itself at the out-patient clinics of such an institution as the National Hospital. Recently Cobb has expressed the view that on so-called eugenic grounds as high a proportion as 90 per cent. of epileptics cannot reasonably be advised against marriage. Statistical reviews by others (*e.g.* Muskens, Marchand) seem to confirm this view. Marchand maintains that after the exclusion of all those cases of epilepsy in which gross external factors, such as syphilis, injury, etc., can be excluded there is nothing that can be called a familial epilepsy capable of direct transmission without the intervention of adventitious factors. In congenital affections resulting from developmental defects of the ectodermal layer, epilepsy when it is seen is no more than the symptom of a cerebral lesion. In support of these views he has been able to marshal a great weight of evidence. On the other hand, recent electro-graphic observations of Lennox suggest that in the parents (one or both) of epileptic subjects abnormally wide fluctuations in the rhythm of the brain potentials are unduly frequent, and it may be that while epilepsy as such is not inherited, some instability of cortical cell function may be inherited, which in combination with other factors (when one or more of these chance to be present) lead to the appearance of epilepsy. This is approximately what Marchand maintains in more general terms.

Exciting Causes of the Fit.—In the majority of cases of idiopathic epilepsy no cause whatever can be found for the occurrence of the first fit. It may follow unpleasant emotional excitement, alcoholic excess, or may occur completely unheralded while the subject is in his normal health and environment. In the case of frankly symptomatic epilepsy, metabolic disturbances in early life, and especially rickets, are potent causes. Acute intoxications with absinthe, lead, bismuth and many other poisons may invoke epilepsy, as may also the poisons occurring in the specific fevers in childhood, in uræmia, cholæmia, hyperpiesia and puerperal eclampsia. And although in these intoxications the epileptic phenomena do not usually recur after the cause has disappeared, yet there is not one of the above-mentioned conditions which has not been followed by persistently recurring epilepsy. Injury to the brain of any nature whatever, whether from violence from without or from disease within, may cause epilepsy. Traumatic cases in which the brain has been severely wounded are not associated with epilepsy in a greater percentage than 5 per cent. Cerebral tumours, agenesis, encephalitis meningitis, cerebral syphilis and vascular lesions give a higher percentage, which in children has been placed as high as 30 per cent. Another form of symptomatic epilepsy that should be borne in mind when a previously normal person who has lived abroad, particularly in the tropics, develops epilepsy is that due to cysticercosis. The cysticercus or bladderworm stage

of *T. Solium* normally develops in the pig, and infestation of man by the adult tapeworm is due to eating pork thus infested. Man may also accidentally eat tapeworm eggs and serve as the intermediate host, the embryos showing a tendency to invade the brain. MacArthur, who has drawn attention to this factor in the development of fits, records 20 personally observed cases of cysticercosis of the brain, 6 of them soldiers invalided from abroad. The fits may be Jacksonian in type or generalised.

Symptoms.—**PRODROMATA.**—The circumstances which immediately precede the occurrence of an attack are of some importance. Speaking generally, it is uncommon for an attack to occur when the attention is fixed, or when some act is being performed, and from this it follows that the epileptic is relatively or absolutely free from attacks when at work and doing, and only in the rarest cases comes to harm or injury from accident. Some patients are able, by an effort of will in fixing attention, or by the performance of some vigorous action, to arrest attacks which have already begun.

Sometimes a change in the general condition of the patient may make him aware, or may acquaint those around him, that an attack is pending, and such signs of altered metabolism may herald an attack for from a couple of hours to a week. Headache, irritability, restlessness, euphoria, lethargy, somnolence, unusual appetite and a peculiar vacant look may all be met with in this connection.

Not infrequently the attack is preceded by paroxysmal manifestations which are in reality minute attacks, such as partial lapses in consciousness, a sense of strangeness, "dreamy state," jactitations of any of the muscles exactly resembling those seen in uræmia, slight auras, giddiness, sneezing and yawning.

DESCRIPTION OF THE ATTACKS.—The varieties of the epileptic attack are legion, and several types may occur in the same subject—indeed, it is unusual for fits to be always of the same type in one subject. They tend to vary both in degree and nature. They are usually divided into the less spectacular "minor" attacks, in which spasm is not a prominent feature; and "major" attacks, in which spasm is conspicuous. This distinction is purely artificial, for most patients have attacks of both varieties, and the two merge by insensible gradations the one into the other. Further, the minor attack often is the initial manifestation of the major attack.

The following description will serve to illustrate the more definite manifestation of epileptic attacks:

1. *Simple jactitation.*—Single twitching of individual muscles or groups of muscles, occurring, now in one part of the body, now in another, are seen in the majority of epileptics at some time or other. They are conspicuous in the convulsions of childhood, where they often constitute the chief clinical feature. They are well known as the "carphology," or "subsultus tendinum," of uræmic and eclamptic attacks, and in the "typhoid state." They may be not infrequently noticed in the epileptic person when he is otherwise well, and engaged perhaps in conversation or other occupation. Gowers emphasised epileptic twitching as a prodroma of an oncoming severe attack; but while in some instances this is undoubtedly true, yet it frequently occurs when no attack follows. It has been called "epileptic myoclonus."

2. *Simple loss of consciousness.*—In this, the commonest of all minor

phenomena, there is a simple break in the continuity of consciousness. The train of thought and action is suddenly arrested for a few seconds, and there is a sudden stillness of posture and facial expression which attracts the attention of a witness. The face may show sudden pallor, a vacant expression, and curious fixity of the eyes, with large pupils. The patient does not fall, or move, or drop anything that he is holding. In a few seconds the attack is over, leaving the patient unable to describe what has happened, perhaps a little confused for some seconds, sometimes emotional and even hysterical. More often he continues what he was about as if nothing had happened. Such attacks sometimes occur very frequently, even hundreds in a day. They are characteristic of pyknolepsy, in which the prognosis is absolutely good, and also of a form of epilepsy in which rapid mental degeneration occurs and in which the prognosis is equally bad. Further, they may occur in organic disease of the brain.

3. *Simple loss of consciousness with falling.*—The patient suddenly falls, without warning, in the extended position, and almost always prone, so that his head reaches the ground first, and his forehead receives the bruise. He regains consciousness immediately, and picks himself up as if nothing had happened. It is not uncommon to see the forehead one region of scars, as the result of repeated falls; to prevent these a pneumatic protector should be worn. This form gave rise among the ancients to the name "falling sickness," or "morbus caducens." In another form of this type the head, or the head and trunk, alone are affected. The patient does not fall, but simply drops the head forward—"nodding spasm," or "spasmus nutans"; or he drops the head and bends the trunk forward—"salaam spasm."

4. *Simple loss of consciousness with slight spasm.*—This forms a gradation from the above types to the definitely convulsive seizures. The spasm is seen as conjugate deviation of the eyes, and perhaps of the head also, or it takes the form of laryngeal and respiratory action, giving rise to a groaning noise, or may involve any part of the musculature.

5. *Local fits.*—First studied by Hughlings Jackson, these events have the name of "Jacksonian epilepsy," and this term has unfortunately become coupled with common errors that are no part of Jackson's teachings. These are (1) that some local disease invariably underlies the Jacksonian fit, and (2) that the Jacksonian fit necessarily consists of local motor convulsion. Actually, in many cases naked-eye and microscopic examination may fail to reveal any local lesion, and none such may be present. Also, the Jacksonian fit may consist of phenomena involving any possible cortical function. It may be added that local disease of the brain quite commonly evokes generalised fits indistinguishable from those of idiopathic epilepsy, and conversely that the latter form of epilepsy may express itself in the form of Jacksonian fits.

Psychic fits.—These may take the form of peculiar mental states, of instantaneous onset, remembered afterwards sometimes in exquisite detail, sometimes only in vague character. Emotional conditions of fear or horror, which may cause the patient to attempt with violence to escape from his surroundings—"cursive" epilepsy—may occur. Or, the attacks may take the form of a sudden feeling of misery, or an intense sense of personal wrongdoing, a sense of intense familiarity in surroundings which are unfamiliar, a sudden sense of strangeness, as in a patient whose fit was "suddenly seeming

to be somewhere else," a sense of euphoria or of intense mental energy, a dreamy state, often associated with smacking of the lips and champing or swallowing movements, which often has a pleasurable emotional tone. Again, the psychic fit may take the form of a highly complex and detailed hallucination.

Visual fits.—These may take the form of negative phenomena, such as dimness of vision, complete darkness or hemianopia, or of positive effects, such as flashes of light, scintillating stars or balls of fire, or of both together in the form of blindness with flashes of light. In the last case they may closely resemble the visual phenomena of migraine, and are not infrequently caused by a local lesion of the occipital region. Complex visual hallucinations may occur.

Auditory fits.—The hallucinations of sound may be of any nature—hissing, booming and elaborate musical sensations, as of bells, being common. There is usually a sense of coincident deafness or "far away" hearing, which passes off with or soon after the sound.

In one case the fits could always be produced by sounding the hallucination note upon the open diapason of an organ. No other note or sound produced the fit. (Such directly excited fits, though very rare, are well known in connection with olfactory, visual, auditory and common sensory stimulation, and have been termed "reflex epilepsy.")

Olfactory and gustatory fits.—These hallucinations are always described as of "favour," usually unpleasant. Very often, movements of the lips, tongue and jaw, or swallowing movements are present, and the dreamy state already referred to may be associated. From the location of the functions of smell and taste in the cortex of the uncinate gyri, and from the common occurrence of fits of this character in lesions of these convolutions, this type of fit is often referred to as the "uncinate fit."

Sensory fits.—These hallucinations may have their seat of commencement in any part of the body. They may remain local, but more commonly they spread from the point of origin in terms of the local representations of the body in the cerebral cortex, and usually from the periphery towards the trunk and head, but a sensory fit may spread to the extreme periphery first. For example, commencing in the fingers, it may spread up the arm to the head, or on reaching the shoulder it may invade trunk and leg before ascending to the head. It may be bilateral, confined to the anterior or posterior aspect of the body.

The sensation may be described as "numbness," "tingling," "pins and needles," "vibration," "rushing," "as if the limb were withering," much more rarely actual pain. Sometimes the sensation is indescribable. The sensory attacks have their origin in a local disturbance of the parietal region of the cortex, and may indicate the presence of an organic lesion in that region. They may be accompanied or followed by temporary loss of sensibility, in the form of astereognosis, loss of sense of position, or anæsthesia.

Another group of sensory fits for which it is impossible to give any definite cerebral localisation at present, is that of the so-called visceral auras, which are mainly referred to the distribution of the vagus nerve. Such are the very commonly occurring "epigastric" sensation, and sensations of choking, dyspnoea, nausea and cardiac sensations.

It is quite possible that the sudden feelings of malaise or of faintness

which may constitute the main feature of some epileptic attacks are expressions of the sudden lowering of blood pressure which is known to immediately precede the epileptic attack.

Disturbances in the realm of the vestibular nerve are common indications of epilepsy. Sudden giddiness may be the sole indication of epilepsy, and is a common initial event in major attacks. It may be indicative of the sudden fall of blood pressure, or the feeling of rotation may be consequent upon early spasm causing conjugate deviation of the eyes.

It must be carefully borne in mind that all the phenomena which have been described above may occur as isolated events and so constitute the epileptic attack. Often, however, the disturbance of the cortex spreads widely, involving general convulsion and loss of consciousness; but the initial phenomena are remembered by the patient as the "warning" of the attack and have from ancient times been termed "auras," when preceding general convulsion. In reality, they constitute the essential part of the attack as showing the region of the brain in which the disturbance starts, and in every patient who has such "warnings" preceding his severe attacks, the warnings occur at times by themselves without any such sequel.

Motor fits (simple paralysis).—This is the rarest of all forms of the epileptic attack. It consists in a sudden inability, relative or complete, to use a limb or one side of the body or the whole voluntary musculature, with no preceding convulsion. There are the usual signs of cerebral paralysis—at first flaccidity with a tendency for the jerks to fail; a few moments later increased jerks, with absent trunk reflexes and extensor plantar reflexes, all of which signs soon disappear. It may occur as an isolated phenomenon. More often a slight "minor" attack or a local sensory attack accompanies the onset of the paralysis. Sometimes such an attack may result from local disease of the brain. Such attacks when involving the right face or right side of the body may occasion aphasia, or the aphasia may occur alone as the attack of simple paralysis. Such attacks of simple paralysis without convulsion are well known in uræmia, hyperpiesia, metallic poisoning and general paralysis of the insane.

Local convulsion.—The common foci of onset are the angle of the mouth, the thumb and index finger, and the great toe, but the spasm may occasionally begin elsewhere. It rarely produces conjugate deviation of the eyes as a primary movement, but usually in association with, and secondary to, deviation of the head. The convulsive movements may remain confined to their place of onset throughout the fit, or may spread widely so as to involve a whole limb, one-half of the body, or the entire musculature. In fits involving the musculature of the right half of the face and tongue, speech is usually lost during the attack and returns shortly after its cessation. Spasm never affects the muscles of one eyeball alone, but the spasm is in terms of conjugate deviation of both eyeballs in one direction. The same rule applies when the neck is affected, for the head is then either rotated to one side or extended or flexed on the chest. With the other bilaterally associated muscles it is different, for the tongue is affected on one side only, as is also the face. The onset is with tonic spasm, which after a little while gives place to broken or clonic spasm, becoming more and more intermittent and finally ceasing. In some cases, but by no means in all, the convulsion

leaves varying degrees of weakness in the affected muscles—Todd's paralysis or post-epileptic paralysis, with transient signs of loss of function of the pyramidal system, such as loss of trunk reflexes, increase of jerks, and extensor plantar reflexes.

Epileptic spasm usually puts the hand in the position of extension at the interphalangeal joints, flexion and abduction at the metacarpo-phalangeal joints, flexion at wrist and elbow, and adduction at the shoulder. The feet are dropped and inturned, with extension at the knee and hip. Usually the trunk is in opisthotonos.

The sequence of tonic spasm at first, followed by clonic spasm, though usual in epilepsy, is not invariable. Purely tonic fits may occur with no clonic spasm, the tonic spasm remitting suddenly. Such fits are usually of slight severity and duration, and are almost always general and very rarely local.

On the other hand, the spasm may be clonic only. The simple jactitation already described may be taken as a simple clonic fit. Local fits, especially of the face and of the hand, may be purely clonic. Again, some of the most severe of all general epileptic convulsions are clonic throughout so far as the limb and trunk musculature is concerned, but some tonic conjugate deviation of eyes and head is usual.

Loss of consciousness in local fits.—This seems to depend upon the extent of the cortex involved. With narrowly confined fits there may be no impairment at all, as in local convulsion of the face or hand, or as in a patient who vividly described to me a slow visual fit as it was occurring. When the fit spreads, consciousness is usually impaired, and when lost, it is lost late in the fit. For example, it is usual for a convulsion which spreads to one-half of the body to cause some impairment, and if it involves both sides generally consciousness is always lost.

General convulsive fits (haut or grand mal).—There is some reason for believing that every major attack has a local commencement in some region of the brain, and that it is in reality a local fit which rapidly becomes general. When such an attack commences with a local aura there is proof positive of local commencement. When it commences with conjugate deviation of head and eyes to one side, this is certain indication that the disturbance commences in the opposite hemisphere. When the spread of the disturbance is so rapid as to cause instant loss of consciousness there is no memory to retain the initial event of the attack. The seizure may begin with any of the local manifestations above described, the epigastric aura and giddiness being two of the most frequent. Or the patient may be only aware of his attacks from the condition in which he finds himself after their occurrence. The tonic spasm commences with conjugate deviation of both eyes to one side, followed by rotation of the head to the same side. The blood pressure falls, the countenance is for a moment pallid, the eyes widely open, the pupils dilated, the cornea insensitive. The march of the tonic spasm usually causes head retraction and opisthotonos; the upper extremities are stiff in flexion and adduction, the lower extremities in extension. If standing, the patient falls usually backwards, but the conjugate deviation of head and eyes may bring his face to the ground first. The respiratory muscles and larynx, going into spasm, produce the epileptic "cry," and the respiratory movements being no longer possible the face darkens with the asphyxia, and the sphincters

may relax, with the evacuation of bowel or bladder. The protrusor spasm of the tongue and the closing spasm of the jaw may cause the tongue to be bitten. After the tonic spasm has lasted some seconds and perhaps has produced such a degree of asphyxia as seems hardly compatible with survival, it begins to break into a series of sudden shock-like, jerky movements—the clonic spasm—which continue for some seconds, becoming less regular and occurring at longer intervals until, with a final jerk, the muscles become perfectly limp. Meanwhile the relaxation of the respiratory and laryngeal spasm have allowed the respiratory movements to return and to churn up the saliva, often bloodstained, which escapes at the nose and mouth in the form of froth. At the end of the attack there is complete and unrousable loss of consciousness, the pupils are dilated and insensitive to light, the corneal reflexes absent, the knee-jerks absent, and the plantar reflexes extensor in type. In a short time the knee-jerks return, the plantar reflexes return to the normal, and consciousness returns. Usually the patient is dazed, feels ill, has marked headache, and if left to himself soon sleeps heavily for some hours. It must be noted that the general convulsive attack almost always leaves the patient face downwards, so that he has drowned in a puddle an inch deep and has been asphyxiated by his own pillow. The latter event is by very far the commonest way the epileptic meets his death from accident in a fit.

The epileptic cry.—There are two quite different sounds that may occur at the commencement of an epileptic attack. The one is a natural, conscious cry of terror at the advent, as in the patient who alternated piercing screams with “It is coming! It is coming!” before the convulsion commenced. It is curious how rarely any memory of such cries or utterances remains with the patient. The other is the epileptic cry proper—a weird, unearthly, hollow sound, produced by inspiratory spasm drawing air over the nearly closed vocal cords. This cry occurs in a minority even of severe cases, for the obvious reason that it is determined by a particular march of the spasm. If the inspiratory spasm occur before the larynx has gone into spasm or after it is in spasm, there can be no laryngeal noise, but only the commonly witnessed pharyngeal and buccal grunting and gurgling. The spasm must be so timed that the inspiratory spasm must occur as the larynx is closing, and this only obtains in a minority of the cases.

Tongue-biting.—Some patients always bite the tongue, others never, and some now and again. The tongue is always bitten at the side and some way from the tip, because it is deviated to one side in the spasm and its thicker part brought between the molar teeth. The same side is always bitten. The tongue cannot be bitten unless protrusor spasm occur either before the jaw has gone into tonic spasm or after it has broken into clonic spasm. If any other march of spasm occur, the tongue escapes. It is remarkable how little scarring occurs even from severe and repeated tongue-biting unless a piece is bitten clean out.

Incontinence.—Though common, incontinence is by no means the rule even in severe attacks. More often it is the urine alone that is evacuated, much more seldom the bowel alone, still more rarely both. A rare phenomenon during an epileptic fit is seminal emission. The occurrence of this has been denied, but the present writer has seen it occur.

Secondary events.—The degree of asphyxia during the attack may

be severe, and blood vessels may give way under the stress, with the production of surface ecchymoses or deep hæmorrhages, including cerebral hæmorrhage. The spasm is powerful and may give rise to much subsequent aching, as if the patient had been beaten all over. It may dislocate joints, rupture muscles and even break bones. A dislocation once produced in a fit always recurs with subsequent fits.

Duration of epileptic attacks.—Two minutes may be given as an outside time-limit for the duration of an individual attack, from its commencement to the end of the active phenomena, and in convulsive attacks to the end of the spasm. Usually the time is much shorter than this, and often is a few seconds only. Sometimes attacks are described as of much longer duration. When analysed, such attacks will be found to be a series of attacks with very short intervals, or slight attacks with post-epileptic functional spasm, or hysterical attacks.

Conditions after attacks.—The epileptic fit may leave no after-effects whatever, even though it be severe, but this is unusual. On the other hand, even the slightest attacks may cause conspicuous sequels. Sleep and headache are very common, especially following convulsive attacks, and they may be alternative effects, in that if sleep occur there is no headache, but if it be prevented there is severe headache. The post-epileptic paralysis of Todd has already been described, and also the aphasia which may follow right-sided attacks. The mental state is usually affected by the attack, and returns to the normal—sometimes quickly, sometimes slowly. Commonly the patient is dull and dazed, speaking at random, unreceptive, irritable, and does not fully recognise his surroundings. During this state of impaired consciousness he may pass into a condition of mental automatism, in which various acts are performed in a conscious manner but of which no recollection is afterwards retained. One patient always prepared for bed after her minor attacks, and proceeded to undress in the stalls of a theatre. The acts performed during post-epileptic automatism may have a true relation to the life and mentality of the patient. He may do spiteful and criminal acts to those he dislikes. This fact has an important bearing as regards the criminal responsibility of the epileptic. In other cases a patient after recovering from the epileptic fit passes at once into a state of hysterical convulsion. Both these post-epileptic conditions occur commonly after minor attacks, but they may also occur after major fits; they seldom occur when convulsion has been severe.

Vomiting may occur after any type of epileptic fit, but it is most often met with after a convulsive attack. As it occurs during the period of unconsciousness, there is some danger of the vomited material being drawn into the larynx. Though Gowers mentions a case in which this event proved fatal, I have not come across any accident from this cause.

MENTAL DETERIORATION AND ABERRATION IN EPILEPSY.—Many epileptics, especially those who have frequent attacks, show signs of mental deterioration, which is often progressive, and which may become severe and end in chronic insanity; while others show no such mental troubles, and some of these fulfil a long life with the highest standard of capacity.

There seems to be no correlation between the type of epilepsy and mental degeneration, though the latter is widely held to be more frequent and more severe when many minor attacks occur.

The tendency to mental failure is greatest in the cases which commence in childhood, and lessens as age increases ; while, again, in the epilepsy commencing in the degenerative period of later life, the incidence again increases. In its slighter form there is merely defect of memory, of attention and power of acquisition. In more severe degree there is greater imperfection of intellectual power, weakened capacity for attention, and often defective moral control. Mischievous restlessness and irritability may develop to vicious and criminal tendencies with advancing age. Every grade of intellectual defect may be met with, to actual imbecility. Paroxysmal outbursts of mental derangement may be met with, sometimes transient and immediately following a fit, sometimes without a fit, and sometimes lasting for weeks or months. From what has been written above upon the cause of the mental disturbance in metabolic dyscrasia, these events will be easily explicable.

PERIODICITY.—While some patients may have fits at any time and at all times, yet there is a tendency in the majority for the attacks to occur at particular epochs and not at others. Epilepsy may be strictly “nocturnal” or “diurnal.” It may occur only on rising in the morning, or solely at the menstrual epoch. The fits may come in batches of several in one day, at intervals of many months, while 7-, 14- and 28-day periods are common. A knowledge of the periodicity when present is of great value in the successful treatment of epilepsy. “Rare” fits, which occur at very long intervals, are apt to present the most severe convulsion ever witnessed.

SPECIAL VARIETIES OF EPILEPSY

EPILEPSY FROM LOCAL DISEASE OF THE BRAIN.—Almost any lesion of the cerebral hemispheres may produce symptomatic epilepsy. But not more than 5 per cent. of all such lesions do this. The convulsions which may occur in cerebral thrombosis, encephalitis and meningitis are examples of epilepsy incident with the onset of an acute lesion. Usually the epilepsy is incident when the lesion has been present some considerable time. Lesions of the brain in childhood seem to be more commonly associated with epilepsy than when occurring in adult life. Agenetic states of the brain of prenatal origin (cerebral diplegias) are associated with epilepsy in 30 per cent. of the cases, and infantile hemiplegia is followed by epilepsy in about the same proportion. Increased intracranial pressure alone seems capable of causing fits, as in hydrocephalus and subarachnoid hæmorrhage, and this may be a factor in the epilepsy of intracranial tumours and meningitis. Abscess seems very rarely to produce fits.

The fits caused by local lesions may be in almost every respect identical with and indistinguishable from the usual type of epileptic manifestation, from the slightest momentary minor fit, all through the local sensory and motor fits, to the severe general convulsion of instantaneous onset and immediate loss of consciousness. There are the same auras and the same sequels. It may perhaps be said with relative truth that the splanchnic auras (epigastric, cardiac, etc.) are uncommon, and that there is a greater tendency for consciousness to be lost late.

The minor attack is the least common fit occurring as the result of a local lesion ; the general convulsion by far the most common ; while the local fit

holds an intermediate position, and its nature is often indicative of the position of the lesion.

PKYNOLEPSY.—This is a form occurring in children, so called because of the great number of the fits which may occur daily. These are of the slight minor type, any sign of spasm being infrequent. It is rare for any major fit to occur. There is no mental impairment whatever, no deterioration of health, and no result is obtained by any form of treatment. The malady invariably ends in spontaneous cure, usually before or at the age of puberty. Its separation from minor epilepsy is of uncertain validity.

CARDIAC EPILEPSY.—This is a convenient term for the epilepsy which occurs in Adams-Stokes' disease, and in paroxysmal tachycardia, and for the fits which may occur in congenital heart disease and in some forms of cyanosis. They cannot be the equivalents of asphyxial convulsions, for they are not met with in severe chronic cyanosis, and, on the other hand, there is usually no cyanosis at all when fits occur in Adams-Stokes' disease.

VASOVAGAL ATTACKS.—Under this misleading title, Gowers described a recurrent paroxysmal symptom-complex with some or all the following components: a sensation of fullness in the epigastrium; precordial pain or discomfort; difficulty in breathing; a sense of impending death; a slowness of mental operations but without disturbance of consciousness; a sense of physical fatigue; and coldness of face and extremities. These symptoms wax and then wane gradually, and may be present for as long as 4 hours from onset to disappearance.

Gowers stated that he used the term "vasovagal" as a purely descriptive one, but without implying any theory of causation. Unfortunately, those who have adopted his terminology have overlooked its lack of foundation. Further, the various descriptions of these attacks to be found in the literature are based almost wholly upon hearsay, the attacks themselves being but rarely observed, and do not provide any evidence of vasovagal involvement. Thus, the pulse is said to be accelerated, not decreased or irregular, while the facial pallor and coldness might equally be the result of local vasoconstriction or of splanchnic dilatation. A further vagueness has been lent to the conception by the different senses in which it has been employed. Thus, Collier has used the term for attacks in which convulsions and loss of consciousness occurred, though Gowers expressly stated that consciousness was not disturbed. In short, the term has no precise meaning, no sound basis of observation, and no proper place in neurological terminology.

The alternative term recently suggested for the attacks described by Gowers, namely, "periventricular epilepsy," must also lack value until we have some precise information as to the nature of the attacks themselves. Lewis has pointed out that the term "vasovagal" may rightly be applied to the common fainting or syncopal attacks, and it is better to restrict the term to these.

MYOCLONUS EPILEPSY.—In this group are included: (1) Epilepsy of an ordinary type in which there is much simple epileptic jactitation of the muscles between the fits; (2) cases of Unverricht's myoclonus in which epilepsy is coincident.

STATUS EPILEPTICUS.—In this condition severe convulsion succeeds severe convulsion at short intervals without any return of consciousness during these intervals. It is as if convulsion recurred so soon as the body recovered sufficiently from the exhaustion produced by the last convulsion.

Meanwhile the temperature rises, and may reach a hyperpyrexia. The difficulty in feeding and watering, the severe muscular exertion and the pyrexia add the dangers of acidosis to those of exhaustion, and the patient is very apt to succumb. Status epilepticus must not be confused with frequently recurring fits in which there is some return to consciousness during the intervals, though it frequently develops from such a condition; for the latter are not accompanied by a rising temperature, are more readily subdued, and are not of nearly so severe a prognostic import. If the convulsions cannot be stopped by treatment, the patient usually dies from sudden collapse, or, the fits ceasing, he remains delirious for a while, with rapid heart and high temperature, and dies of cardiac failure. Status epilepticus may be met with in acute lesions of the brain and in chronic lesions such as general paralysis of the insane. It may occur in acute poisoning with lead, bismuth and absinth. It may develop suddenly in any type of epilepsy whatsoever, sometimes without apparent cause, sometimes as the result of over-exertion and excitement, sometimes when medicines which have been regularly administered and which have kept the fits in check are suddenly cut off.

Diagnosis.—The recognition of epilepsy requires a working acquaintance with the nature of its many manifestations and especially of the slight forms, little exteriorised, which may be easily overlooked or misinterpreted. The sudden unexpected onset, without cause, the transiency, the recurrence, and the circumstances of the moment, are useful aids.

From syncopal attacks (rapid lowering of blood pressure) epilepsy can often be distinguished by the slow onset, the gradually increasing pallor or greyness, the distancing of sound, the nausea and flatulence, the presence of an obvious cause, the length and the stillness of a fainting attack.

The hysterical attack is easily distinguished by the fact that only the convulsion of epilepsy can possibly be confused: the other manifestations of epilepsy are never simulated by hysteria. Hysterical convulsion has not the manner nor the march of epileptic spasm. It never begins with conjugate deviation of head and eyes to one side, there is not the orderly spread of convulsion, and there is never but a poor imitation of the sequence of tonic followed by clonic spasms. The movements in the hysterical fit are purposive, spectacular, violent, and are liable to be increased by restraint and are rapidly abolished by complete inattention. The functional fit never occurs except in the presence of an audience, for it would then be purposeless, and it never occurs during sleep, the tongue is never bitten, though other parts of the body and other people may be. There is no transient abolition of the tendon jerks, nor transient appearance of the Babinski plantar response. The sphincters are never relaxed. Intense converging spasm of the eyes is a common feature of the functional attack, but this sign is not met with in epilepsy. When functional manifestations follow slight and rapidly transient epileptic attacks, the distinction between these and purely hysterical attacks is often difficult and sometimes impossible, except after long observation. For the initial epileptic attack may be practically unnoticeable, and the subsequent events may be typical of hysteria and are usually amenable to the same line of treatment. Often some point in the circumstances under which the attack occurs will settle the diagnosis. Any attack having occurred during sleep, or any attack in which the patient has fallen in circumstances of serious danger, as among the traffic of a London street, or any attack

occurring when the patient cannot attract the attention of others, establishes the diagnosis of epilepsy. The best plan is to regard every hysterical fit as possibly epileptic, and every fit of doubtful type as probably epileptic, until time and circumstance bring definite conviction.

Migraine may sometimes closely simulate epilepsy when sudden paralysis, or sensory auras, or visual hallucinations occur without headache. But while the sensory phenomena of migraine may last for 5 to 30 minutes, those of minor epilepsy have a duration of seconds only.

Careful search must be made in every case for all the bodily conditions with which epilepsy may be associated. Papilloedema, headache and vomiting may reveal increased intracranial pressure from some lesion of the brain; while local paralysis, sensory loss, visual or other defect may indicate a local lesion of the brain, past or present, and this may also be suggested by the nature of a local fit. The presence of rickets, infantilism, undue adiposity, etc., may indicate the presence of some definite metabolic dyscrasia or endocrine disorder. Renal function and the condition of the blood pressure should always be examined, for even in early infancy fits may be uræmic and in the recurring epilepsy associated with small white kidney, and with cystic renal disease, the causal disease is frequently unrecognised. Where syphilis is likely, the reactions in the blood and cerebro-spinal fluid should be examined. Lastly, any evidence of chronic intoxication by metals, alcohol, absinth, etc., should be sought for.

Cysticercosis epilepsy should be thought of when the patient has lived abroad. Diagnosis depends upon the palpation of cysts in the tissues, or the shadow in radiograms of calcified cysts in the muscles, or within the skull.

Prognosis.—The outlook in epilepsy is so variable that it is difficult to indicate any but the broadest principles in prognosis. Nor can a definite forecast be made in any case until the result of treatment has been watched for some time; for cases apparently favourable may prove rebellious, and those most unfavourable may turn out brilliant successes. Speaking generally, a cheerful outlook is justified in all cases except those in which there is progressive mental deterioration, and in these the outlook is hopeless in proportion to the rapidity of the mental change. Naturally, in those cases which are associated with serious bodily disease, such as brain tumour, renal disease and hypertension, the prognosis involves that of the exciting condition.

The danger to life from the epileptic attack itself, either directly or indirectly, is not great. However severe the fit, it is extremely rare for death to occur, and when this happens it is from turning over and smothering with the wetted pillow or from choking with the aspiration of vomited material. Injury, burning and drowning may cause death, yet the number of epileptics who meet their death in this way is so infinitely small as almost to remove the danger of accident from practical perspective. In the rare status epilepticus, however, the danger to life may be very great. Spontaneous cessation of the attacks occurs in a small proportion of cases. The convulsive attacks of infancy, which continue for some years after all cause to which they can be attributed has passed away, often cease for ever at the age of 4 to 6 years. Again, after 20 years of age spontaneous cessation is met with, and it becomes more frequent as life advances. It is, in my experience, a

much more frequent event than writers upon this subject, with the exception of Gowers, have been willing to admit.

The probability of cure, arrest or amelioration by treatment may be entertained in all cases where no mental deterioration exists and where no insuperable bodily disease determines the epilepsy, in proportion as the only method of cure—the securing arrest of the attacks for a considerable time by drug treatment—can be adequately administered over a long period. It is greater when periodicity in the occurrence of fits allows these to be anticipated by drug administration. It is much greater when the following out of education, or the continuance of regular employment, allows of a fully occupied and satisfying life, and much less when education is stopped, pleasures and sports forbidden, and the patient condemned to social inferiority and ostracism, and to a gloomy, narrow life of inanition because he has a few fits. It is perhaps smallest when severe attacks occur daily or at short intervals and when both major and minor attacks occur in the same subject.

Treatment.—*General treatment.*—The general principles for the maintenance of health if good, or for its improvement if poor, should be adopted. Whenever possible, no change whatever should be made from the régime of life of a normal person. In childhood, education, discipline and pleasures and school life should be continued upon strictly normal lines, and the adult should continue with work and occupation. No advantage has accrued from the adoption of special diets, such as the abrogation of meat, the exclusion of salt or the use of purin-free foods. The production of a low grade of acidosis by a ketogenic diet is occasionally of value in the epilepsy of children. Alcohol seems to be an excitant of the epileptic attack and should be forbidden.

The forbidding of such pastimes as may be fraught with danger should a fit occur, such as swimming, boating, cycling and car driving, is necessary.

Marriage and pregnancy.—The subject of epilepsy sometimes seeks—but rarely heeds—advice as to the expediency of marriage, both in its effects upon himself (or herself) and in respect of any heritable qualities it may possess. Marriage has no necessary effect upon the course of epilepsy, and, as we have seen, direct transmission of the disease is rare. Therefore the sweeping medical prohibitions once so frequent in these circumstances are not in fact warranted by such knowledge as we possess. Every case must be considered on its merits. It has been noted that in the family history of the epileptic subject, migraine is a far more common antecedent than epilepsy, but no one would venture to advise the migrainous subject against marriage or parentage on any so-called eugenic grounds. In respect of pregnancy it is usual, though not constant, for fits to cease in the epileptic woman when pregnant, and in any event the occurrence of fits at this time constitutes no special danger and is not an indication for the artificial termination of pregnancy.

Institutional treatment.—In cases where there is low mentality, much mental degeneration or insanity, and with frequent fits, where no adequate care and occupation can be provided at home, there is every advantage in a colony, institution or asylum for epileptics. In such patients little or no good can be done by medicinal treatment, whereas regular work, discipline and interest often mitigate greatly the burden of the malady.

Surgical treatment.—There still remains a very widespread impression that local fits and fits following upon injury to the skull are likely to be benefited by decompression. There is, however, little evidence that such procedures benefit epilepsy of any kind. Cases are on record in which a cortical scar, or a meningeal adhesion to the cortex, has become an epileptogenic focus the excision of which has been followed by cessation of attacks. The presence and situation of such a scar having been previously determined by the lumbar insufflation of air and the taking of an encephalogram. In general the dictum of Pierce Clark is just, that "all operative measures upon the brain in epilepsy are allowable only when they are indicated by definite physical signs other than the fits." Thus, in a case of cerebral tumour producing epilepsy, operation is justifiable for the relief of the papilloedema, headache, etc., and with the hope of possible removal or of obsolescence of the growth following the decompression.

Medicinal treatment.—Further than the measures above described, the treatment of epilepsy is purely medicinal. There are two groups of drugs which have a remarkable effect in arresting or mitigating the occurrence of the attacks in epilepsy. They seem to have much the same effect, and may conveniently be combined or alternated in the treatment of any given case. Sometimes one group is found to suit an individual patient better than the other. No advantage seems to accrue from administering these remedies more than twice in the 24 hours, nor from using large doses. Moderate doses, such as will cause no deterioration in bodily or mental health, even if taken regularly and for years, seem to bring about the best results. The first group is that of the compounds of bromine, of which sodium bromide seems to have an advantage over the others, both as regards efficacy and toleration. The organic compounds of bromine are not so useful. Sodium bromide should not be given in larger doses than 25 grains (1·5 G.) to an adult, nor should more than 60 grains be given in the 24 hours. It is conveniently combined with arsenic (min. 2 to 3) in the form of liquor arsenicalis, since this has the effect of checking the occurrence of acne. If it be advisable to conceal the fact that bromide is being administered, Gelineau's "dragées," each of which contains 15 grains of potassium bromide, may be prescribed.

Bromism.—Even in ordinary doses, the bromides may cause some acne of the skin, especially in subjects who are prone to acne, but this is the sole derogatory effect of this remedy, which is of common occurrence. The true bromide rash, which was met with in the early days of bromide treatment when huge doses (even an ounce thrice daily) were in vogue, is highly characteristic. It is hardly ever seen in these days, but I have twice met with it from moderate doses of bromides. Mental dulling and conditions of sub-coma, which may occur from poisonous doses of the bromides, are rarely met with from appropriate medicinal administration, except in elderly subjects. The mental deterioration due to the epilepsy in certain cases is often attributed by the laity to this cause, but this occurs, and sometimes in much greater degree, in the absence of bromides.

The second group is that of the malonyl-urea compounds, of which phenobarbitone (luninal) and soluble phenobarbitone are examples. These are very powerful drugs, and must be used with care. Phenobarbitone has certainly the advantage over soluble phenobarbitone in being more pro-

longed in its action. It is conveniently prescribed in doses of $\frac{1}{2}$ grain to a child, and 1 grain, with a maximum dose of $1\frac{1}{2}$ grains, to an adult. In larger doses it is a powerful hypnotic, and in patients who have idiosyncrasy it may produce a troublesome kind of frenzy. It appears to be a more certain means of warding off attacks for many hours after its administration than is bromide.

Whatever remedy is chosen, whether it be the bromide or luminal or a combination of the two, it is essential if possible to anticipate the occurrence of the fit by the administration of the drug. Thus, if fits are nocturnal only, the remedy is given in a single dose at night, or if diurnal only, in a single dose in the early morning. Again, if, as often happens, the fits occur soon after waking, then the single nightly dose should be used. Or, if the fits occur or are more frequent at the menstrual epoch, they should be anticipated by increased dosage before and during that epoch. With fits that are diurnal and nocturnal, a night and morning dose should be used. As it is less important in patients who have employment when fits occur by night, and often most disastrous when they occur at work, for with the present Workmen's Compensation Act no company will insure a known epileptic, I prefer to give phenobarbitone as the morning remedy and bromide as the nightly remedy, since I consider phenobarbitone to be the greater safeguard against the occurrence of the attacks. The question at once arises, Why should two remedies be used? The answer is that these drugs are by no means identical in action, and that the nature of the cause of epilepsy certainly varies in individuals. Some patients do best on phenobarbitone alone, others on bromide alone, and others on a combination of the two, and the best course can only be determined after trial.

Recently, Merritt and Putnam in Boston have introduced the use of sodium diphenyl hydantoinate (dilantin, epanutin) for epilepsy. Favourable results are claimed for this in cases that have not responded well to bromide or phenobarbitone, and some confirmation of its value is being obtained in this country. Obtainable here under the names of epanutin and solantoin, it is dispensed in capsules containing $1\frac{1}{2}$ grains. For small children dosage is begun with $1\frac{1}{2}$ grains twice daily, increased to three or even four times daily until optimal results are obtained. For adults $1\frac{1}{2}$ to 3 grains twice or thrice daily may be given. Symptoms of intolerance or of overdosage are tremor and unsteadiness, and these call for reduction of dosage. It seems safe to say that for adults a dose of 3 to 4 capsules daily is without untoward consequences, or unpleasant symptoms.

When the change is being made from some other medication to epanutin, this should be gradual, one of the daily doses of bromide or of phenobarbitone being substituted in the first week, a second one in the following week, and complete substitution being achieved in the third week. It is perhaps too soon to estimate the value of this drug in comparison with its many predecessors, but in some instances it is certainly more effective in controlling fits. Its use is said to be contra-indicated in elderly persons with hypertension and in debilitated subjects.

Many other remedies have been advocated in epilepsy; a few only have stood the test of time and are still in use, both as alternatives and adjuvants to the treatment above given. These may be placed in order of merit as belladonna, digitalis and allied drugs, especially adonis vernalis, opium and borax.

Belladonna has a striking effect upon a few isolated cases. It makes some cases definitely worse. Digitalis and adonis vernalis are commonly used as adjuvants, and I think sometimes with benefit. Morphine given in careful doses is of great use in status epilepticus. Thyroid is often of value in the epilepsy associated with cerebral agenesis (cerebral diplegia), mental and bodily backwardness and infantilism. It probably acts by rendering bodily metabolism more normal and in enhancing development.

STATUS EPILEPTICUS.—The treatment of this condition, and that of rapidly repeated fits which not infrequently merges into status epilepticus, is quite different from that of epilepsy in general, for the remedies useful in the latter condition are useless and even do harm in this urgent and dangerous state. The first thing to be done is to check the convulsion, and this is best achieved by the hypodermic injection of $\frac{1}{4}$ th of a grain of morphine. (Gowers preferred hyoscine.) Another remedy is paraldehyde, in large doses (360 minims), and this has recently been successful at the National Hospital. It has the obvious advantage that it is stimulating and not depressant. The remedies formerly used such as bromide and chloral by the rectum are worse than useless. The next measure is to secure that the patient shall be provided with adequate stimulants in the form of food, water and even alcohol. To which end a nutritious liquid meal of high stimulating value and containing sugar to combat acidosis should be given by means of the nasal tube at regular intervals. An action of the bowel should be obtained as soon as possible, with a rapidly acting aperient administered with the food, and by warm water enemata. The pyrexia should be controlled by sponging repeatedly, and if high by continuous immersion, and this alone will sometimes have a dramatic effect in checking convulsion when pyrexia exists. When consciousness returns, feeding and stimulation must be carefully continued, with a gradual resumption of the routine treatment of epilepsy.

NARCOLEPSY.—In this remarkable and, as Adie has shown, by no means rare condition, two quite different kinds of attack occur. The one is the sudden onset of apparently normal sleep, which comes usually at a moment of inattention, several times a day. The sleep lasts from a few seconds to a few minutes; it is rousable, and the patient is wide awake at once and knows that he has slept, and sometimes that he has dreamed and can describe the dream. Many of the patients have a warning in the way of a feeling of intense fatigue and, thereafter, can so far repel the onset of the attack by an effort of will as to be able to get out of harm's way; but the attack is inevitable, and it is always the more severe the longer it is resisted. The second variety of attack is called the "cataplectic" attack, and this is produced mostly from a sudden emotion, which may be of any kind, but is usually an emotion which provokes laughter. There is a sudden feeling of intense weakness in the limbs, which become flaccid. The patient drops anything that he may be holding and crumbles to the ground, but often only into the sitting position. The eyelids drop and the head falls forward with the jaw dropped, and there is sometimes twitching of the muscles of the face, tongue and neck. There is complete inability to move the limbs and generally inability to speak, but consciousness is completely retained, so that the patient is afterwards able to recount every event and repeat every word spoken during the attack. In one typical case a cataplectic access was habitually induced by feelings of amusement or of anger, but

surprise was equally effective. Thus, when fly-fishing, a suddenly rising fish caused the patient's rod to droop in his suddenly weakened arm, and when shooting his gun drooped helplessly if a bird broke unexpectedly into view near him.

Usually an idiopathic malady, narcolepsy has been recorded in a few instances as a sequel of encephalitis lethargica. The malady appears never to be familial, and it does not occur before the age of puberty. Once developed, it usually continues throughout life, with variable frequency of the attacks. In most cases (amphetamine) benzedrine sulphate is specific in its action on narcolepsy. An initial dose of 1 tablet (10 mgm.) after breakfast and another after luncheon. This may be cautiously increased to 2 or even 3 tablets after these two meals, with a smaller dose after tea. Symptoms of overdosage are sleeplessness, restlessness and tremulousness. Less efficacious, but yet of distinct value in some cases, is ephedrine sulphate of doses of $\frac{1}{2}$ grain twice daily.

MIGRAINE

Synonym.—Paroxysmal headache.

Definition.—A common malady of which the only essential characteristic is recurring intense headaches, which usually develop on waking in the morning, and may be unilateral, frontal, occipital or general. The attacks usually date from childhood, but sometimes commence during later life. The headaches are often associated with vomiting, which has given rise to the designation "sick headaches" or "bilious attacks," with which is associated much vestibular disturbance as in sea sickness, and with peculiar disturbances of vision. Less common symptoms of the disease are peculiar slow sensory auræ, which occur in no other malady, attacks of hemiplegia or monoplegia or of aphasia, and attacks of ophthalmoplegia. Some of these phenomena may accompany the headaches, but others occur in attacks quite apart from the headaches, and may for that reason give rise to difficulty in diagnosis.

Ætiology.—The malady commonly makes its appearance at about the age of puberty, and tends to persist, with fluctuations in severity and frequency of attacks, until middle age, disappearing in women with the menopause. Its persistence in old age is exceptional. A history of familial incidence is common, and the subjects are commonly of an energetic and intelligent type.

Nothing is known with certainty of its essential nature of causation, and in consequence hypotheses are rife. Thus, digestive disorders, errors of refraction, of metabolism and of endocrine function have all been evoked as responsible factors, though without clear evidence in the case of any of them. Yet the correction of such errors when they obtain does not suffice to cure the malady, and it is probable that they are never more than precipitating factors in susceptible subjects. Spasm of cerebral arteries has also been suggested as a cause of the attacks, but again without adequate grounds.

The subjects of migraine are often bad sailors and bad train travellers, and mental and bodily fatigue and emotional disturbances are commonly

followed by an attack. It is important to remember that tumours of the occipital lobe and also intracranial aneurysms may be associated with attacks exactly resembling migraine.

Symptoms.—The subjects of migraine are usually otherwise quite healthy, and are often robust and strong. No peculiarities of blood pressure are noticeable. Premontory signs of the attacks are present in some cases, and these may take the form of an unusual feeling of well-being and intellectual acuity, or, on the other hand, of lassitude and depression. The attack commences most commonly on waking in the morning, when on raising his head from the pillow the patient experiences a sense of vestibular disorientation with giddiness, ocular confusion and nausea, such as is commonly felt at the commencement of sea sickness. It is at this stage of the attack, and within a few moments of its commencement, that the visual phenomena occur when these are present. Often the patient vomits at once from the vestibular disturbance, but sometimes vomiting is delayed for hours, and the vomiting may be continued as long as the giddiness persists. The visual disturbances last but a short time (from 10 to 20 minutes), but leave, as a rule, some confusion of vision and discomfort throughout the attack. The headache follows shortly upon these initial symptoms. It is cumulative, and expansile in character, and often begins constantly in a localised spot in the temple, forehead or eyeball, as a sharp boring pain which gradually spreads, and may involve the neck and arm. The pain may be unilateral, frontal, occipital or quite general. As the headache increases the face becomes pale and grey, the patient becomes much prostrated and incapable of mental or physical effort, and is unable to take food. Light, noise and movement aggravate the pain. After remaining in this condition for many hours, he falls into a heavy sleep, and awakes next morning shaken by his illness, but otherwise well. The above description covers many attacks of migraine, but many variations occur. The attacks do not always occur on waking, they may come on at any time of night or day. They may be rapidly transient, lasting but a few hours only, or they may last for days and even as long as 3 weeks, and give much anxiety in the attempts to provide nourishment and sleep for the patient. In some cases of long standing, the attacks become less severe towards middle life, and a persistent aggravating headache may develop between the attacks. When such a persistent headache is complained of alone, it is very important to inquire about preceding migraine, for the same treatment is applicable to the two conditions. One of the most characteristic features of the headache is that when once it is in full swing, no remedies will relieve it except natural sleep.

Visual phenomena.—Considering how very common migraine is, it must be clearly understood that any visual phenomena except slight confusion of vision accompanying the attacks, are rare. These may take the form of general mistiness of vision, floating spots, scotomata, bright stars and colours, hemianopia, double hemianopia with complete blindness, or psychic hallucinations of vision. In connection with scotoma and with hemianopia, the phenomenon of teichopsia may occur as follows: Upon the dark background of the scotoma or hemianopic field, a ball of light appears, which grows larger and becomes dark in the centre. This ring of light breaks at one spot, opens out and takes the form of a series of entering and retreating

angles (castellation figure) which become gloriously coloured (fortification spectrum) and which later become fragmented and fade. These visual events usually occur at the very beginning of the attack, before the headache develops, and they are rapidly evanescent, but they may occur as isolated phenomena, when no headache occurs.

Aphasic attacks may take the form of confusion of speech, word-blindness, or even of loss of speech-acceptance and exteriorisation. They accompany the headaches and occur at the commencement of the attacks. They are not of common occurrence.

Hemiplegic and monoplegic attacks usually occur quite apart from the attacks of headache. They, too, occur on waking, and consist of a transient uselessness and weakness of limbs, which lasts a few hours only. They are characterised by their occurrence in young subjects who suffer from pronounced migraine; they are rapidly transient and are not accompanied by organic signs, and almost invariably other members of the family are migrainous, and suffer with similar attacks of paralysis. In Michell Clarke's cases, 11 members of one family in three generations were so affected.

Sensory aura.—These are somewhat rare events, but they are pathognomonic of migraine, and usually occur quite apart from the headaches. The aura commences upon the periphery of a limb and is likened to that which would be produced by a multitude of cold-footed insects creeping on the skin. It travels very slowly towards the proximity, taking half an hour or more to reach from the fingers to the head, and is very alarming to the patient. It disappears rapidly without further event. It is the only aura with an exceedingly slow spread.

Ophthalmoplegia.—This is a very rare but most important event. It occurs only at the height of the headache, in severe attacks. Indeed, the patients usually say that the headache, during which the ophthalmoplegia occurred, was the very worst they had ever experienced. It is a paralysis of the oculomotor nerve trunks, most commonly of the sixth nerve alone, but sometimes of the third or fourth nerves, or of a combination of these three. It is generally unilateral, but may occur simultaneously on both sides. Severe diplopia results. It passes off in from a few days to a few weeks. When once it has occurred, it is apt to recur with subsequent attacks. In one of my patients, paralysis of the sixth nerve persisted for 18 months, gradually lessening between the attacks, and becoming complete with each fresh attack of headache. It disappeared completely, with the cessation of the headaches, when adequate treatment was adopted.

Diagnosis.—This presents little difficulty, if it be borne in mind that long installed recurring headaches on waking in the morning are surely migraine. The condition of the urine should exclude those renal cases with migraine-like headaches. Each case should be carefully examined for signs of organic nervous disease, and especially for persistent hemianopia and papilloedema, which would indicate an organic lesion of the occipital lobe. Those who are not familiar with the full range of sensory symptoms that may precede the onset of the headache, and do not realise the severe disturbance of the speech function which in some cases accompany them, are apt to take an unduly grave and erroneous view of the history given by a subject who has experienced them. Thus, a diagnosis of petit mal, or of cerebral tumour is not rarely made. It should be remembered, therefore, that the disturb-

ances which occur in a minor epileptic attack are momentary in duration and never persist, as do the migrainous symptoms in question, for several minutes. Again, consciousness is neither lost nor blunted in migraine. Although, as has been pointed out, a rare case of migraine ultimately turns out to be one of cerebral tumour, the latter diagnosis cannot readily be made unless there are supporting signs of a structural lesion. It is probable that the so-called ophthalmoplegic migraine, in many instances at least, is a symptom-complex bearing no relation to true migraine but dependent upon a gross intracranial lesion, perhaps most often an unruptured aneurysm of the internal carotid artery.

Treatment.—There are but few cases of migraine that cannot be materially benefited by treatment. Some are completely cured, while in others the attacks become milder and occur at much longer intervals. Careful attention should be paid to improvement of nutrition and general health, should these be defective. The avoidance of undue fatigue and of worrying emotions, and of any factors which are known to produce the attacks, is important. Errors of refraction should be adjusted if they are important. Medicinal treatment is by far the most useful agent. A mixture containing min. 1 of liq. trinitrinæ, min. 5 of liq. strychninæ, min. 10 of tinct. gelsemii, and 10 grs. of sodium bromide, made acid to preserve the stability of the nitro-glycerine, and administered thrice daily for many weeks or months, was advocated by Gowers; and truly there are few cases of migraine which do not derive great benefit or complete cure from this treatment. Phenobarbitone, in doses of 1 grain given every night, is most valuable, and may be used in addition to the foregoing prescription. The individual attacks are difficult to relieve. Sometimes a full dose of phenazone, acetanilide, phenacetin or aspirin given at the very commencement of the attack will ward it off, but these are useless when the headache has fully developed. The use of ergotamine tartrate—by mouth or hypodermically—has recently been recommended as an effective method of cutting short an attack of migraine. In some subjects, though by no means in all, it is useful for this purpose. Sometimes a full dose of alcohol has the same effect. It remains to keep the patient as comfortable and quiet as possible till sleep occurs. Where the attacks last over the 24 hours, and especially when they last for days, the only remedies are to induce sleep and to keep the patient nourished, the latter object being difficult to attain in long-lasting attacks. To this end hypnotics, such as barbitone or carbromal (adalim) may be used.

JAMES COLLIER.

Revised by F. M. R. WALSHE.

CHOREA

Synonyms.—St. Vitus' Dance; Sydenham's Chorea; Rheumatic Chorea.

Definition.—Chorea is an affection of the nervous system caused by rheumatic infection, and characterised by the occurrence of spontaneous involuntary movements, irregular both in time and in place of occurrence and in nature; by inco-ordination of voluntary movements; by muscular weakness, and by a variable degree of psychic disturbance.

Ætiology.—Chorea is rare among negroes, Indians and coloured races, whilst it is especially common in Jewish races. It is much more common among the poorer classes than among the well-to-do. Its incidence is upon nervous highly-strung subjects rather than upon the phlegmatic, and this is probably to be explained by the fact that the rheumatic subject is likely to be nervous and highly strung. Chorea is practically unknown during the first three years of life, and is very rare before the fifth year has passed. Common between the ages of 5 to 10 years, it reaches its maximum incidence between 10 and 15 years. After the age of 20 it is rare, except in connection with pregnancy; but a few cases have been reported up to the age of 60 years which have certainly been examples of rheumatic chorea. Females are affected twice as frequently as are males. Heredity concerns the incidence of chorea in two ways: firstly, as regards the inheritance of the rheumatic tendency, which is the most important cause of chorea; and secondly, in respect of the inheritance of the neuropathic tendency, for it is when these two are coincident that chorea is most prone to occur. As early as 1802 rheumatism was regarded as the cause of chorea, and all subsequent investigations have upheld this theory. The family history of a choreic patient generally brings to light the occurrence of acute rheumatism, of cardiac disease and of other rheumatic manifestations among other members of the family. Often the patient has suffered with rheumatic erythema, purpura, rheumatic nodules, recurrent sore throat and growing pains before the appearance of the chorea; less often an attack of acute rheumatism or cardiac disease has occurred. A large percentage of those patients who have never shown any sign of the rheumatic state before or during the attack of chorea subsequently suffer with rheumatic symptoms. The British Medical Association Collective Investigation Committee found that rheumatism preceded the chorea in 26 per cent. of the cases, and that in 46 per cent. of the remainder rheumatic signs accompanied the chorea, or appeared subsequently. If to the total of choreic patients who present rheumatic signs at some time or other, one adds those with no personal history of rheumatism, but with a family history of rheumatism, it will be found that there are but few cases of chorea in which a personal or family history of rheumatism is absent.

Psychical disturbances.—Any emotional disturbance, such as fright, anxiety, depression or overpressure in school, may sometimes act as immediate determining factors, but much more often these events simply aggravate symptoms which are already present in slight degree.

Pregnancy.—The relationship of pregnancy to chorea is very definite. It is generally met with in first pregnancies, and before the age of 25 years, and in most cases the pregnancy appears to be the only obvious cause for the chorea. The onset of the chorea is usually between the first and third months of pregnancy. It is liable to recur with subsequent pregnancies.

Pathology.—The complex nature of the involuntary movements in chorea, and their association with psychical disorders of greater or lesser degree indicate the cerebral cortex as the seat of the pathological process. The essential lesion has proved very difficult of detection by microscopical investigation, but according to Greenfield and Wolfsohn it consists in a diffuse meningo-encephalitis affecting mainly the cerebral cortex, the basal ganglia and the pia-arachnoid.

Symptoms.—The onset is usually gradual, but it is sometimes abrupt,

when emotional disturbance has been the determining cause. The appearance of choreic movements is often preceded by alterations in the mental and physical condition of the child. She becomes nervous and more impressionable than before. She is increasingly unable to apply her attention. She becomes clumsy in her movements—and lets fall objects which she is holding. Anæmia, apathy and languor and irregularity of appetite are commonly present. At this time, careful observation will discover slight involuntary movements of the face and fingers which are often unilateral in distribution. From day to day the movements become more marked and spread to the limbs and trunk. The face is constantly grimacing, and the hands and arms scarcely cease from turning about, and affection of the legs makes the walking irregular and clumsy. The child can no longer keep still, the respiration movements become irregular and spasmodic, and the chorea is fully developed. The characteristic symptoms of a well-marked case of chorea are—(1) involuntary movements; (2) weakness of voluntary movements; (3) ataxy or loss of precision of voluntary movement; (4) emotional instability and other psychic disturbances.

1. THE INVOLUNTARY MOVEMENTS are always irregular as regards time and as regards the nature of the movement. Similar movements are never repeated successively in the same part. Each movement begins rapidly, and ends suddenly, and one frequently sees the involuntary movement complicated by the addition of a voluntary movement to cover the fault. The majority of the movements are complicated, involving several muscles and often more than one joint. In the face, the more simple movements take the form of asymmetrical twitches in the lips, and about the angles of the mouth and orbits. In more severe cases, the strangest grimaces may occur. The tongue is thrust into one cheek, then projected just in time to escape the sudden snap of the open mouth. When asked to show the tongue, the child puts it out rapidly and holds it there by closing the teeth upon it. Smacking of the tongue and palate may often be heard at a distance. Lateral movement of the jaw is common. According to the severity of the case, speech may be difficult, the words being articulated slowly in slurred monosyllables. For the same reason, swallowing may be difficult or impossible in severe cases, and may necessitate nasal feeding. That the ocular muscles participate in the involuntary movements only in very severe cases.

In the upper extremities the movements appear first in the hand. The thumb is more restless than the fingers, which are spread and pressed together, flexed and extended, alternately; the wrists twist about irregularly, the forearms are constantly agitated with movements of pronation and supination, flexion and extension; while all possible movements of the shoulder occur. When the upper extremities are outstretched, the hands assume the position of flexion at the wrist and over-extension at all the finger joints in so many of the cases as to make this a characteristic feature of chorea. The lower extremities are less severely affected than is the rest of the body, and here the movements are best seen when the child is lying down. The gait tends to be clumsy and insecure, and in severe cases walking becomes impossible. Alteration of the rhythm of the respiratory movements is conspicuous and is highly characteristic of chorea. The breath is often taken rapidly and held for some time, then let go with a loud sigh. The trunk is often involved, and movements of a writhing nature are characteristic.

So far as the limbs are concerned, the movements may be confined to one side, more commonly the left side, and the condition is then called hemichorea; but the involvement of the face and trunk is always bilateral and is generally equal upon the two sides. In hemichorea, the movements are always of slight severity. Severe chorea is never confined to one side. Choreic movements cease during sleep, and, except in severe cases, can be controlled more or less by voluntary effort; the attempt to write, for example, will generally cause cessation of the movements in the right arm for the time being. They are generally increased by observation, emotion and self-consciousness, but in a few cases it will be found they are worse when the child is alone and unobserved. The violence of the movements of the limbs may cause the skin over the prominences to ulcerate from friction against the clothing, and the head and limbs may be badly bruised from contact with adjacent objects, and unless the patient be properly protected, wounds may occur, which are liable to infection with such grave consequences as abscess, erysipelas and pyæmia.

2. LOSS OF POWER is shown in the mild cases by incapacity for exertion and undue fatigue. More severe degrees of paresis may accompany or succeed the appearance of the movements. It may be observed that in one limb, or upon one side of the body, the choreic movements are becoming less marked, and that the limbs are becoming progressively weaker. Soon the arm hangs loosely by the side, and the leg is dragged in walking. The degree of choreic paralysis bears no relation to the severity of the movements, for the former may be severe, when the latter are slight and vice versa. Choreic paresis is apt to return with successive attacks of chorea, but not always in the same region.

Limp Choreia (chorea mollis).—This is a more severe degree of choreic paralysis affecting the whole musculature. It may be preceded by the usual symptoms of chorea. More often the paralysis is the first noticeable symptoms, and this develops rapidly in from 24 to 48 hours. The paralysis is characterised by complete flaccidity of the limbs; the child lies upon its back and does not move, and if one of the limbs be raised from the bed and then released, it falls limp and lifeless. The head is no longer held in a natural position, but falls round on to the ear. Careful investigation, however, rarely fails to reveal some slight choreic movements, either in the face or in the fingers. Paretic chorea and chorea mollis run a benign course, and recovery is said to be almost invariable.

3. INCO-ORDINATION OF VOLUNTARY MOVEMENT may be the first symptom of chorea to draw attention, and it may precede the appearance of the choreic movements. It may be very obvious when the movements are slight, and it is most noticeable in those of the hand and forearm, which lack precision, and in those of articulation, deglutition and respiration. The involuntary movements that have been described are superimposed upon voluntary movements which they render inco-ordinate, at times interrupting them abruptly and at other times tending to prolong them.

4. PSYCHICAL DISTURBANCES are common, some degree of emotional instability, failure of attention and depression being present in most cases, and, generally, in proportion to the severity of the affection. Delirium may occur in acute and grave cases. It is usually violent and loquacious, and resembles other forms of toxic delirium, and it is of serious prognostic import.

Mania is quite exceptional in children, but it is not an uncommon complication in adolescents and adults. The form of the aberration may be acute mania, melancholia or delusional insanity. The psychical disorders, slight or severe, usually disappear with the chorea, and in all cases the prognosis as regards permanent mental recovery is good.

OCULAR PHENOMENA.—The pupils are frequently dilated and may be unequal and excentric, and hippus may be present.

Sensibility is not impaired. The sphincters are not affected. The skin reflexes are normal. The deep reflexes are also normal in a large proportion of cases, but often the knee-jerk shows an alteration which is peculiar to chorea. On tapping the patellar tendon, the resulting contraction of the quadriceps is unduly sustained, and the leg remains in a position of extension at the top of its excursion for several tenths of a second. This is not invariably present even in a series of knee-jerks taken successively from a single subject, and the occasional appearance of an apparently sustained jerk is due to the coincidence of reflex contraction and choreiform movement. In severe cases, the deep reflexes may be diminished and rarely may be absent for months.

RHEUMATIC MANIFESTATIONS.—Cardio-vascular changes are common in chorea. In nearly all the cases, careful and repeated examination of the heart will reveal slight dilatation and reduplication of the second sound, often with reduplication of the first sound, and increased rapidity of the pulse. Doubtless these are signs of a slight myocardial weakness, resulting from the rheumatic infection. Irregularity of the pulse is probably dependent upon the altered rhythm of respiration. Systolic murmurs are common, and these may be hæmic in nature, or may be the expression of cardiac dilatation, but in the majority of cases they are indicative of endocarditis. Endocarditis is present in 90 per cent. of the fatal cases. At least one-half of all cases present cardiac murmurs, which are suggestive of the presence of endocarditis, while some cases with no cardiac murmur during life are found post mortem to have endocarditis. The mitral valve is commonly affected, lesions of the aortic valve being quite rare. Pericarditis is a frequent associate of endocarditis; only in rare instances does it occur alone. The valvular affections which are met with in chorea may be the result of antecedent rheumatism, or they may develop in the course of the chorea; or while no signs of endocarditis are present during the attack, the patient may shortly afterwards present the signs of organic valvular disease. Cutaneous affections which occur in rheumatism are met with also in chorea, namely, erythema, purpura and subcutaneous nodules. Acute articular rheumatism is comparatively rare, and when it occurs it is usually accompanied by a cessation of the choreic movements. When rheumatic phenomena are present and in the acute mania of chorea, pyrexia is usually present, but uncomplicated chorea is an apyrexial disease.

RECURRENCE.—One-third of the subjects of chorea have more than one attack. Females are more prone to a recurrence than males in about the same proportion as they are more liable to original attacks. The average interval between the attacks is one year. If, therefore, a patient has remained well for 2 years, it is improbable that a recurrence will take place. The greater the number of choreic attacks, the more likely is the heart to be found affected, and, therefore, cardiac complications are more often met with

in recurrences. In a recurrence of chorea the symptoms are usually less severe and their duration shorter than in the original attack.

Course and Prognosis.—The disease tends to a spontaneous termination after a variable time, which is usually from 6 weeks to 6 months. The duration rarely falls short of the earlier period. The average duration of cases treated in hospital has been found to be 10 weeks. Cases which last for more than 12 months are not rare, and slight cases with remissions may last several years. The course of the malady is that after a gradual development of symptoms, there is a stationary period during which symptoms are well marked, followed by a period of gradual diminution. In some of the more severe cases of chorea where deglutition is difficult the patient is likely to be insufficiently fed; and this constitutes a grave danger, since in the condition of semi-starvation so induced, the chorea develops apace. Articulation and swallowing become impossible, and the movements become ceaseless, so that both rest and sleep become impossible; the patient wastes rapidly, and is in danger of death from exhaustion unless prompt measures for restoring the depleted nutrition are taken. This is the condition known as "chorea gravis."

The proportion of fatal cases occurring in chorea is less than 2 per cent. Death is most often met with in first attacks, occurring about the age of puberty, and it is very uncommon in young children and in recurrences of chorea.

Diagnosis.—The nature of the involuntary movements of chorea is usually so characteristic as to make diagnosis easy, and to avoid any confusion with other maladies which present conspicuous involuntary movements. Nevertheless, occasionally a case of multiple tics in a child does present difficulties, for the movements are not—as is so commonly stated—invariably repetitive. In chorea the involuntary movements may lead to the dropping of objects from the hands. This does not happen in the case of tics. Again, when the choreic subject gives the observer a firm and sustained handclasp, the irregular waxing and waning of the muscular contraction may be felt throughout by the observer. In a case of tics, the contraction is steadily maintained as in the normal subject. In myoclonus, the movements are short and shock-like, while in athetosis they are slow and rhythmic.

Treatment.—It is all-important in the treatment of chorea, from the mildest to the most severe cases, that physical and mental tranquillity should be secured. It is well to commence treatment in every case with several days' absolute rest in bed, provided that such treatment can be carried out without entailing the fretting which enforced imprisonment may produce. A bright room, an interesting companion, and varied amusements during the period of rest, are desirable, and isolation from other children is advantageous. It is, however, better to abandon enforced rest than to allow it to become irksome to the patient, and result in mental depression and emotional upsets—conditions above all things to be avoided.

When absolute rest is considered inadvisable, or after it has been carried out, the ordinary periods of rest should be prolonged. The child should be well clad in woollen garments, especially at night, since the spasmodic movements are liable to leave her uncovered. Improvement in the condition of bodily nutrition is to be aimed at in all cases. Choreic children are mostly

ill-nourished and thin, and the effect of a liberal supply of food and nutritious food upon the course of the disease is striking. When swallowing is difficult, it is best to resort at once to nasal feeding, which rarely causes as much discomfort as the ineffectual and exhausting endeavours to take food with the spoon. A china feeding-cup must never be used, since the spout may be broken off; an enamelled metal cup is safe. It has been pointed out above that chorea gravis is dependent upon a condition of relative starvation, and here nasal feeding should be employed, and the meal should consist of strong beef-tea, Bengel's food, lactose and milk; it should not measure more than three-quarters of a pint for a child of 8 years old. Severe cases, in which the movements are violent, call for skilled attention, and a trained nurse is required night and day. The patient should lie upon a water mattress, placed upon a large guarded bed, the sides of which are everywhere protected by pillows, which must be fixed. When a cot is used, it is easy to pad all the ironwork with cotton-wool, over which bandages are wound. If the limbs are injured, they should be wrapped in cotton-wool applied with a light bandage.

When the patient is improving, measures calculated to enhance control of the limbs, such as exercises under supervision and simple drill, are very useful in hastening the disappearance of the movements. Warm and tepid baths and douches applied regularly and in such a way as to be grateful to the patient, and to produce no fright, are very useful adjuncts. The compounds of salicylic acid are of great value, and of these aspirin is the most useful. It should be given thrice daily after meals in doses of 10 grains for a child between the ages of 6 and 14 years, and 15 grains for an adult, and it should be continued until convalescence is complete. It is well borne and has no deleterious effect, and even more frequent doses may be given. A larger dose given at night is the best remedy for sleeplessness. In very severe cases, the administration of hyoscine is sometimes very useful. It should be given in doses of $\frac{1}{100}$ th grain thrice daily. The administration is followed immediately by wide dilatation of the pupil and slight flushing, and by peaceful sleep. The bromides have little or no value as sedatives. In addition to the above remedies, tonics such as iron, glycerophosphates, hypophosphites, strychnine, cod-liver oil and malt are often valuable, especially during convalescence.

HUNTINGTON'S CHOREA

Synonym.—Hereditary chorea of adults.

This is a somewhat rare disease, in which symptoms almost identical with those of rheumatic chorea, namely, involuntary spontaneous movements, ataxy, paresis and slow and slurring articulation, gradually appear in adult life, and usually about the age of 40 years, and are accompanied by progressive mental failure, with delusions and suicidal tendency. The choreic movements are never severe, but the inco-ordination may be well marked. Maniacal outbursts are not uncommon. The disease always progresses slowly to a fatal termination in from 5 to 30 years, and treatment is entirely unavailing. It is a familial disease, and the transmission is direct from parent to child; but if a generation escape the malady, it seems not to reappear subsequently. Sporadic cases, in which no heredity can be traced,

do, however, occur. The sexes are equally affected. Further than the heredity no causal factors are known. The morbid anatomy consists in a slow progressive degeneration of the nerve-cells of the cerebral cortex and basal ganglia, with consecutive atrophy of the convolution, neuroglial overgrowth and meningeal thickening.

SENILE CHOREA

A malady in which typical choreic movements constitute the chief feature is met with in elderly people, and is possibly due to a progressive neuronc degeneration in that region affected in the other forms of chorea. It differs from Huntington's chorea in the late onset, the absence of heredity, and in the absence of mental changes.

MYOCLONUS

Synonym.—Paramyoclonus multiplex.

The characteristic symptom of this condition is the occurrence of sudden shock-like contractions of the muscles, which may vary in intensity from simple fibrillary twitching to contraction which causes a violent movement of a limb. The movements are often symmetrical, and are especially incident in the proximal muscles of the limbs.

Ætiology.—The malady appears in children usually between the ages of 5 and 15 years, while in adults it commences between the ages of 25 and 40 years. Both sexes are liable to the affection. Many instances, in which several children of the same parents have been affected, have been recorded, and in a few the malady has been transmitted through several generations. Nothing further is known as to the causation.

It is probable that the seat of the morbid process is in the cells of the cerebral hemispheres, since myoclonus is further associated with epilepsy and with progressive mental failure.

Symptoms.—The movements of myoclonus are simple sudden movements, and may exactly resemble the movement resulting from a single faradic stimulus. Each movement commonly involves a single muscle only, and it may concern no more than a few fibres, resembling then the fibrillary twitching common in progressive muscular atrophy. In other cases, many muscles may be implicated in the shock-like spasms, which may be of so violent a nature as to throw the patient to the ground. The distribution of the contraction is never determined by that of the nerve supply, nor do the muscles contract according to their synergic association. Myoclonic movements are irregular as regards rhythm and range of successive movements. The upper limbs are more affected than the lower, and the proximal parts more than the distal, while the periphery, the hand and foot, often escape. Voluntary muscular effort usually checks the myoclonic movements, but in rare instances it excites or augments the spasm. The electrical excitability of the muscles is unaltered, and there is no muscular wasting, but the mechanical excitability of the muscles is increased, and percussion of a muscle may evoke the spasms. The sphincters are unaffected. The reflexes, both superficial and deep, are normal. Sensory phenomena are

absent. Speech may be seriously interfered with when the muscles of jaw, tongue, palate and larynx are implicated, and spontaneous laryngeal and pharyngeal noises may occur. The ocular muscles seem never to be the seat of the movements. Epileptiform convulsions are present in some cases, and for these the term "epileptic myoclonus" has been used.

Diagnosis.—This is not difficult when the simple shock-like movements in symmetrical muscles, without any resemblance to volitional movements and entirely destitute of rhythm, occur in this disease alone.

Course, Duration and Prognosis.—Myoclonus, as a rule, is a slowly progressive affection up to a certain stage, and when this is reached it may remain stationary for years, having little tendency to shorten life, death ultimately occurring from some other disease, without any period of freedom from the spasms. Rarely the disease has ended fatally within a few months of the onset, with progressive mental failure and coma.

Recovery may take place spontaneously, or as a result of treatment, but the affection is very prone to recur.

Treatment.—Every available measure should be used to improve the general bodily condition so as to bring about a more stable condition of the nervous elements, by improving their nutrition. The only drug which influences the disease is arsenic. It must be borne in mind that the malady is an intractable one in proportion to the time the symptoms have persisted, and that some cases recover spontaneously.

SPASMODIC TORTICOLLIS

Definition.—A disease of the nervous system, characterised by tonic and clonic contraction of the superficial and deep muscles of the neck, causing the head to assume either a position in which it is turned to one side and upwards, or a position of marked retraction (retrocollic spasm). It is more correctly to be regarded as a disturbance of movements than of muscles, and perhaps, physiologically considered, it may be spoken of as a disorder in the carriage of the head. This carriage is a more complex and highly co-ordinated function in the erect posture than in the quadrupedal posture; it is a function peculiar to man, and in this sense is of recent evolutionary development. We may perhaps see in this a factor determining its frequent derangement, as in spasmodic torticollis.

Ætiology.—The disease is most frequently met with in middle-aged adults, but it may occur at any age from puberty onwards. It is twice as frequent in females as in males. The causation is most obscure. Not infrequently neuropathic heredity, such as epilepsy and insanity, exists, and the patients are often of highly-strung, nervous, irritable dispositions. Nervous shock, prolonged anxiety, and general ill-health have frequently preceded the onset of symptoms. Less often local strain, or injury and exposure to cold, have been the presumably exciting causes. In a few cases it appears to develop from an occupation neurosis; it developed, for instance, in a tailor who in drawing each stitch had the habit of making a short jerking movement of the head to one side. It occasionally occurs as a symptom of hysteria; but such cases should be carefully separated from those in which there is no hysterical manifestation, as being more susceptible

to treatment and having no tendency to recur when once cured. A torticollis movement may occur as a variety of tic. In one case under our care typical torticollis was the end-result of lethargic encephalitis.

Pathology.—No morbid anatomical changes have been found. On account of the involvement of several muscles, effecting special movements, in this disease (as is well instanced by the over-action of the frontalis in retro collic spasm, for retraction of the head is always normally associated with raising of the eyebrows in the act of looking up), it is probable that torticollis is due to disorder of those centres which direct such associated movements of the affected muscles.

Symptoms.—The onset is usually insidious, but in rare cases may be quite sudden, as in the case of a man aged 40 years, who, when walking along a London street, suddenly turned his head at the sound of an accident which shocked him severely; he was unable to turn his head back without using his hands to do so, and he subsequently developed the most severe torticollis. The initial symptom is always spasm, which may be either tonic or clonic, and frequently both forms of spasm are combined in the same case. In the tonic form, the head is retracted and the face turned to one side, usually the left, and owing to the retraction of the head the face is turned upwards. The shoulder on the side to which the head is inclined is usually raised. In severe cases all the muscles of the upper extremity, the scaleni and the face muscles, may become involved. The spasm, except in the earliest stages, always involved muscles of both sides of the neck. Where the bilateral involvement is general and equal, the rotation of the head does not occur, but it becomes strongly retracted, and the condition is then known as retrocollic spasm. Such retrocollic spasm is always accompanied by marked over-action of the frontales, the skin of the forehead being thrown into transverse wrinkles. In the clonic variety there is jerking movement of the same muscles, usually associated with some degree of tonic spasm. The eyes do not follow the movements of the head in the jerking. The muscle primarily involved is the sterno-mastoid, the action of which is to incline the head forwards and towards the shoulder of the same side, and rotate the face to the opposite side. The next muscle involved is the splenius of the opposite side, which inclines the head backwards and rotates the face towards its own side, its rotatory action thus coinciding with that of the opposite sterno-mastoid. When the splenii of both sides act together, the head is strongly retracted. Next to be affected are the upper part of the trapezii, the trachelo-mastoids and other deep neck muscles, and with further spread of the spasm, any neighbouring muscles of the shoulder and upper extremity may be affected. Sleep causes cessation of the clonic spasm, but not always of the tonic spasm when the case is severe. The spasm is always increased by fatigue and excitement. There is no wasting of the muscles involved, but on the other hand, they may be even hypertrophied if the spasm has existed for long, and their electrical excitability may be increased. The amount of pain associated with the spasm varies greatly. There may be a slight feeling of cramp only, but usually there is a great deal of aching pain, which may radiate down the arm and into the side of the head, and make life unbearable to the patient. More rarely, sharp neuralgic pains are present.

The course of the disease, which has no tendency to shorten life, is chronic

exacerbations and remissions under treatment being common, and recurrence, after temporary cure, frequent.

Diagnosis.—This is usually quite simple. Fixed positions of the head associated with spasm occur in disease of the cervical spine, especially in spinal caries, and are also associated with enlarged lymphatic glands in the neck. The local signs of these conditions, however, are characteristic.

Treatment.—Spasmodic torticollis is a most intractable condition, and in many cases temporary alleviation is all that can be secured. It is usually best to begin treatment by rest in bed, the patient lying supine with the head low and between sandbags or pillows. The regular administration of phenobarbitone, of chlorbutol, or of chloral and bromide may then be tried. Many years ago Bastian claimed good and permanent results from a continuous narcosis lasting 3 weeks and induced by chloral hydrate. Probably a combination of rest as above described, together with massage and resistance exercises is the most useful line of treatment. In some cases the application of a plaster mould, fixing head and shoulders, and worn for one or more months, or a more easily removed and lighter metal splint will give complete respite from muscular spasm while it is worn, and very occasionally permanent respite after removal. In severe and disabling cases this is well worth trial. Surgical measures (tenotomy, excision of the sternomastoid, posterior root section) have all proved disappointing and are not to be recommended.

There is a *congenital form of torticollis* which is of a very different nature. The disease is prenatal and analogous to congenital talipes, the sternomastoid alone is affected, and nearly always that of the right side. Such a muscle is frequently ruptured during birth, and this has given rise to the opinion that the birth injury and subsequent hæmatoma of the muscle were responsible for the torticollis. In many of these cases there is marked facial asymmetry, the face being smaller on the side of the affected sterno-mastoid. This association points strongly to some defect in the nerve centres of the medulla.

Treatment.—This consists in tenotomy of the contracted muscle.

THE TICS

Synonym.—Habit spasm.

Definition.—A group of maladies characterised by the occurrence of either—(1) sudden, rapid, twitch-like, involuntary co-ordinated movements, always of the same nature and in the same region; or of (2) sudden psychical phenomena, imperative ideas and explosive utterances; or (3) of a train of deliberate highly co-ordinated actions produced by an imperative idea. Any combination of these phenomena may occur.

The tics are both ætiologically and clinically related to spasmodic torticollis, into which some of the motor tics graduate. A torticollis movement may occur as a tic, and it may in rare cases pass over into an established torticollis.

The tics may be conveniently divided for clinical purposes into the following groups, between which any combinations may occur:

1. The clinical picture is made up by the occurrence of sudden twitch-like co-ordinated movements, which resemble reflex or defence movements. The

movement is always of the same nature and occurs in the same region, though several different tics may occur in the same patient. The usual region affected is the face, with the pharynx and larynx, the neck and upper extremity. This form occurs chiefly in children, and usually runs a favourable course—Simple Tic.

2. The spasms are more severe and complicated than in simple tic, and imperative ideas and explosive utterances are common and important symptoms. The condition is met with soon after puberty, and more commonly in males—Convulsive Tic.

3. There is no spasm or other motor manifestation, but the psychic tic is expressed by uncontrollable imperative ideas, explosive utterances, arithmomania, etc.—Psychical Tic.

4. Under conditions of mental stress and embarrassment, and in conditions of boredom, the patient performs some highly complicated and co-ordinated act which relieves his nervous tension and fascinates him—Co-ordinated Tic.

The tics are expressions of unrest and of physiological embarrassment in consciousness in a nervous system which is highly sensitive and not too stable. There is always the desire to relieve the embarrassment by the occurrence of the tic, and a feeling of relief when it has occurred, coupled often with disappointment at the failure of its suppression.

While the more simple forms of motor tic from their pattern suggest strongly that they are caused by some peripheral irritation from the conjunctiva in the case of a blinking tic, from the nose in a case of snuffling tic, and from the larynx in a case of laryngeal tic, and that constant irritation from these regions has set up a habit, yet it cannot be too strongly pointed out that no such peripheral irritation precedes the onset of tic, for the irritation and cause come from within the nervous system. Severe peripheral irritation does not cause tic, nor does the correction of errors of refraction, the removal of tonsils and adenoids and of teeth, or circumcision aid in the cure of the malady, though it is only too common to see cases in which these procedures have been inflicted upon the tiqueur one after the other, to the detriment of his tic and of his general health.

1. SIMPLE TIC

Synonym.—Habit spasm.

This is a common disorder of late childhood, the majority of the cases occurring between the fifth and the tenth year. Either sex is prone to the disease. The onset may be preceded by deterioration of health from any cause, and sometimes fright and emotion bring on the tic. Often the malady arises in perfectly healthy children without assignable cause. The children are usually highly strung and intelligent. It is a rare event to see a dull and backward child with a tic.

Symptoms.—The recurring tic appears somewhat suddenly, and may reach its height in a few days. The movements are of the nature of a simple *spasm*. They occur suddenly and without warning, and are executed rapidly. Usually the movement is of one kind only; but sometimes several movements coexist. The common site of the spasm is the head, face and neck. Blinking, winking, alternate elevation and depression of the eyebrows, side to

side movements of the mouth, tossing the chin in the air, sudden movements of the tongue, palate or larynx, accompanied by an unpleasant fidgeting sound, are of frequent occurrence, while any movement of the head upon the shoulder, torticollic movements, shrugging of the shoulder, and any movements of the arm may be met with. Respiratory movements are often associated with those occurring in the tongue and larynx. Tic affecting the legs is much less common. The movements cease during sleep. Generally a variable time of some length separates the individual movements, but in severe cases these may follow one another almost unceasingly. They are increased by excitement and by observation, and can usually be controlled by the will, but only for a limited time.

Diagnosis.—The movement of tic is so peculiar that it cannot be confused with any other spontaneous, involuntary movement. It is the same movement, repeated with very rapid execution, in the same place. It is short and sharp, like a twitch. In chorea the movements are slow compared with those of tic, and are irregular in nature, in time and in place.

Prognosis.—Most cases of simple tic recover, whether they are treated or not. They recover much more quickly under treatment, and two or three months suffices in most cases to see the end of them. The longer a tic lasts, the more difficult it is to cure. In the rarest cases only does a tic of this nature persist or merge into one of the more severe forms.

Treatment.—A scrutiny of the general health should be made, and any defects attended to. Matters of hygiene, diet, education, exercise and pleasure should be correct and normal. Observation and remarks upon the child's defects, and anything tending to increase self-consciousness should be avoided. The confidence of the child should be gained if possible, and any source of mental worry, or grief, or annoyance should be ascertained and comforted. Restraint and discipline should be kindly taught, and an orderly life followed in which the child is happy, and in which his time is fully and congenially employed. In severe cases only is it necessary to interdict all physical and mental exertion and excitement, and enjoin rest in bed, and these measures should only be employed for a short time. Aspirin in 10-grain doses 3 times a day is a most valuable remedy, hardly to be dispensed with in any case. Tonics are often useful.

2. CONVULSIVE TIC

In this malady, which was first described by Gilles de la Tourette, and which bears his name, the same movements as are met with in simple tic occur; but these are more severe and more widely spread, and they may involve the whole body in spasm at one time. In addition, there are psychic tics, which cause irresistible impulses, among which are explosive utterances, repetition of words, sounds and gestures, and also imperative ideas.

Ætiology.—The stigmata of physical and mental degeneracy are rarely absent, neuropathic and sometimes direct heredity is often present. The malady is said to be more common in males, and is met with more often in France than in England—where it is a rare disease. The symptoms appear usually between the ages of 10 and 15 years, and commonly follow physical or mental shocks or acute illness of any kind.

Symptoms.—The spasmodic movements resemble at first those of simple tic in their nature and rapidity, and favour the same sites ; but they are not restricted to the repetition of the same movement, but successive movements may vary widely in position and extent and sometimes involve the whole musculature of the body. The great variety of facial grimaces, head jerking, grotesque attitudes and ridiculous pantomime which may occur in this affection lead commonly to the belief that the patient is shamming. The tic is not continual as in the simple form. It occurs in the form of bouts in which the same pantomime is reproduced. These are often excited by observation and emotion. They can often be controlled, but with much fatiguing effort on the part of the patient, who becomes so worn out with half successful efforts to control them that he ceases to make the attempt. Between the attacks the patient seems quite normal. The psychic phenomena are the same as in psychical tic, about to be described, and the treatment of the two conditions is identical.

3. PSYCHICAL TIC

In this condition there is no muscular spasm ; but the sudden event takes the form of explosive utterances, imperative ideas and impulsive acts. This condition often occurs as a part of convulsive tic. The exclamatory tic consists of some sound or word or group of either, which is habitually uttered, with complete irrelevancy of time, place or sense. Sometimes the words are of an obscene nature and cause the greatest distress to the patient, who, often of innocent mind, is never safe from putting himself to shame. The utterances may be single, or may be repeated over and over in rapid succession. Echolalia, which is an uncontrollable impulse to repeat sounds heard, or to repeat words which the patient or others have just spoken, may be met with. The great characteristic of the condition is that though the patient desires above all other things to prevent their occurrence he cannot do so by any effort of will. Other symptoms that are commonly met with in this condition are imperative ideas and impulsive acts of all sorts, and in addition insanity of doubt, agoraphobia, acrophobia, mysophobia, etc., and arithmomania. In severe cases grave signs of mental deterioration slowly supervene, judgment and memory fail, will power and attention are lost, and the patient becomes incoherent and insane.

Diagnosis.—Both in the convulsive and psychical tics the diagnosis is placed beyond doubt, both by the nature of the movements and by the peculiarity of the psychic disturbance.

Prognosis.—Permanent recovery has occurred from both these conditions ; but such an event is rare. Most of the cases follow a downward course despite treatment, and many end in suicide or insanity.

Treatment.—General tonic treatment, with change of circumstance and kindly moral and physical discipline, with healthy pursuits and congenial intellectual and physical occupation are the most likely to benefit. When fixed and imperative ideas are present the patient must be guarded, as one of unsound mind.

4. CO-ORDINATED TIC

In this condition complicated co-ordinated movements are habitually repeated without apparent cause or purpose—especially in conditions of

mental stress. It can be best illustrated by the account of an individual case under our care : A brilliant scholar at a public school was noticed to absent himself for no apparent reason, and when sought for, on the occasions on which he could be found, was always discovered in some secluded place rapidly revolving the two index fingers round the inside of a loop of string. He explained to me that this act had always given him relief from mental stress and anxiety, and that he was ashamed of it, and did his best to overcome it, and often succeeded, but that sometimes the desire for relief from stress overcame him. He was treated, and had no return of the tic for some years. He had become head boy of the school and captain of the football team, and on the occasion of the match of the year with another great school with much anxiety and responsibility upon his shoulders, at the moment of the commencement of the match the captain was not to be found. He was recovered from the act of revolving his index fingers round the inside of a loop of string in a secluded place, in time to perform brilliantly and win for his side.

CRAFT PALSY

Synonyms.—Occupation Palsy ; Occupation Cramp.

Definition.—A peculiar malady determined by the habitual use of one set of muscles for the constant repetition of an act of short range, to the exclusion of acts of wider range and acts involving a different set of muscles. The symptoms are: (1) local pain in the muscles concerned ; (2) local spasm of the muscles ; (3) loss of volitional control of the range and nature of the movements ; and (4) weakness of the movements. These symptoms may occur separately or together.

Ætiology.—This disease may be occasioned by any occupation which requires the constant repetition of movements of small range, and which necessitates the holding of the limb rigidly for the fine co-ordination, to the exclusion of free and wide range movements. Consequently it is almost confined to those employments involving finely co-ordinated movements of the fingers, hand and upper limb. As far as is known, a neuropathic inheritance is neither an essential nor a common factor in the production of this disability. Yet at the same time, the fact that young persons are apt to break down while in training, while others follow the occupation of scrivener or telegraphist for years with impunity, it has been suggested that some inherent weakness in the nervous system may underlie the development of the cramp. But, however, it is not necessary to invoke these obscure factors, when faulty motor habits are known to play an important rôle and are adequate to account for the disability.

In the case of writing there are two motor components, pen holding and pen moving. The former is of no importance in the production of writer's cramp, but defects in the latter are of primary importance. The pen may be moved solely by the muscles which hold it, the hand being fixed and the digits alone moving. Less faulty is the habit when writing is done largely by movement at the wrist. Best is the method in which writing is a free movement involving all the limb muscles. Of note in this connection is the fact that subjects of writer's cramp may be able when standing to write on a blackboard. It is those who confine the muscular action most rigidly to

the small hand muscles and the flexors of the index and middle fingers who are most likely to develop cramp. In the use of the Morse key such restriction of activity to small muscles groups is more or less inevitable, but the more modern machines which do not involve this, and have come into greater use, have not this fault and thus telegraphist's cramp is less common now than formerly.

It has been reported that focal lesions in the subthalamie region have resulted in a disability resembling writer's cramp, but the observation is not necessarily relevant to the ordinary example of this condition.

The following is a list of some of the occupations in order of frequency in which this malady occurs: writers with the pen, telegraphists, seamstresses, violinists, machinists, cigar and cigarette rollers, dairy milkers, pianists and typists. It has been said that shorthand writers never suffer from this complaint. In most instances the disability in the use of the hand is confined to the particular movement which first gave rise to it, but in some severe cases other movements may ultimately come to be difficult: *e.g.* shaving, sewing, knitting. It should be borne in mind, however, that when at an early stage of what seems to be writer's or telegraphist's cramp other movements become deranged the presence of some organic nervous affection should be suspected. This point is further dealt with under the heading of diagnosis. The very general use of the typewriting machine, with its free and multitudinous movements required for its manipulation, has made writer's cramp a very rare disease compared with years ago; but it must be borne in mind that when the malady is once installed it is likely to follow the sufferer from one occupation to another. As an example, a telegraphist developed cramp in manipulating the Baudot instrument. He was rested and transferred to lighter duties involving the use of the Morse instrument, where he after a time failed. He was then transferred to counter duties, involving the use of the pen, and became incapacitated with writer's cramp. He was then used to close envelopes, and developed cramp over that act, and ended his service as a messenger. Several of the occupations above mentioned are scheduled in an Act of Parliament as dangerous trades, owing to the liability to cramp, and the employer is bound to compensate for such disability arising in his employ.

Symptoms.—These are of two orders, namely: subjective, consisting of discomfort, pain and the sense of fatigue; and objective, comprising muscular spasm and the abnormalities of movement arising from it and from the effort to avoid both pain and spasm. In some subjects pain, in others spasm predominates.

The onset is gradual, the movements of the pen become inexplicably difficult and tend to be irregular, the strokes extending too high or too low. The subject then finds himself grasping the pen with excessive force, and the correct adjustment of the finger ends becomes hard and apt to fail, the index slipping off the penholder. This he tries to correct by a still firmer grasp. The hand then begins to ache, and feels heavy and tired. With the passage of time all these symptoms increase, and the writing becomes more irregular and the nib is driven more firmly into the paper which it penetrates, the ink spluttering over the sheet. Some tremor may develop in the limb. As the condition grows worse, the cramp appears more and more readily when writing is started, so that even taking the pen in the hand

may evoke cramp. At the same time, other fine and repetitive movements of the hand may be performed with normal ease and facility. The pain which in varying degree accompanies the cramp tends as the affection grows worse to spread from the small hand muscles up the limb until the whole arm and shoulder ache.

TELEGRAPHIST'S CRAMP

There are four instruments commonly used for sending. The Morse instrument is a hard contact key provided with a spring and knob, and the manipulation is performed by making the contact against the spring pressure with the knob held loosely in the palm of the hand. The movements involved are flexion and extension of the wrist only. The arm is supported from the shoulder only. This is a bad cramp-producing instrument, but it has the advantage that it can be manipulated with either hand. If telegraphists are taught to be ambidextrous from the first the incidence of cramp falls to a very low level.

The Baudot instrument consists of a piano-board with five keys, which are manipulated by three fingers of one hand and two fingers of the other, and with the permutations and combinations of these five keys all the signals are made. The movements have to be synchronised to the beats of the commutator. The forearms and wrists rest upon the table. This is the worst cramp-producing instrument that ingenuity could devise, for the movement is of the fingers only, and it is utterly restricted both in space and in time. The Hughes instrument is a piano keyboard with many more keys, and its use is not often productive of cramp. The Gell instrument is a typewriter keyboard, and the movements are free. It is the best instrument in so far as it is not a producer of cramp. Two points of interest stand out, especially in connection with cramp in telegraphy. The one is that, notwithstanding the discomfort, spasm and obvious disability, the subject is rarely reported for faulty sending, so great is his aptitude for dodging his disability. The second point is that so many subjects will work for years, and often to the end of their service, with very obvious cramp, which never proceeds to incapacity. On the whole, the incidence of telegraphist's cramp is now small.

Diagnosis.—From what has been said of the character of the symptoms in these forms of cramp, of the mode of their production by a particular movement-complex, and of their occurrence in the absence of signs of organic nervous disease, it seems reasonable to state that errors of diagnosis should not occur.

Nevertheless, errors are not infrequent and consist in the diagnosing of writer's or of telegraphist's cramp when in fact some organic affection is present. Paralysis agitans, with little or no tremor, and post-encephalitic Parkinsonism provide fruitful sources of error. In the clinical picture thus presented, the initial symptoms may involve the right arm and hand, and at first consist in a difficulty in the normally rapid and free performance of fine movements. Not unnaturally the handwriting may be affected early. It becomes slow in performance, spidery and progressively smaller, and the effort to continue writing may be irksome and even painful. The total clinical picture in such a case is made up of such small deviations from the normal that the inexperienced or careless observer may miss them and may note no more than the patient himself has noted ; namely, that it has become

difficult and uncomfortable to write. Amongst other organic conditions which may be encountered under the erroneous diagnosis of writer's or telegraphist's cramp may be included cervical rib, any organic nervous affection which impairs fine hand movements, arthritis, and painful affections of muscles. The general principle which underlies accuracy of diagnosis here as elsewhere is careful and systematic clinical examination.

Course and Prognosis.—In a young subject, who shows signs of the malady during training or soon thereafter, the outlook is hopeless with regard to continuance of the occupation, and the progress is from bad to worse. In older subjects the course varies greatly. Some cases recover completely and permanently, even though they continue with the occupation. In others—and this class is much larger than is usually supposed—the condition of cramp becomes stationary, and persists though not in disabling fashion. In a third and numerous group it progresses to incapacity, and tends to reappear with every change of occupation. In a few cases the patients become incapacitated for all the finer movements of both hands. The prognosis is usually serious; but a correct forecast can only be made from the history and progress of each individual case.

Treatment.—The responsibility and costliness which the Compensation Act entails upon employers are slowly enough but surely leading to the abandonment of those instruments, the manipulation of which may produce cramp. Good teaching of unconstrained methods of manipulation and encouragement of ambidexterity in all the occupations concerned are important prophylactic measures. Long hours and the speeding-up of work should be avoided. After long absence from work, the work should be gradually resumed and not recommenced at full pressure. When the malady appears, rest and change of work afterwards are absolutely essential. Long-continued rest, be it remembered, cuts both ways for, as has been pointed out above, resumption after long rest is actually a cause of cramp, for long unemployment decreases the stability and the aptitude of the mechanism. General hygienic and tonic treatment are important. It is doubtful whether local treatment, in the way of massage, electricity, etc., can do any good, except to satisfy the patient. Sporting exercise of any and every kind is most useful. The Post Office authorities adopt the very admirable plan of re-training cramp subjects by daily practice with the instruments for a few minutes, the time of practice being gradually lengthened as capacity increases.

CRAFT ATROPHIES

Under this title are described a medley of conditions in which local atrophy of muscles, pain, numbness and sensory loss occur in connection with regions which are habitually over-exerted. These conditions have been met with in platers, filers, file-makers, locksmiths, rowers, glassworkers, etc., and seem to be really examples of local traumatic fibrositis, involving the nerves, and produced in some cases by the continued pressure of the tools.

Many of the cases recover with rest and treatment appropriate for a local interstitial neuritis.

JAMES COLLIER.

W. J. ADIE.

Revised by F. M. R. WALSHIE.

COMPRESSION OF THE SPINAL CORD

In compression the lumen of the spinal canal is reduced in a small part of its vertical extent, and the spinal cord is injured at this point, either directly by pressure, or indirectly by interference with its vascular supply. With the exception of acute inflammation of the membranes, all the extramedullary lesions of the spinal cord come under this heading. The characteristic clinical feature of compression is the combination of two sets of phenomena: local or root symptoms in those regions supplied by the roots arising from the cord at the level of the lesion, and remote or cord symptoms due to interruption of the conducting paths in the white matter. It is convenient to divide this subject into two parts—slow compression and compression of rapid onset.

1. SLOW COMPRESSION

The commonest causes are tuberculous spinal caries, vertebral tumours, meningeal tumours and cysts; rarer causes are aneurysm, gumma, leukaemia, Hodgkin's disease, Paget's disease, syphilitic caries, spondylitis deformans, and other chronic inflammations of the bones and joints of the spine.

Ætiology and Pathology.—1. *Tuberculous spinal caries (Pott's disease).*—Spinal caries is the most frequent cause of slow compression. It occurs most often in children, but is common in adults, and may begin late in life. Signs of injury to the cord develop in about 1 case in 20, and are usually preceded by obvious deformity of the spine; but in many cases they appear before disease of the bone is suspected. Rarely paralysis comes on for the first time in an adult who has had a curvature since childhood.

The cord may be damaged by direct pressure of displaced bone, or by an abscess beneath the periosteum of the diseased vertebræ; but in almost all cases the injury is indirect, and results from œdema of the cord, arising from interference with its blood supply by tuberculous granulation tissue, which forms on the outer surface of the dura mater and fills the epidural space (pachymeningitis externa). The functions of the cord may be temporarily deranged for long periods by this œdema, without permanent damage to the nervous tissues; hence, when the disease is cured, the œdema subsides and the cord recovers. In cases of greater severity necrosis of the nervous structures follow thrombosis of the vessels, or prolonged pressure causes atrophy of nerve roots, and complete recovery is impossible.

2. *Tumours of the vertebral column.*—Vertebral tumours are about twice as common as all the other forms of extramedullary tumours together, and almost all of them are malignant. Carcinoma is always secondary, and is a frequent and distressing complication of cancer elsewhere. A very small primary carcinoma, e.g. of the breast, thyroid or prostate, may produce extensive disease of the vertebræ, and signs of compression may appear before the existence of the primary growth is suspected. On the other hand, they may appear several years after complete removal of the primary growth, and may be the first evidence of a recurrence. Sarcoma, the commonest form of primary growth, begins in the bone or periosteum of the bodies or laminae, often in several at once, or simultaneously at different levels. Secondary

sarcoma arises by metastasis from sarcoma elsewhere, or by direct extension from a growth in neighbouring soft parts, *e.g.* of tumours in the mediastinal and retro-peritoneal spaces.

The growth of vertebral tumours is usually rapid, and extensive portions of the spinal column may be completely destroyed. The cord is compressed by the growth itself, by displaced bone, or by a process of the growth which invades the spinal canal through an intervertebral foramen. As a rule, the dura mater sets bounds to its inward extension. Benign tumours of the spine are rare. They usually grow forwards, but occasionally an osteoma, a chondroma, or an exostosis produces signs of compression.

3. *Meningeal tumours.*—These are divided into two groups—intradural and extradural. The first are twice as common as the latter, and more than half of them are simple, encapsuled, and easily removable. Myxoma, fibroma, sarcoma, endothelioma, and psammoma are common. Other forms are rare. In most cases they lie posterior or postero-lateral to the cord, and are seen when the cord is exposed by laminectomy; but in a few cases they lie in front and may escape detection.

Sarcoma is the commonest extradural tumour. It is sometimes encapsuled, but more often it is a diffuse growth difficult to remove.

These tumours usually grow very slowly, and several years may elapse between the onset of the first symptom and the time when an accurate diagnosis can be made. They do not invade the substance of the cord, nor penetrate the dura, nor give rise to metastases.

4. *Cysts.*—Cysts, parasitic and non-parasitic, may compress the cord and produce symptoms indistinguishable from those of solid tumours. In some countries hydatid cysts form a high proportion of all spinal tumours. They may invade the spinal canal from adjacent soft parts or from the vertebræ, or arise primarily in the membranes. They are often multiple, and are nearly always extradural. Cysticercus cysts, which are very rare, are usually single and intradural. Non-parasitic cysts are collections of fluid contained within slightly thickened adherent membranes. They are among the most frequent of spinal tumours. They are most likely a result of circumscribed inflammation of the pia-arachnoid, and have been known to follow an injury; but their ætiology is obscure. The cyst is often opened inadvertently during operation, and the only evidence of its former presence is flattening of the cord and atrophy of the nerve roots at the point where a solid tumour was expected. Sometimes the position of the cyst can be inferred at operation, from the absence of normal pulsation below it. On puncturing the membranes in this position fluid escapes under pressure, and the pulsations reappear. The name *meningitis serosa circumscripta* is applied to this condition.

5. *Aneurysm of the aorta* is a rare but well-known cause of spinal compression. The dorsal region is most often affected, three or four vertebral bodies being slowly eroded until the dura mater is exposed. Rupture into the spinal canal has been observed. Berry aneurysms upon the surface vessels of the cord occurring in connection with occult coarctation of the aorta have been many times recorded. The pathognomonic sign of this cause of compression paraplegia is the high arterial blood pressure in the arm as contrasted with the low pressure in the leg.

6. *Syphilitic caries.*—Gummata and caseous masses of syphilitic origin

in the bones of the spine may cause a condition not unlike that of tuberculous caries. It is very rare, and usually occurs in the cervical region where the cord may be compressed.

EXTRADURAL COMPRESSION

Symptoms.—*Local or root symptoms.*—Pain in parts supplied by the sensory roots arising from the cord at the level of the lesion is often the first symptom. It may be a dull ache, a feeling of constriction, a sharp cutting pain, or pain so severe as to be almost unbearable. It is often brought on or greatly increased by movement of the spine or by coughing. The skin in the painful area is sometimes hypersensitive at first, but very soon its sensibility is diminished, while the pains persist (*anæsthesia dolorosa*). The nerve trunks are not tender as in ordinary neuralgia. Severe pains are rarely absent in cases of vertebral tumour. In spinal caries they are usually absent or slight. Injury to the motor cells or anterior roots leads to weakness, wasting and loss of tone in the corresponding muscles. In some cases root symptoms are absent throughout the course of the disease, and the first effects of compression are referable to interruption of the conducting paths in the cord.

Remote or cord symptoms.—Although all the tracts are submitted to the same degree of compression, their functions are not impaired at the same time. While variations are common, the symptoms usually arise in the following order: first weakness and spasticity in the lower limbs, then impairment of sensation, position and passive movement, temperature, pain and touch being affected in this order. Defective sphincter control often precedes and sometimes follows sensory loss.

Motor symptoms.—Interruption of the pyramidal tracts produces spastic paraplegia in parts below the lesion. The clinical features are—(1) diminution of voluntary power; (2) alterations in the amount and distribution of muscle tone and in the attitude of the limbs; (3) changes in the tendon and skin reflexes; (4) the occurrence of certain involuntary and reflex movements.

The phenomena of spastic paraplegia have been analysed by Walshe as follows:—It is essential to remember that the muscles of the lower limb are divided into two distinct groups—the flexors and the extensors—and that the muscles which dorsiflex the foot and toes are physiologically flexors, while the corresponding plantar flexors are extensors. In all that follows these important muscles will be grouped according to this nomenclature.

1. Loss of voluntary power varies from slight weakness of one group of muscles to complete paralysis of both limbs, and depends on the degree of damage to the pyramidal tracts. It usually begins in the distal segments of the limb, and is greater in the flexors than in the extensors. Dorsiflexion is the earliest and remains the most severely impaired movement.

2. The tone in all the muscles increases early, and is greatest in the extensors. Hence an early symptom is generally stiffness of the limbs, especially a difficulty in flexing them. If the limbs are handled passively, the resistance to flexion is found to be greater than to extension. As power diminishes spasticity increases, until at length the limbs are held constantly in an attitude of complete extension. This combination of weakness and spasticity with extended lower limbs is known as “paraplegia in extension.”

3. Exaggeration of the tendon reflexes is a constant early sign. The abdominal reflexes below the level of the lesion and the cremasteric reflexes are lost early. The normal plantar reflex is also lost, and is replaced by a different kind of reflex—Babinski's sign, the "extensor" plantar response.

As the damage to the cord increases, and when certain extra-pyramidal motor tracts are affected, the extensor muscles gradually lose their tone, for which connections with the brain-stem through these extra-pyramidal tracts are essential, while the tone in the flexor muscles, which depends on a reflex arc which is purely spinal, is retained. The result is that the knee- and ankle-jerks, which indicate tone in extensor muscles, are lost while the reflexes from flexor muscles (hamstring-jerks) persist. At the same time, in some cases, the limbs are gradually drawn up by the unopposed action of the flexors. This combination of weakness and spasticity with flexed lower limbs is known as "paraplegia in flexion." At first the flexed position is occasional—flexor spasms—later it becomes constant, but is still due entirely to excess of tone in the flexors. Finally, contractures occur in the muscles, and the deformity becomes permanent. In many cases of compression the stage of paraplegia in extension gradually merges into one of complete flaccidity of all the muscles, without the occurrence of paraplegia in flexion, and all the tendon reflexes are lost.

4. While the limbs are still rigid in extension, the commonest involuntary movement is a spontaneous clonus of the extensor muscles, in which the whole limb trembles as it does when ankle clonus is elicited in a case with marked spasticity. In the later stages, where the extensor muscles are beginning to lose their tone, a new kind of movement appears, in which the limbs are drawn up suddenly from time to time by an involuntary contraction of the flexor muscles—flexor spasms. Further, by appropriate stimulation may reflex movements can be produced in the paralysed limbs. The most important of these is the "flexor reflex of the lower limb." It is elicited most easily by stimulating the outer border of the sole by firm pressure or a pin-prick, and in its complete form consists in flexion of the hip and knee, dorsiflexion of the foot, and an upward movement—so-called extension but physiological flexion—of the great toe. When the damage to the motor tracts is slight, when the limbs are rigid in extension and the movement of flexion is prevented by the hypertonus of the extensors, or when almost all reflex activity has disappeared, the reflex appears in its minimal form. A part of this minimal response is an "extension" of the great toe. The normal plantar response is obtained from the sole alone. The pathological reflex, of which the "extensor" response is a part, may be obtained not only from the sole, but when well developed by stimulating the skin and deeper structures on any part of the lower limb. In the light of this the nature of many reflexes which have been described as isolated signs of pyramidal tract disease, *e.g.* the "extensor" plantar response, Oppenheim's and Gordon's signs, and many well others, becomes clear. In all of them a stimulus is applied to some part of the lower limb, and the response is a flexion reflex, whose most obvious component is "extension" of the great toe. It is unfortunate that the term "extensor response" is commonly used to describe a movement which is physiologically one of flexion.

Sensory symptoms.—Sensory loss may appear first in the area supplied by the roots arising from the cord at the level of the lesion, or in parts below.

As a rule motor disturbance is severe before any remote sensory loss is found. In some cases, especially of spinal caries, the limbs are completely paralysed before sensation is affected. The reverse condition, severe sensory loss with slight motor disturbance, does not occur in compression. Remote sensory loss appears first in one of two positions. In most cases the soles first become less sensitive, then the legs, and later the thighs. In a smaller number the loss appears first over the lower sacral segments, and extends upwards in segmental progression. When both sides are equally compressed all forms of sensation may suffer equally; but in most cases position and passive movement, temperature, pain and touch are impaired in this order.

If one side of the cord is more affected than the other the signs are those of a modified Brown-Séquard syndrome with the superficial sensory loss greatest in the limb in which most power is retained, and loss of position and passive movement greatest in the weaker limb. Ultimately, whatever the order of loss at the beginning, as the compression increases, sensation of all kinds is diminished or lost in all parts below the lesion. In exceptional cases the skin in the distribution of the lowest sacral segments retains its sensibility when the loss in all other parts below the lesion is severe. Such a distribution of sensory loss suggests a lesion damaging the more mesial fibres of the sensory path. Occasionally the onset of symptoms in extramedullary compression is rapid, and severe paraplegia develops in a few days. This is seen most often in cases of sarcoma of the mediastinal or retro-peritoneal spaces where the growth, which has infiltrated one or more vertebral bodies, surrounds the dura and produces œdema of the cord. In cases of rapid onset the limbs are flaccid from the beginning, and the tendon jerks are diminished or lost.

INTRADURAL COMPRESSION

When the pressure is equal on both sides of the cord, the symptoms are the same as in extradural compression. If one side is affected before the other, as by tumours growing from a nerve root, the symptoms are at first unilateral, and in many cases spasticity and weakness are confined for a long time to the lower limb on the same side as the tumour. Occasionally alterations in subjective sensation (*paræsthesiæ*) in the limb of the opposite side precede motor symptoms by a considerable interval, and form the grounds for the patient's first complaint.

At a later stage the following characteristic syndrome appears:

1. A band of sensory loss, on the side of the tumour in an area corresponding to the distribution of the sensory roots arising from the cord at the level of the lesion, often with root pains in the same area.
2. Weakness and spasticity confined to or greatest in the limb on the same side (pressure on the pyramidal tract).
3. Diminished sensibility to temperature, pain, and touch on the opposite side (pressure on the crossed sensory tracts).
4. Impairment of the sense of position and passive movement in the weaker limb (pressure on uncrossed sensory tracts in the posterior columns).

As the compression increases both limbs become weak, spastic, and insensitive, and the symptoms progress as in extradural compression. Examination of the cerebro-spinal fluid often reveals a condition which is practically

pathognomonic of compression--*the loculation syndrome* of Froin. This consists, in its complete form, in an increase in the amount of albumin with absence of or slight increase in the number of cells, and a yellow colour (xanthochromia) in the fluid. The normal amount of albumin is about 0.025 per cent. In compression it is often increased a hundredfold or more, and readings above 1 per cent. are very common. A low cell count and 0.1 per cent. of albumin are very strong evidences of compression, and as the amount increases the diagnosis becomes more certain. Xanthochromia is common; but it occurs in other conditions, and its value as a sign is slight. These changes are found in the fluid only below the site of compression.

Diagnosis.—A complete diagnosis establishes: (1) the existence of compression; (2) its situation; (3) its pathological nature.

1. When signs of injury to the cord or nerve roots are found associated with disease of the spine at a compatible level the diagnosis is obvious. What root pains are the only symptom the diagnosis is difficult. They are often falsely interpreted as referred pains, or as indications of disease in the painful part itself. Thus angina pectoris, gall-stones, pleurisy, renal colic, hip-joint disease and other painful conditions have been diagnosed, and unnecessary operations have been performed. Pain of root distribution should always arouse suspicion, and provoke a careful examination of the spine and of the nervous system.

If the signs are those of spastic paraplegia, spinal syphilis, disseminated sclerosis, syringomyelia and amyotrophic lateral sclerosis must be excluded. *In many cases this can be done by examination of the cerebro-spinal fluid.* Patients with removable spinal tumours are still allowed to develop incurable paralysis because this examination is not made. Such an omission in a case of paraplegia of doubtful origin amounts to neglect. The loculation syndrome in the fluid is almost pathognomonic. It is never absent in chronic cases, and is never found in any of the focal or system diseases, for which compression might be mistaken.

Spinal syphilis is detected by examination of the blood and cerebro-spinal fluid. Valuable time may be lost, however, or an irretrievable error made, when the blood of a patient with a removable tumour happens to react positively to Wassermann's test. In a series of cases operated upon by Sargent at the National Hospital, the reaction was positive in four patients from whom a non-syphilitic tumour was removed.

Disseminated sclerosis may present the picture of progressive spastic paraplegia with considerable sensory loss, and confusion between it and compression by slowly growing tumours is common, each disease being mistaken for the other. The more serious error is to mistake the curable for the incurable disease. It is advisable to feel dissatisfied with the diagnosis of disseminated sclerosis, so long as the symptoms are purely spinal, and to re-examine the patient at intervals in the hope of finding evidence of a simple tumour. The transient nature of the early symptoms and the presence of signs of cranial nerve troubles serve to distinguish disseminated sclerosis in most cases. Slight nystagmus must not be accepted as decisive evidence against tumour, as it may be present in compression, especially of the cervical region.

Amyotrophic lateral sclerosis simulates compression in the cervical region, in that wasting in the muscles of the hands and arms is associated with

signs of spastic paraplegia in the lower limbs; but is distinguished by exaggeration of the tendon reflexes in the wasted arms, by fibrillary tremors in the muscles, and by the absence of objective sensory disturbances.

Syringomyelia is easily recognised by the characteristic sensory changes. Dissociation of sensation to the degree which is common in this disease is never seen in extra-medullary lesions.

2. SEGMENTAL DIAGNOSIS.—As the motor sensory and reflex functions of each segment of the cord are known, the level of the lesion can be deduced by noting the highest point at which these functions are impaired.

Motor localisation.—Each segment of the cord contains nuclei for several muscles, and most muscles receive nerve fibres from more than one root; but as each muscle seems to have one main root of supply, the weakness, wasting and loss of tone vary in distribution with the segment affected. The muscles which suffer most when the corresponding segment is damaged are named hereunder:

*C*₄. Supraspinatus, infraspinatus. *C*₆. Biceps, deltoid, brachialis anticus, supinator longus. *C*₆. Pronators of forearm. *C*₇. Triceps, extensors of wrist and fingers. *C*₈. Flexors of wrist and fingers. *D*₁. Small muscles of the hand. *D*₂₋₁₀. Intercostal muscles. *D*₇₋₁₂. Muscles of abdominal wall. *D*_{11-L}₄. Ilio-psoas (mainly *L*₅). *L*₄. Adductors of thigh. *L*₅. Abductors of thigh, extensors of knee. *L*₅. Hamstrings. *S*₁. Glutei—calf muscles. *S*₁. Anterior tibial muscles—peronei—small muscles of foot.

Wasting of the muscles in an intercostal space is a valuable guide, as the muscles of each space are innervated from one segment alone. If the lesion is at the level of the ninth dorsal segment the rectus abdominis is paralysed below a point about an inch above the umbilicus. In such a case, when an attempt is made to raise the head against the resistance of a hand placed on the forehead when in the supine position, the upper part contracts and the umbilicus is drawn upwards (excursion of the umbilicus). If the lesion is at the twelfth dorsal segment the entire rectus contracts, but the iliac regions bulge, owing to paralysis of the lower part of the oblique muscles.

Localisation by changes in the reflexes.—Above the lesion the reflexes are normal, at its level they are diminished or lost, below it the skin reflexes are diminished or lost, and the tendon reflexes are exaggerated. The segments on which important reflexes depend are:

*C*₆. Biceps- and supinator-jerks. *C*₆. Pronator-jerks. *C*₇. Triceps-jerks. *D*₇₋₁₂. Abdominal reflexes. *L*₂. Cremaster reflexes. *L*₂. Knee-jerks. *S*₁. Ankle-jerks. *S*₁. Plantar reflexes.

In lesions involving the fifth cervical segment of the cord, such as may be found in syringomyelia and in injuries associated with dislocation of the cervical spine, Babinski has recorded that the supinator jerk may be abolished and replaced by finger flexion when the lower end of the radius is tapped. This is known as "inversion of the radial reflex," and is a useful localising sign in lesions of the segment in question.

Sensory localisation.—The sensory areas supplied by each segment of the cord are shown in the diagram on the opposite page. Root pains in the distribution of one or more of these areas form a sure guide to the affected segment. When they are absent, the level of the lesion is determined by ascertaining the highest point at which sensation is impaired. Very often when

the two sides of the cord are compressed unequally the anæsthesia is confined to one side, or extends higher on one side than on the other. In these cases, when the segmental diagnosis is made from the sensory signs alone, the lesion, a tumour for example, is found several segments higher than the point indicated by the signs. The uppermost limit of sensory loss in these circumstances is a result of interruption of the sensory paths in the cord, and the discrepancy in the signs is due to the oblique course taken by the sensory fibres in crossing the cord. In the mid-dorsal region the decussation for pain and temperature is complete one segment above the point of entry of the root conveying these impressions to the cord, that for touch in two segments. At higher levels, crossing takes place more slowly, until in the upper cervical region impulses which enter together in one root ascend on the same side of the cord for five or six segments before all of them reach the opposite side. At all levels pain crosses soonest, then heat, then touch and touch slowest of all.

It follows that in unilateral lesions the upper level of the anæsthesia on the opposite side of the body, caused by injury to sensory paths in the cord, is below the segmental level of the injury. Also that the level is higher for one form than another. The fibres which cross slowly escape by ascending beyond the lesion on the uninjured side before they cross, while those which cross quickly are caught after crossing. Hence the level of sensory loss is highest for pain and lowest for touch, with temperature intermediate. Occasionally the tumour is found below the level predicted. In these cases the functions of the segments above the lesion are impaired by œdema.

Surface anatomy.—If the cord is to be exposed at the level of the affected segments their relation to the spinous processes of the vertebræ must be known. This is obtained as follows: In the cervical region to the number of the spine add 1—the fifth cervical spine lies over the sixth cervical segment; to the number of the upper five dorsal spines add 2—the fourth dorsal spine lies over the sixth dorsal segment; down to the tenth dorsal spine add 3—the tenth dorsal spine covers the first lumbar segment. The eleventh dorsal spine corresponds to the third lumbar segment, and the twelfth to the first sacral. The cord terminates just above the level of the first lumbar spine.

Intrathecal injection of lipiodol.—The upper level of a lesion which narrows or obliterates the lumen of the spinal canal, or simply blocks the spinal subarachnoid space, can be ascertained by injecting lipiodol through the occipito-atlantoid ligament into the subarachnoid space, the lipiodol falls rapidly to the point of constriction, where it is arrested and can be seen clearly by X-rays. If there is no constriction it falls to the bottom of the thecal space, where it remains indefinitely and does no harm. This procedure is of great practical value when the existence of a compressive lesion or its exact site is in doubt.

3. **DIAGNOSIS OF THE CAUSE OF COMPRESSION.**—When spastic paraplegia develops in a patient who is known to suffer from *spinal caries*, the cause is obvious; but when it precedes the appearance of signs of bone disease the diagnosis is difficult. In all cases of compression the spine must be examined repeatedly for deformity, tenderness and limitation of movement. If tenderness is found constantly in the same place, and the nervous symptoms are compatible with disease of the underlying segments, disease of the bones is

almost certain. In young persons disease of the spine is usually caries, and in adults caries is also the commonest cause; but tumours of the spine and aneurysm must be excluded. Severe root pains are rare in caries but are the rule in vertebral new-growths. An aneurysm would present other signs.

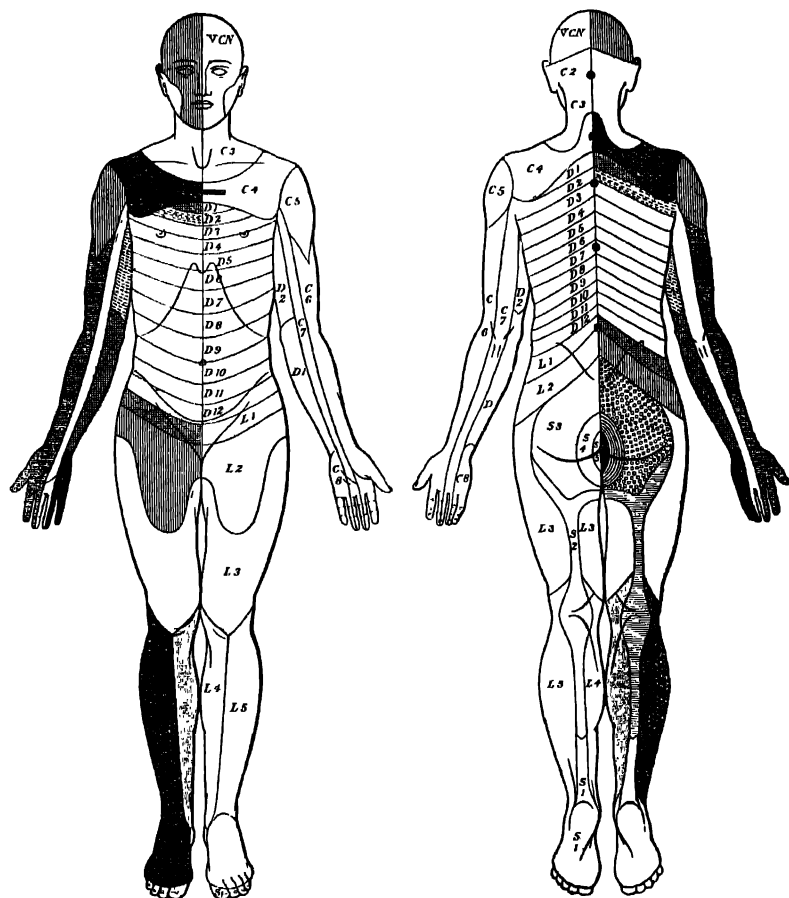


FIG. 99.—Diagram of cutaneous areas of posterior nerve roots (after Collier and Purves Stewart).

An X-ray picture will usually demonstrate the presence and nature of the bone disease.

Vertebral tumours.—When root pains occur in a patient with malignant disease, or from whom a malignant growth has been removed, the diagnosis is clear. Mistakes are easily made when pains are the first symptom, as their

root origin is not recognised. Diminished sensibility in the painful area indicates the nature of the pain, and this directs attention to the spine, where tenderness or deformity is discovered. As most vertebral tumours are secondary, the next step is to examine the parts where carcinoma is common, remembering that a small primary growth, *e.g.* in the breast, thyroid or prostate, may give rise to widespread metastases in the bones. In the absence of a history or signs of new-growth in other parts, the diagnosis is founded on the combination of local tenderness or deformity and rigidity of the spine with root or cord symptoms. The severity of the root pains, and their great aggravation by movement, are characteristic.

Meningeal tumours.—Severe pain of root distribution is present in many cases, and when this is followed after some time by spastic paralysis of slow onset and steady uninterrupted progress, affecting first one leg and then the other, the combination and especially the course of the symptoms are almost pathognomonic. In caries the root pains are rarely severe, signs of bone disease are rarely absent, the paralysis is usually bilateral from the beginning, and is severe before any sensory loss is found. The distinction from vertebral new-growth may be impossible when the latter occurs without bone symptoms or X-ray signs. Practically the diagnosis between meningeal and intra-medullary tumours is impossible. If the signs and symptoms in any case are those of compression, and an approximate indication of the level of the lesion can be given to guide the surgeon, then, if other obvious causes such as caries, aneurysm and vertebral tumour have been excluded, an operation must be performed. Until the lesion can be seen the diagnosis is in doubt.

Course and Prognosis.—*Spinal caries.*—The course of the bone disease does not always run parallel with the paralysis, and either may alter in severity independently; but if the caries undergoes cure the paralysis usually diminishes. Considering the severity of the paralysis, the prognosis is favourable and astonishing recoveries occur. The outlook is best in young people with disease in the dorsal region. Many recover completely, but more often, especially in adults, recovery, though considerable, is partial. So long as the lower limbs remain spastic in the extended position with exaggerated tendon reflexes the prognosis for complete recovery of power is good; but if the limbs become flexed, if they become flaccid, if the knee- and ankle-jerks are lost, if sensory loss is severe, or if there is wasting in the limbs following damage to lower motor neurones, the outlook is very bad. Many patients live for years with severe paralysis; but life is constantly endangered by sepsis from bed-sores, ascending infections of the urinary tract, chest complications, and tuberculous disease in other parts.

Vertebral tumours.—When sarcoma or carcinoma spreads to the vertebræ from surrounding parts the duration of the disease is measured in weeks or months, and death is due to the primary condition. In primary sarcoma, and in some cases of carcinoma of the vertebræ, life may be prolonged for a year or two, and death is due rather to complications of the cord disease—bedsores, cystitis, etc.

The course of *tumours of the meninges* is often extremely slow. Root symptoms may precede paralysis by months or even years, and the weakness may increase gradually for several years before walking becomes impossible. Malignant growths are fatal, and simple growths equally so, if not removed. Most patients with simple tumours come to operation during the second

year after the onset of the first symptom. The mortality after operation for the removal of simple tumours is very low in skilled hands. The prognosis for recovery of power depends in part on the duration of the weakness in the lower limbs. Complete recovery may be expected if it has not lasted more than a year, and if sphincter control has not been lost. When the paralysis is of longer duration recovery, though gratifying, is rarely complete. Nevertheless, full return of power has been seen after three years of severe paralysis.

Treatment.—*Spinal caries.*—This is to be directed to curing the bone disease in the hope that cure of the paralysis will follow. Complete rest on the back and fixation of the spine for many months is the routine treatment. The general condition of the patient is to be improved by fresh air, a liberal diet and cod-liver oil, iron and arsenic, and great care is to be taken to prevent bedsores, cystitis and deformities of the limbs. For adults, especially if they are bread-winners, a more rapid cure is desirable. This is provided by surgery in Albee's operation, or a modification thereof, in which a bone graft from the tibia is wired into a cleft made in the spines of the affected region. In this way fixation is effected, further deformity is prevented, and the time lost is reduced from a year or more to a few months. Adults sometimes recover after a short rest in bed, if a suitable jacket is worn to immobilise the spine.

Operation must be considered—(1) when a sudden increase of deformity or severe root pains and great increase of paralysis come on together, and suggest pressure by displaced bone; (2) when an abscess forms; (3) when paralysis persists long after the bone disease is cured, or when in an adult there is no improvement after 6 months' rest; (4) when life is endangered by respiratory paralysis.

Vertebral tumours.—In slowly growing primary growths of the vertebræ laminectomy is indicated to relieve pressure, or to prevent pain by cutting sensory roots or dividing the antero-lateral columns of the cord. The operation is merely palliative, but is often followed by considerable temporary recovery.

Meningeal tumours.—If the symptoms suggest an intraspinal tumour, and a segmental diagnosis has been made, an exploratory laminectomy should be done in the hope of finding a removable tumour.

2. COMPRESSION OF RAPID ONSET

Ætiology.—The commonest causes of rapid compression are fracture or dislocation of the spine.

Dislocations occur most often between the atlas and axis, or between the fifth and sixth cervical vertebræ. They are sometimes incomplete, and thus may cause compression of slow onset. More often they are complete, and the cord is compressed between the laminae of the dislocated vertebra, which is displaced forwards, and the body of the underlying vertebra. Fractures are commonest in the lower dorsal and upper lumbar regions, and follow most often a fall from a height on to the feet or buttocks. They also occur apparently spontaneously in spinal caries and in vertebral tumours. A blow on the back may fracture the vertebral arches, and cause compression of the cord. The degree of damage to the cord varies greatly.

Symptoms.—Sudden severe compression produces a complete interruption of conduction through the damaged segments; that is, a physiological transection of the cord. There is total flaccid paralysis, total sensory loss, abolition of tendon reflexes, and retention of urine. For a few days there may be no plantar response, but a Babinski response then makes its appearance. Bed-sores often develop with great rapidity, and many patients succumb within a few weeks. But in those that survive, retention of urine gives place to overflow incontinence, the tendon jerks may return, and the condition described above (p. 1718) as “paraplegia-in-flexion” may ensue. In this case, any stimulus applied below the level of the lesion produces violent flexion spasms in the legs, contraction of the abdominal wall, and the expulsion from the bladder of some—but not of all—its contents. Head and Riddoch have applied the term “mass reflex” to this phenomena.

Dislocations between the fifth and sixth cervical vertebræ may produce severe damage to the cord as described above, with the addition that the arms are paralysed also, the sympathetic pathways in the cord are interrupted, and there is narrowing of the palpebral fissures, and sometimes priapism. Less well-recognized are the various clinical pictures of minor injury to the cord as the result of vertebral injury. These include slight weakness, with ensuing wasting of the hands and forearms, slight spastic paresis of the legs, with the corresponding changes in the reflexes, narrowing of one or both palpebral fissures, and inversion of the radial reflex on one or both sides. There may be no sensory loss after the first two or three weeks. The slow progress of the wasting in the arms for some time after the injury may lead to a diagnosis of amyotrophic lateral sclerosis, but care in examination and history-taking should prevent this error.

Prognosis.—The prognosis is always extremely grave in severe cases, death resulting in high cervical lesions from paralysis of all the muscles of respiration, in lesions at lower levels from sepsis following bedsores, or from infection of the urinary tract.

Treatment.—When the signs are those of complete division of the cord, treatment by operation is directly contra-indicated. If some voluntary power is retained, or if sensory loss is not absolute, operation may be considered, especially if the compression is caused by fracture of the vertebral arches. The cord should be exposed in every case where the level of the fracture points to injury of the cauda equina. These roots being peripheral nerves have a chance of regenerating, and this may be enhanced by freeing them from compression by displaced fragments of bone. With skilled nursing, patients with complete division of the cord may survive and live for many years.

GENERAL MANAGEMENT OF PARAPLEGIA.—In all cases of severe paraplegia from spinal cord lesion where sensory and sphincter functions are also impaired or lost, whatever the nature of the lesion, there are certain general principles of treatment. The patient should be nursed on a fracture bed with an air, water, or rubber mattress. The back should be attended to four hourly, first washed with soap and water, then carefully dried, rubbed with surgical spirit or eau-de-Cologne, and powdered. These measures harden the skin and make it less likely to break down under the constant pressure of the body weight. The patient's position should be changed from time to time to prevent the development of sacral bedsores. As far as possible

care should be taken to prevent the skin from becoming wet and sodden when there is incontinence of urine, and the toilet of the anus after defæcation should be careful and thorough. There are various remedies for the sacral or trochanter bed sore when it develops. Separation may be hastened by wet dressings of eusol, sometimes by fomentations—though the latter should be used with caution. The ulcer is packed with eusol gauze, or with an ointment of zinc oxide and castor oil. When it is clean and healing begins, it may sometimes be hastened by dressings of gauze soaked in red lotion. The heels should also be carefully watched for the appearance of the hæmorrhagic blisters which herald the development of a sore. Rings for the heels may avert them, and air rings for the sacrum may also be needed.

SYRINGOMYELIA

Synonym.—Slatu Dysraphicus.

Definition.—Syringomyelia is a very chronic and irregularly progressive disease of the spinal cord and brain stem, dependent upon a peculiar lesion of the grey matter, glial increase and the formation of irregular cavities being the most conspicuous features of this lesion. Clinically, the malady is characterised by a deep loss of sensibility to pain and to temperature, other forms of sensibility remaining relatively unaltered, and by muscular atrophy and weakness of varying distribution in the upper extremities, and further by spastic weakness of the lower extremities, owing to involvement of the pyramidal tracts at the level of the lesion.

Ætiology.—Both sexes may be affected, and males are more prone to suffer than females. Heredity plays no part in its causation. Age is most important, in that this disease appears either to be congenitally installed, or to commence during the period of growth. It has been diagnosed with accuracy as early as the sixth year of childhood, and rarely if ever do the symptoms commence later than the age of 30 years.

Pathology.—The primary lesions of syringomyelia are always found in that region of the spinal cord which was originally occupied by the central canal, or in close connection with the ventricular system of the brain stem; and it is certain, therefore, that syringomyelia is referable to a pathological process affecting the central canal and its surrounding glia, and that this pathological process, in many cases at least, is installed before the completion of the development of the central canal of the nervous system. Two essential lesions and four other commonly occurring lesions make up the morbid process of this disease:

Essential lesions.—(1) Cavitation of the posterior part of the grey matter; (2) gliosis, with liquefactive degeneration of the abundant glia—*other lesions commonly but not invariably present*; (3) degeneration of lower motor and vasomotor neurones; (4) degeneration of lower sensory neurones; (5) distension of cavities producing pressure effects; (6) secondary ascending and descending degenerations.

The seat of commencement of the disease is invariably in the dorsal grey matter of the lower half of the brain stem and upper half of the spinal cord, and most commonly of all, in the lower three cervical and upper three dorsal segments. The cavitation occurs primarily always in that part of the grey

matter which held the original central canal, namely, the region of the posterior commissure, posterior median septum and posterior horns.

Degeneration of the lower motor neurones occurs in at least half the cases. It is most marked in the cervical and upper dorsal regions of the cord, and may be very local or extensive. It is commonly explained as caused by the extension of the gliosis and cavitation into the anterior horns of the grey matter, and by the distension of the cavities exerting pressure upon these regions, and causing atrophy of the cells. To the pressure exerted by such distended cavities are also attributed the ascending and descending degenerations which are commonly found in the pyramidal tracts below the level of the lesion, and in the posterior columns above.

Tumour formation is not uncommon in cases of syringomyelia. Massive growth may be found in the pons and in the spinal cord.

Symptoms.—*Disturbances of sensibility.*—By far the most constant and characteristic feature of syringomyelia is a sensory loss of a peculiar kind which was named by Charcot "the dissociated sensory loss." This is a loss of sensibility to painful impressions and to thermal stimuli, while sensibility to touch, to vibration, to position, to passive movement and to the appreciation of location upon the skin, remain relatively or entirely intact. In other words, those forms of sensibility which travel by a path crossing in the commissures of the spinal cord are lost, because the lesion of syringomyelia destroys especially the region of the commissures, while these forms of sensibility which travel by paths which are uncrossed in the spinal cord and do not traverse the region especially affected by syringomyelia, but are conducted by the posterior columns, are not affected. Further, the lateral region of the dorsal reticular formation of the medulla, mesial to the restiform body and ascending root of the fifth nerve, and a little ventral to these structures, is especially prone to the lesion of syringomyelia, and it is this region which contains the whole path for pain and temperature sensibility from the opposite half of the body, and a lesion in this situation will produce hemianalgesia and hemithermanæsthesia, while the paths for other forms of sensibility, situated mesially on either side of the *raphé* of the medulla, escape. Again, the lesion excavating the ventral horn in any part of the cervical region may extend so as to interrupt the spinothalamic tract which lies immediately dorso-lateral to the ventral horn, and so cause loss of pain and temperature sense on the opposite side everywhere below the level of the lesion.

The destruction of the commissures in the lower cervical and upper dorsal regions produces the dissociated sensory loss symmetrically over the thorax, upper extremities, neck and face, the distribution varying with the extent of the lesion. The sensory loss over the face is explained in that the sensory root of the trigeminal nerve has its ending in the upper three segments of the cervical spinal cord, and the pain and temperature sensibility of the face is interfered with if the posterior grey commissure in the region of these segments is damaged. Only rarely does the symmetrical sensory loss extend below the thorax, for the reason that the spinal lesion does not often extend below the mid-dorsal region. The sensory loss will vary in depth, extent and symmetry of distribution according to the completeness, extent and symmetry of the lesion. Thus, in early and slight cases, the sensory disturbance may not amount to more than a relative loss of pain and temperature confined to the

hands and ulnar borders of the forearms, while in an advanced case there is usually complete inability to appreciate painful and thermal stimuli over an area which would be covered by a sleeved jacket, and this area often extends over the neck and the face. Combinations of the "sleeved jacket" sensory loss with hemianalgesia and hemithermanæsthesia often occur in cases where both the spinal lesion and the medullary lesion are present. The dissociated sensory loss makes its advent insidiously, and is often unnoticed by the patient and discovered for the first time on medical examination. Or it may appeal to the patient, who on bathing finds that he appreciates heat and cold upon some parts of the skin and not on others. Not infrequently he finds that he injures himself or burns himself without noticing it at the time.

Subjective sensibility is not often affected, and for the most syringomyelia may be described as a painless disease; but there are very notable exceptions. Sensations of heat and cold, dull fixed pains, lasting neuralgic pains, and lightning pains in no way differing from those of tabes, may occur. These pains are confined to the regions which are the seat of the other symptoms. Especially important in this connection are those cases in which the distension of the cervical spinal cord is so great as to cause that structure to press upon the bones of the spinal canal. Here constant and often intolerable aching pain in the neck, upper extremities and thorax may result, with rigidity of the neck, and this may render life so insupportable as to necessitate surgical interference for the relief of the pressure.

Muscular atrophy.—This common clinical feature of syringomyelia is met with in considerably more than half the cases. As may be gathered from the nature of the lesions, though usually bilateral, it is often not symmetrical, and may be entirely confined to one side. The intrinsic muscles of the hands and the muscles of the ulnar side of the forearms are first and most affected in the ordinary run of cases. The atrophy is often here confined, but it may extend up the arm; but it is unusual for the whole upper limb to be affected. Sometimes the shoulder muscles are first affected, and again the scapulo-thoracic and humero-thoracic muscles may be early involved. The upper intercostals, and that section of the muscles which supports the spine, supplied from the upper six dorsal segments suffer, but the scalenes seem generally to escape. The muscular atrophy is strictly limited, and is apt to become complete in the muscles affected. The lesions of the medulla may involve the motor nuclei of the cranial nerves. Atrophic paralysis of the muscles supplied by the vago-accessory nerve is far from uncommon, and the discovery of this paralysis in a young subject should always arouse suspicions of the presence of syringomyelia. The paralysis is unilateral and involves palate, pharynx and all the muscles of the larynx upon the affected side. Similarly but in much rarer cases, atrophic paralysis of the face, of the trigeminal muscles, of the sternomastoid and trapezius or of the hypoglossal muscles may occur from a unilateral involvement of the corresponding motor nuclei. Fibrillation in the affected muscles is said by most writers to be of common occurrence. It has been conspicuous by its absence in most of the large number of cases which have come under our observation. One would expect it to be confined in syringomyelia to such times as the muscular atrophy is progressing.

Contractures resulting from the muscular atrophy are commonly seen in the hands, and the deformity resulting tends towards the "griffin's paw"

type, but hardly reaches the degree seen in ulnar nerve paralysis, and is often much modified by trophic and vasomotor changes, and by the results of injuries and whitlows.

The lower extremities escape so far as atrophy of muscles is concerned. Spinal curvature is present in many cases. It consists essentially in a kyphosis or kypho-scoliosis of the upper dorsal region, with a compensatory lordosis and lateral curve in the lumbar region. The upper convexity is to the left from the major use of the right hand. It is dependent upon paralysis of the trunk muscles, from involvement of the anterior horns in the upper dorsal region, and, in addition, dystrophic changes in the bones may be factors in its production. It is more marked the earlier it commences during the period of growth, and where heavy manual occupation has been followed.

Trophic and vasomotor disturbances.—Thickening of the bones or a condition of osteoporosis and brittleness may be met with. More often Charcot's arthropathy occurs. It differs in no way from the similar condition in tabes dorsalis, and is confined to the joints of the analgesic region, and affects the joints of the lower extremity only when there is a hemianalgesia from a lesion of the spinothalamic tract either in the cord or in the medulla. In syringomyelia Charcot's joints are seen chiefly in workmen who are engaged in occupations which constantly expose the analgesic joints to jarring and bruising.

The most characteristic of the trophic changes consists in thickening of the subcutaneous tissue and of the skin itself, which is seen in the hands. The fingers become thick and swollen and lose their natural outline, the tips become blunted, and the knuckle-folds thick and coarse, and some vasomotor paralysis renders them unduly red, or even blue. They have been termed "sausage-like" fingers, and often stand out in contrast to the wasting of the intrinsic muscles of the hand. A similar condition affecting the whole hand is common, and was termed by Charcot the "fleshy hand" or "main succulente." The analgesic condition of the hands and the thermanæsthesia present expose them unduly to injuries and, since these injuries are likely to be unnoticed or disregarded, septic infection arises easily, and the results of injuries, burns and whitlows are frequently seen, giving rise to further deformity from scars, loss of the terminal phalanges, from whitlows and contractures, and from sepsis extending to the tendons.

The lower extremities usually present a slight spasticity, with the signs of involvement of the crossed pyramidal tracts. This does not often produce much disability in the use of the lower limbs. In cases, however, where the lesions involve the lateral regions of the cord, either by direct extension or by the pressure of distended cavities, severe spastic paraplegia may result. And again, in very rare cases, such pressure may lead to total evascularisation and total transverse lesion of the spinal cord with the appearance of a complete flaccid paraplegia with incontinence, total sensory loss and absent deep reflexes.

Sphincter trouble is usually absent, or slight and occasional; but in cases where paraplegia is severe any degree may occur.

The skin reflexes of the trunk are diminished or absent, and the plantar reflexes are of the extensor type, according to the degree of pyramidal involvement. Some degree of pes cavus is often present. The knee-jerks and ankle-jerks are increased, and foot-clonus, etc., is present.

Considering that the efferent neurones of the cervical sympathetic system have their origin in the brain stem, and their exit from the spinal cord in the lower cervical and upper dorsal segments, thus traversing the whole of the region usually affected by the lesion of syringomyelia, the frequency with which paralysis of the cervical sympathetic occurs is easily understood. It may be complete or incomplete, unilateral or bilateral, and is recognised by smallness of the pupil, narrowing of the palpebral aperture (sympathetic ptosis), and a peculiar flatness of expression on the side of the face affected, with decrease or loss of sweating. These signs are much more obvious when unilateral than when bilateral, for, in the absence of the contrast which a normal side of the face gives, they are often overlooked when bilateral.

Ophthalmoplegia is very rare, but it may occur, since the syringomyelia lesion may be found as high as the region of the third nucleus. Nystagmus is an almost constant feature of syringomyelia, as it is also of most lesions of the cervical spinal cord.

MORVAN'S DISEASE.—This variety of syringomyelia is so peculiar in its clinical aspect as to need especial description. In addition to the lesion of the spinal cord characteristic of syringomyelia, there are intense changes in the periphery of the nerve trunks of the limbs. Instead of the usual loss of pain and temperature sensibility, distributed in jacket form upon the upper limbs and trunk, there is absolute loss of all forms of sensibility in the hands, wrist high, and in many cases also in the feet, ankle high. Progressive atrophy of the intrinsic muscles of the hands and feet occurs. Severe vasomotor paralysis brings about permanent cyanosis of the hands and feet, with much thickening of the skin and subcutaneous tissues, to which are added the effects of injury and septic processes in insentient regions, in the form of whitlows, necrosis and loss of digits. Another peculiarity of this malady is that the extremities are exceedingly painful in the early stages and until the sensory loss becomes deep. Morvan's disease resembles Raynaud's disease in the cyanosis and tendency to necrosis of the fingers and toes, but it is easily distinguished by the complete absence of intermitting vascular spasm and by the peculiar loss of sensibility. Anæsthetic leprosy may be distinguished from Morvan's disease by the characteristic skin lesions in other parts of the body, by the palpable thickening of the nerve trunks, and by the less definitely limited areas of sensory loss. Every transition between typical syringomyelia and Morvan's disease has been described.

Course and Duration.—The malady, commencing insidiously, progresses very slowly, and often ceases to progress for periods which may amount to many years. The tendency to the destruction of life is not great; but when rapid extension of the physical signs, and especially of paralysis and muscular atrophy of the upper extremities and respiratory muscles, occurs, the end is likely to come quickly. Signs of great distension of the cavities, such as pain and rigidity of the neck, and also severe and increasing paraplegia, with sensory loss of all forms of sensibility below the level of the lesion, point to a rapidly fatal termination.

It is not unusual to meet with well-marked cases in which the signs develop and increase during late childhood and early adult life, and then remain more or less in a stationary condition, allowing an occupation to be followed until well after middle life has been reached; but with the advent of the degenerative period of life, from the age of 45 years onwards, there is

always a slow increase of the disability which puts an end to useful capacity. Many of the cases become incapacitated in early life, after which the disease becomes arrested, and the patients live on for many years, sometimes in a bedridden condition. Few reach the age of 60 years. Rapid extension of the physical signs leads to death from involvement of respiratory muscles. Otherwise the patients succumb to intercurrent disease. Sudden and unexpected death sometimes occurs, and it is especially to be remembered that this is likely to occur after the administration of anæsthetics, and as a result of surgical procedures.

Diagnosis.—Syringomyelia has to be differentiated, in its early stages, from those diseases which cause slowly progressive muscular atrophy in the upper extremities, and, in its later stages, from other lesions of the central region of the spinal cord. Those cases in which the lesions are chiefly in the ponto-medullary region must be distinguished from other slowly oncoming lesions of the brain stem.

The age of onset, during the later years of childhood and the earlier years of adult life, is important, and during this period slowly developing paralysis, with or without muscular atrophy and with sensory loss, should always suggest the possibility of syringomyelia. Other causes, which may produce this symptom group, and which may be confused with syringomyelia, are local lesions of the peripheral nerves, local lesions of the brachial plexus, and, especially, the lesion produced by the presence of cervical ribs, root lesions, lesions of the central grey matter of the spinal cord, especially central tumours of the spinal cord, hæmatomyelia, and lastly certain general diseases of the nervous system, progressive muscular atrophy, peroneal atrophy and myotonia atrophica. That the sensory changes of syringomyelia of peculiar nature are usually the first signs of that disease is important; but unfortunately is not without many exceptions, both as to the nature of the sensory changes and as to their time of appearance.

Local lesions of the peripheral nerves produce signs which are confined to the distribution of the nerve involved; the sensory loss is to all forms of sensibility, and the condition is commonly unilateral. In syringomyelia, however, the lesion in the early stages may be confined to one side of the cord, and to one posterior horn so far as the production of sensory loss is concerned, and the muscular atrophy may be so narrowly confined to the distribution of the ulnar nerve as to cause close resemblance between the two conditions. Any sensory loss over the trunk, or signs outside the distribution of the peripheral nerve, will, if present, clearly divide the two conditions.

Cervical ribs may produce slowly progressive atrophy of muscles, pains and sensory loss, very difficult to distinguish from those resulting from syringomyelia. The diagnosis in these cases is beset with peculiar difficulties, for so frequently do cervical ribs produce no nervous symptoms at all that their presence, when demonstrated, does not argue that they are the cause of the symptoms. Again, cervical ribs are among the commonest of the developmental peculiarities which are so frequently seen in the subjects of syringomyelia. Slow muscular atrophy and slowly oncoming sensory loss and perhaps pain characterise both syringomyelia and cervical rib paralysis, and the distribution may be unilateral or bilateral in either condition; but it is only when the signs and symptoms are strictly confined to the upper

extremities and neck that difficulty arises. The slightest definite physical sign outside of this region at once turns the diagnosis in favour of syringomyelia, and of these signs cervical sympathetic paralysis, sensory loss on the trunk, and alteration of the abdominal and plantar reflexes are most important. A very careful search must be made for any such signs, and the patient observed over a considerable time before a certain diagnosis is made.

Lesions of the nerve roots, either from inflammatory conditions, bone disease, pachymeningitis or neoplasms give rise to more severe pain than does syringomyelia, and the development of the symptoms is much more rapid. Lesions of the central grey matter of the spinal cord may produce a symptom complex, closely resembling that of syringomyelia. Central tumours of the spinal cord, when of slow growth, are hardly distinguishable, inasmuch as the lesion of syringomyelia is in reality a central tumour of the cord. The majority of central tumours, however, are of more rapid development, and speedily produce severe paraplegia. The presence of Froin's syndrome (hyperalbuminosis in the cerebro-spinal fluid) is much in favour of tumour.

Progressive muscular atrophy in its early stages may cause difficulty in diagnosis, since the muscular atrophy in syringomyelia may in rare cases precede the appearance of any sensory loss or may be well marked when the sensory loss is slight. In this connection widely distributed fibrillation is of great importance in indicating a diagnosis of progressive muscular atrophy, particularly if it be seen in muscles not conspicuously wasted. In peroneal atrophy the atrophy of the intrinsic hand muscles is always preceded by a more extensive atrophy of the muscles below the knee, which are never atrophied in syringomyelia.

Syringomyelia of the brain stem may be distinguished from other lesions of this region by its insidious onset and the special tendency to the involvement of the lateral region of the medulla containing the vago-accessory nucleus and the central pain and temperature path, so giving rise to a unilateral paralysis of palate, pharynx and larynx with hemianalgesia and hemithermanæsthesia on the opposite half of the body. Often some signs of cervical syringomyelia coexist; but the medullary lesion may exist alone, and it cannot be too prominently borne in mind that any very slowly progressive lesion of the brain stem of insidious onset may be of the nature of syringomyelia.

Prognosis.—Recovery never occurs; but arrest of the disease for long periods is frequent. Those disabilities, which are the result of pressure or distension, may abate spontaneously or as the result of treatment, and in arrested cases training may bring about lessening of the disability. Increasing symptoms, especially if the increase be rapid, are always a cause for anxiety, and increasing involvement of the respiratory muscles is the gravest of events.

Treatment.—Some authorities believe that mercury and iodide of potassium have a definite effect in benefiting the disease when the symptoms are progressing. Application of deep X-rays to the cervical and upper dorsal regions of the spinal cord has been followed by arrest of the progress of the disease and, rarely, improvement of symptoms. Pains are to be relieved with the common analgesics. Massage, exercises and training are all likely to make some improvement in the disability in arrested cases.

HÆMATOMYELIA

Ætiology and Pathology.—Hæmatomyelia, or hæmorrhage into the spinal cord, has in the past figured in neurological literature to a much greater extent than its frequency warrants. It has been commonly supposed, for example, to be an almost invariable consequence of serious local injuries of the cord, whereas in fact in these circumstances hæmorrhage plays a very minor rôle. G. Jefferson has in fact expressed the view that it is doubtful whether such a state as hæmatomyelia actually exists. It is true that small punctate hæmorrhages may be seen in the damaged areas, but as Holmes, from his study of the spinal injuries of warfare, has pointed out, the essential lesion in these circumstances is œdema, followed by degenerative changes in the cells of the grey matter, and in severe cases by central necrosis. Large central hæmorrhages are not found. Similarly, certain inflammatory affections of the cord, such for example as acute poliomyelitis, may show minute hæmorrhages, but here again nothing occurs to which the term hæmatomyelia can reasonably be applied. In short, hæmatomyelia is an exceedingly rare form of spinal cord lesion.

A recent study by C. Richardson made on material at the National Hospital, Queen Square, has lent a necessary sense of proportion to our views on this matter. By the term hæmatomyelia is now usually meant a large central hæmorrhage, with a tendency to spread longitudinally over several segments. The central region of the cord and the dorsal horns are the situations in which this hæmorrhage is commonly observed in the rare instances in which it is to be found. If we agree that traumatic hæmatomyelia is an extremely rare condition, we are left with what has been called "spontaneous hæmatomyelia" and with secondary hæmatomyelia.

SPONTANEOUS HÆMATOMYELIA.—This rare condition appears to arise only when there is some abnormality of the spinal vessels. Such abnormalities are angioma and other malformations, and—excessively rarely—syphilitic and arterio-sclerotic disease of spinal arteries; in other words, the so-called spontaneous hæmatomyelia is really secondary hæmatomyelia.

Symptoms.—From what has been said it is apparent that the clinical picture usually given of hæmatomyelia is that of local trauma of the cord, and since such local crushing is commonly associated with fracture, fracture-dislocation, or transient dislocation of the cervical spine between the fifth and sixth vertebræ, this picture is that of a quadriplegia of sudden onset with slow recovery of variable degree. True hæmatomyelia may indeed occur in the cervical region of the cord, when it is apt to prove fatal. Richardson's analysis indicates that the characteristic clinical picture is that of a sudden paraplegia, accompanied by pain and sensory changes, subjective and objective. It appears that all forms of sensation are lost at first.

In the non-fatal cases a variable degree of recovery is possible. In a few reported cases, it appears that a syringomyelic type of dissociation of sensation may ensue.

Diagnosis.—The diagnosis of primary hæmatomyelia rests upon the sudden onset, the rapid development of symptoms which soon come to a standstill, and the physical signs of a central lesion of the spinal cord. The

distinction has to be made from acute myelitis. Acute myelitis, though rapid in onset, does not show the sudden development of symptoms seen in hæmatomyelia. Prodromata often precede the onset.

Treatment.—The general treatment is that of any severe spinal cord lesion with paraplegia. When there is evidence that a syphilitic lesion of the spinal arteries is in question, the treatment is that of spinal syphilis. Angioma and other malformations of the spinal vessels are not amenable to surgical intervention.

MYELOMALACIA

Synonym.—Softening of the Spinal Cord.

The term "myelomalacia," which implies softening of the Spinal Cord, has been applied by some authors to those conditions of local destruction of the spinal cord consequent upon the cessation of blood supply, and especially upon thrombosis of its blood vessels, as apart from the extensive local destructions which may result from inflammatory conditions. Such a distinction does not rest upon any logical, pathological or clinical basis, for thrombosis and ischæmia make up a part of the pathological process of all traumatic, inflammatory and pressure lesions of the spinal cord, and may occur as terminal events in certain diseases of the spinal cord where vascular lesions are otherwise conspicuous by their absence. Therefore, since softening of the spinal cord may be the result of widely different pathological processes, and since it does not constitute a definite clinical entity, it will suffice here to refer to those maladies in which it is chiefly observed.

TRAUMATIC CONDITIONS.—As a result of high explosives bursting in the neighbourhood of the spinal column, even without signs of external injury or signs of damage to the bones, the spinal cord may be found to be completely diffuent over several segments. The same result may be met with from the passage of a high velocity bullet through the spinal canal, whether the spinal cord be touched by the bullet or not; and again, the same condition occurs from the vibration of an impact when a missile hits and lodges in the surrounding bone, without directly involving the spinal canal or cord. A slighter degree of the same condition may be seen in fracture dislocation. When, as the result of injury to the spinal column, the spinal cord is torn across, the distal segment may soften completely.

PRESSURE LESIONS.—Pressure upon the spinal cord abrogates function chiefly by producing ischæmia and, if the pressure be prolonged or severe, necrotic softening occurs, and the more readily, if there be strangling of the segmental vessels which supply the cord and accompany each nerve root.

INFLAMMATORY CONDITIONS.—In acute spreading myelitis, in which the spinal cord is infected with micro-organisms secondarily to a general blood infection, as may occur in small-pox, gonorrhœa, dysentery, etc., the cord softens and may become diffuent. In acute transverse myelitis, softening depends upon the severity of the initial œdema and its duration, the degree of obliterative arteritis, and the consequent thrombosis that may occur. It may be largely avoided by the energetic and early application of anti-syphilitic treatment.

SENILE PARAPLEGIA.—This condition, which is not very rare, and in which

spasticity of the lower extremities with weakness comes on gradually in later life, and does not, as a rule, reach a severe degree, has been attributed to ischæmia and even to softening of the spinal cord from arterial disease and the failing circulation of old age, by Moxon, who first described it. The pathology of these cases seems by no means certain, and there are few records of the anatomy. It seems certain that no appreciable softening can occur, on account of the slightness of the paraplegia and the absence of any sensory loss. Gowers doubted whether they were spinal in origin at all, and attributed some to the occurrence of cortical changes in the brain, while others he placed in the category of paralysis agitans. From the occurrence of definite mental failure in some of the cases, a cerebral site for the lesion is likely. Dr. Greenfield has recently examined for us a very typical case and found no changes in the spinal cord, but extensive degeneration of the pyramidal cells of the motor cortex.

SUBACUTE COMBINED DEGENERATION

Synonym.—The Anæmic Spinal Disease.

Definition.—Subacute combined degeneration is a disease most common in the second half of adult life, of which the onset is usually insidious and the course progressive. The lesions in the nervous system consist of a primary demyelination, commencing in the centre of the white columns and affecting the long fibres first and most, and the short intersegmental fibres which lie close to the grey matter last and least. Neuroglial condensation follows very slowly upon the demyelination. The posterior and lateral columns of the spinal cord are early affected, and it is to the affection of both these columns that the term "combined degeneration" alludes. The clinical features are usually strikingly distinct, in that subjective sensations, such as tingling, numbness and burning, occurring usually at the periphery of the limbs, are early, obtrusive and persistent, and are accompanied or followed by the development of a paraplegia which may be of a spastic, or a flaccid and ataxic, or of a mixed type, according to the degree of affection of the lateral and of the posterior columns in each case, and the degree of involvement of the peripheral nerves. We owe to Risien Russell, Batten and Collier the first complete pathological and clinical account of this disease.

In the late stages of the malady, the paraplegia tends to become complete and of the flaccid type, with loss of the deep reflexes. Anæmia accompanied by a peculiar "buscuit-like" discoloration of the skin is present in all cases at some period of the disease, with the exception of some few of the cases of short duration. This anæmia tends in every case, if life is prolonged, to develop into a pernicious anæmia which is typical, both clinically and pathologically.

Ætiology.—First met with in the third decade of life, the malady becomes increasingly frequent until a maximum incidence occurs in the sixth decade, while cases commencing in the seventh decade are not uncommon. The sexes are equally affected. Familial incidence in this disease, as also in pernicious anæmia, has been recorded by Hurst, Piney and others.

Pathology.—The essential lesion is demyelination of the axis cylinders, and subsequent degeneration of the latter, in the posterior and lateral columns of the cord. It has also long been known that some degeneration of peri-

pheral nerves occurs in this disease, and recently Carmichael and Greenfield have confirmed that the same process of demyelination seen in the cord is to be found in the peripheral nerves. At first the myelin sheath swells and later disintegrates. This change first occurs in the lower dorsal region of the cord, and is first seen in the centre of both posterior columns, and soon afterwards in the centre of either lateral column, as small areas of a darker and more translucent appearance than the normal white matter. It is only at an early stage of the disease that the anatomical picture is strictly one of postero-lateral degenerations, for soon after, spots of degeneration appear on either side of the anterior median fissure and in other parts of the antero-lateral columns. The degenerated areas increase in size centrifugally, coalesce with one another, reach the surface of the cord and eventually involve the whole of the white matter of the cord as seen in transverse section, with the exception of the narrow zone of short internuncial fibres which everywhere clothe the grey matter. This "annular or ferrule-like" degeneration in the lower dorsal region is highly characteristic, and occurs in no other disease.

From its starting-point in the lower dorsal region the degeneration spreads upwards and downwards in the white columns of the spinal cord, and for this reason the term "funicular myelitis" was applied to it by Henneberg. This extension depends upon the occurrence of small isolated spots of degeneration in the posterior, lateral and antero-lateral columns, which increase in size and thus join the area previously degenerated. The degeneration tends to extend upwards indefinitely, and in severe and advanced cases has been found as high as the internal capsule in the pyramidal tract.

The lesions of the white columns entail the usual secondary degenerations, both ascending and descending; but these occur late, and are often much less obvious than might be expected from the severity of the local lesions. The destruction of the axons by the local lesions also causes a series of retrograde changes in the corresponding nerve-cells, and tigrolysis, vacuolation, shrinking and neurophagy may be conspicuous, especially in the cells of Clarke's column and in the cells of Betz, which gave origin to the pyramidal fibres. Occasionally the disease is entirely confined to the posterior columns of the spinal cord. The muscles are conspicuously wasted in the later stages, and the muscle fibres show great diminution in size and poor striation. There is not any considerable increase of the muscle nuclei, and little or no fibrosis occurs.

Blood.—In a few instances, anæmia has been absent throughout, the hæmoglobin content and the cytology being normal; this has occurred chiefly in cases which have run an acute and fatal course in a few months. Usually the blood shows an anæmia of varying severity; the hæmoglobin ranges from 35 to 75 per cent., the lower of these figures being common; the colour index is usually above the normal, and may be as high as 1.6. Macrocytosis is present. Anisocytosis, poikilocytosis and polychromatophilia are common. Normoblasts are often numerous and megaloblasts may be found in numbers. A relative lymphocytosis is almost always present, and may reach as much as 55 per cent. This change occurs early, and is helpful in the confirmation of the diagnosis of the nervous disease.

A careful investigation of the blood-changes at various stages of the disease and of the post-mortem findings in a large series of cases has proved beyond any possible doubt that the blood-changes in every case are identical with

those met with in the various stages of pernicious anæmia, and that a typical post-mortem picture of pernicious anæmia occurs frequently in subacute combined degeneration. The cerebro-spinal fluid presents no abnormalities either as regards albumin, sugar or cells.

The early writers believed that the anæmia was the essential part of the disease, and that the degenerations in the nervous system were the result of vascular changes consequent upon the anæmia. This view is negatived by the facts that some cases progress to a fatal issue without any evidence of anæmia, and that in others the nervous manifestations may become severe long before any anæmia is evident; and, most importantly, no case has been recorded in the literature, nor has one occurred in the very large series examined by the writers, in which the nervous manifestations developed in a patient already under observation for anæmia.

The experimental evidence and the clinical and pathological features of the disease suggest, therefore, that the anæmia and cachexia and the degeneration of the nervous system are not dependent the one upon the other, but that they are the concomitant but not necessarily synchronous results of one and the same cause, which is deprivation from a product of gastric digestion in the presence of hydrochloric acid which is subsequently stored in the liver, and which is essential to the normal formation of the erythrocytes. Hurst and others have shown that achlorhydria is present in nearly all the cases.

Symptoms.—In a large majority of instances the symptoms appear insidiously and without any exciting cause. Sometimes the onset is more rapid, and may be preceded by severe gastro-intestinal symptoms such as vomiting, diarrhœa, jaundice, malaise and pyrexia. In a few cases the onset has been so rapid as to suggest the diagnosis of acute myelitis, and in one of these which was under our observation and pathologically verified, two attacks of temporary paraplegia has preceded the onset by 8 and by 4 months respectively.

The cardinal signs may be summarised as follows: peripheral subjective sensations, which occur early and are remarkably obtrusive, are complained of in the periphery of the limbs in most cases, but may occur in the perineum, neck and back of the head and in the tongue. Sensory loss is found, which commences upon the limbs with peripheral "stocking and glove" distribution, and reaching on to the trunk ascends in segmental distribution. Astereognosis occurs in the upper extremities. Paraplegia may be (a) flaccid from the first, with loss of deep reflexes; (b) spastic, remaining spastic throughout (rare); (c) spastic, changing to flaccid paralysis with loss of the deep reflexes. The first of these three clinical types is the commonest, and it provides almost all—if not all—the cases which respond favourably to treatment. Both forms of paraplegia are accompanied by marked ataxia. Girdle sensations, lightning pains, fixed pains, gastric crises, exaggeration of superficial reflexes, are all encountered. Sphincter paralysis is late. Loss of sexual power is early. There are muscular wasting and lowering of electrical excitability of general distribution in the paraplegia region. Anæmia, which may be absent throughout or may become apparent at any period in the course of the disease, is conspicuous at the time of the onset of the nervous symptoms in about one-half of all cases.

Peripheral subjective sensations are so constantly the earliest symptom, discomforting to the patient and so persistent, as to form a most distinctive

feature of the disease. These sensations are variously described, but tingling and numbness are the most common. Creeping sensations, smarting, burning, icy coldness, tightness and pain are all common. They are usually felt first upon the tips of the fingers and toes, and subsequently spread up the limbs. A girdle sensation is the rule, and it is sometimes painful.

Sensory loss first appears at the periphery, and spreads up the limbs like the sensory changes of a polyneuritis, waning in severity as the base of the limb is approached. In time it reaches the abdominal wall, but rarely extends as high as the thorax. Earliest to diminish, and first to disappear, are the postural modes of sensibility and vibration sense; tactile sensibility may also be severely affected; but thermal and cutaneous pain sense are usually least affected. A marked and almost constant feature of the disease is the tenderness of the calf and plantar muscles to pressure. Such tenderness is not usually found in association with a spinal cord lesion, and its presence is one of the reasons for regarding the peripheral nerve changes already referred to as of importance in determining the symptomatology.

In the common *flaccid type* of paraplegia, weakness and unsteadiness of gait are commonly preceded by the paræsthesiæ and sensory changes described above. There is at first a ready fatigue on exertion, a dragging of the feet and an ataxy of gait when tired, and also aching pains in the muscles of the legs. Examination reveals some weakness, especially of flexion and dorsiflexion in the legs, the tenderness already mentioned, and diminution or loss of the knee-jerks, and loss of the ankle-jerks. Sooner or later an extensor plantar response betrays the development of lesions in the lateral columns of the cord; but at first the plantar response may be of the flexor type, and when this is the case, the nervous clinical picture is not readily distinguishable from that of polyneuritis. In some cases it is doubtful if the distinction could be made were it not for the accompanying abnormalities in the blood and gastric secretions.

In this clinical type, of course, some of the signs may be the expression of a posterior column lesion, and it is known that in a few cases the spinal cord lesion is confined to this column. In the less frequently seen *spastic type*, the clinical picture is one of a predominating lateral column lesion in the cord. The case may pass through the stage of paraplegia-in-extension to the final one of paraplegia-in-flexion, with extremely painful flexion spasms, loss of sphincter control, and the development of bedsores. In some such cases, the limbs may become flaccid, and the tendon-jerks disappear before contracture ensues. But whether this change from spasticity to flaccidity occur or not the clinical type is one which progresses ruthlessly and does not respond to treatment.

Sudden exacerbations of the symptoms may occur at any time, and these are commonly associated with malaise, pyrexia, vomiting or other signs of gastro-intestinal disturbance and by an increase in the anæmia, as if there had been a sudden increase in the condition, which is responsible both for the anæmia and for the spinal degeneration. As the disease advances, the paraplegia involves more and more of the trunk, progressing upwards. In some cases the upper extremities are affected early, and may even be the first regions to show signs of the disease. In the course of time the paraplegia becomes complete, with great wasting of the muscles and reduction of their faradic excitability.

The paraplegia does not, as a rule, reach the upper limits of the region supplied by the cervical enlargement of the spinal cord, and even in the most severe cases the condition of the upper extremities is one of partial paralysis, most marked in the periphery and associated with considerable wasting of the muscles of the hands and forearms. In addition to the muscular wasting, there is usually conspicuous wasting of the subcutaneous fat. In late stages of the disease the general bodily wasting becomes extreme.

Dysuria generally appears when the paraplegia becomes pronounced. It does not often occur in the early stages of the malady, and sometimes its appearance is delayed until remarkably late. When once established, it does not show any tendency to improve with treatment. Finally, the control of the rectum and bladder becomes completely lost.

Soft translucent oedema of the extremities and trunk is frequent, especially when the anæmia is severe, and is dependent upon the anæmia and upon the impaired innervation of the paraplegic region.

General mental deterioration, mild delirium, drowsiness and torpor frequently occur at any stage of the disease, and are referable to the anæmia and the metabolic disturbance, and possibly also to widely spread cell changes in the cerebrum. General convulsions have been reported in a few cases.

Dimness of vision is common when anæmia and debility are severe. Papilloedema of slight degree is sometimes met with, and doubtless in relation to the anæmia. Optic atrophy has been reported in a good many cases. Small retinal hæmorrhages are not uncommon. A minor degree of nystagmus is the rule, and may depend upon the involvement of the cervical spinal cord, all lesions of which seem to be regularly associated with nystagmus, or this may be attributed to affection of the cerebellum, for changes in the Purkinje cells of this organ have been repeatedly found. Herpes is not infrequent. It may occur anywhere, and has several times affected the distribution of the trigeminal nerve. A hæmorrhagic lesion of the sensory ganglion has been found.

Although anæmia is one of the most characteristic features of subacute combined degeneration, since it occurs in every case of long duration at some time or other, and since it is sufficiently striking as at once to suggest the diagnosis in at least two-thirds of all the patients when they first come under observation for nervous symptoms, yet it may be absent throughout the course of the disease in a rapid case, and its appearance may be delayed until several years after the disease of the nervous system is manifest. The anæmia in almost every case is identical in every respect with pernicious anæmia. Of those cases in which the blood picture is not typical, nearly all show megalocytosis, with a relative lymphocytosis and a high hæmoglobin index, as do early cases of pernicious anæmia, and it may be said with certainty that the longer the patient survives, the greater the likelihood of typical pernicious anæmia developing. The spleen has been enlarged in many cases, and the marrow of the bones is typical of pernicious anæmia, as may be also the iron reaction in the liver and the changes in the myocardium and other muscles. As in pernicious anæmia, the tongue is clean, and this occurs so regularly that any appearance of furring of the tongue may justly be said to exclude the diagnosis of this disease. Fractional test meals show an absolute achlorhydria, or a relative achlorhydria, in the same proportions

as do cases of pernicious anæmia. The colour of the skin is often peculiar and striking, even when anæmia is not severe, and is best described as "biscuit-coloured." A bright malar flush upon this yellowish biscuit-coloured background gives a characteristic and vivid facial aspect in the earlier stages of many of the cases. The symptoms and signs common to all anæmic states, breathlessness, headache, cardiac and venous murmurs and œdema, are commonly present, but hæmorrhages are uncommon. Syncopal attacks may occur. Irregular pyrexia is almost invariably present at some period in the course of the disease, and this quite apart from fever-producing complications, such as cystitis and bedsores. In the later stages progressive emaciation is constant, and if life be prolonged it becomes extreme.

Diagnosis.—In the earliest stage, and before the appearance of any definite sign of organic spinal disease, there may be such disability as to suggest the diagnosis of functional paraplegia. When organic signs appear, it is especially from disseminated sclerosis, spinal tumour and tabes dorsalis that the diagnosis has to be made. The preponderance of the peripheral subjective sensations, and the presence of a florid complexion with anæmia, should always suggest the diagnosis. Slight spastic ataxy is the common clinical picture of subacute combined degeneration, of disseminated sclerosis and of spinal tumour. Peripheral sensations and peripheral numbness are not features of disseminated sclerosis, and the presence of peripheral sensory loss should always challenge that diagnosis, whereas diplopia, nystagmus, transient amblyopia and intention tremor are not early symptoms of subacute combined degeneration. Spinal tumour is especially distinguished by a sharp line of sensory loss, transverse to the axis of the body, which does not spread up from below in slow fashion.

When subacute combined degeneration commences with flaccid ataxy and loss of the deep reflexes, the distinction must be made from tabes dorsalis. The extensor plantar reflex, which is almost always present in the former disease and which is rare in early tabes, the entirely different distribution of the sensory loss in the two diseases, the loss of power in subacute combined degeneration, and the results of the examination of the blood and cerebro-spinal fluid for syphilitic reactions and of the latter fluid for lymphocytosis, are important aids in the differential diagnosis.

In the well-developed stages of the disease, its recognition presents no great difficulty. Attention is quickly attracted by the conspicuous anæmia and biscuit-coloured skin. Following a period of slight paraplegia, often lengthy, the steadily increasing paralysis of the lower extremities, with perhaps sudden exacerbations, producing complete and lasting helplessness, the characteristic distribution of the sensory loss which spreads upwards towards the cervical region, the severe lightning pains, the irregular pyrexia, the anæmia and the relatively late onset of sphincter trouble serve to separate this disease from other forms of paraplegia. The change from the spastic to the flaccid type of paraplegia with loss of the deep reflexes and persistence of the extensor response, which occurs in some of the cases in the late stages, is highly characteristic.

It is also necessary to bear in mind the strikingly close resemblance which the disease we are considering may bear to polyneuritis. The differentiation may in the early stages depend chiefly, if not wholly, upon the examination

of the blood and the result of a fractional test meal. But, sooner or later, the appearance of an extensor plantar response will indicate the presence of a cord lesion. On the other hand, in the spastic type, the presence of muscular tenderness in the legs is a strong indication in favour of subacute combined degeneration.

Course and Prognosis.—The duration of the disease varies within wide limits, but the rapid downhill progress, ending in death within a few weeks or months which was formerly common, is now exceptional. Conflicting claims are made as to the possibility of cure, and it is probable that some at least of the difference of opinion depends upon a failure to appreciate that in the common flaccid type of the disease some at least of the signs and disability may be due to a peripheral nerve lesion and not to degeneration within the cord. This is certainly the clinical type which responds most favourably to liver, stomach, iron and thyroid therapy; while all but the very slightest cases of the spastic type—in which the important lesion is plainly in the lateral columns of the cord—fail to respond to the most intensive therapy.

In the flaccid type, appropriate treatment—if given early enough—will effect considerable restoration of muscular power, of co-ordination and of sensation. Usually, however, paræsthesiæ in the legs and feet, and sometimes in the fingers, remain. The restoration of lost knee-jerks is rare, and that of lost ankle-jerks still rarer. Vibration sense may be partially restored, and very occasionally an extensor plantar response may disappear. It is yet too early to say how enduring these improvements may be. It is clear that for most sufferers, the prognosis of subacute combined degeneration is far better now than it was formerly, but the enthusiasm that would speak of cure as within reach, or prevention of nervous lesions in cases of pernicious anæmia when treated sufficiently early, as certainly possible, has still to justify itself. It is not shared by the neurologist.

Treatment.—Whatever the degree of anæmia present, intensive liver treatment is essential. Daily parenteral injections of the chosen preparation is necessary until the red cell count reaches the five million figure, or as near it as possible. Even this may be insufficient to secure improvement in the nervous symptoms. Indeed these require more and longer continued liver treatment than does the anæmia. The spastic type of case responds badly, but the more numerous flaccid cases may make remarkable recoveries if taken in hand before the malady is too advanced. Previous to the introduction of liver feeding we found that thyroid extract had a remarkable effect in removing the anæmia, and that it could be tolerated by patients suffering from subacute combined degeneration in very large doses, even as much as sixty grains a day. The more advanced the stage of the disease is, the less result may be expected from any form of treatment. Any suppurative condition of the body should be energetically treated. Every care should be taken to delay the advent of bedsores and cystitis. When present, these are often amenable to treatment in the early stages of the disease and in less acute cases, but in the more acute cases and in the later stages they are inevitable and the bodily vitality is too low for any reparative process to take place. Lightning pains and other pains are relieved by such analgesics as aspirin, acetanilide, amidopyrine, phenazone, etc. Reflexor spasms are among the most troublesome of the symptoms, since their frequent occurrence

denies sleep to the patient, and they are most important factors in the occurrence of bedsores. The remedy which seems to have most effect in checking these spasms is barbitone.

MOTOR NEURONE DISEASE

Synonyms.—Progressive Muscular Atrophy; Amyotrophic Lateral Sclerosis; Chronic Bulbar Palsy.

Definition.—A disease of gradual onset which may develop at any age from puberty onwards, and in which the anatomical findings consist invariably, whatever be the clinical picture, of three orders—(1) a progressive degeneration, shrinkage and disappearance, cell by cell, of the upper motor neurones or cells of Betz in the ascending frontal convolution, with consequent degeneration of the corresponding fibres in the pyramidal tracts; (2) a similar atrophy, cell by cell, in the lower motor neurones with corresponding degeneration of motor fibres in the peripheral nerves and atrophic degeneration of the muscles innervated by the affected cells; (3) a diffuse atrophy of the white matter of the spinal cord, the posterior columns conspicuously excepted.

A most mysterious feature of the disease is the non-correspondence between the anatomical findings and the symptomatology. In the first place, though the upper motor neurone lesion is constant, many cases run their course without the slightest external evidence that the pyramidal system is involved.

The clinical picture is one of gradually oncoming weakness and disability, due either to atrophy of the muscles from the lower motor neurone lesion, in which case the paralysis is flaccid and atrophic, or to spastic paralysis of the muscles from the upper motor neurone lesion, in which case the paralysis is spastic without atrophy, or to the combined lesion of both upper and lower motor neurones, in which case the paralysis is both spastic and atrophic, and the muscular atrophy never becomes complete. Fibrillary twitchings of the muscles are always present, and form an important diagnostic feature. Any of the skeletal muscles may be affected from the ocular muscles to those of the feet.

The clinical aspect varies greatly according as the incidence of the palsy is upon the muscles supplied by the brain stem, or upon the muscles of the trunk and limbs, and again, according as the atrophic element or the spastic element is present alone, or as both coexist in the same region or in different regions of the body.

The following are the usual clinical types, but it must be borne in mind that every transition between these types may be met with:

(A) With incidence upon the muscles supplied from the brain stem; Progressive bulbar paralysis: 1. Pure atrophic bulbar paralysis. 2. Spastic atrophic bulbar paralysis. 3. Pure spastic bulbar paralysis.

(B) With incidence upon the muscles of trunk and limbs: 1. Pure atrophic type—(a) local and slowly progressive; (b) general and rapidly progressive. 2. Spastic atrophic type; amyotrophic lateral sclerosis—(a) the spasticity and atrophy are coincident in the same muscles; (b) the atrophy affects the upper limb and the spasticity the lower limb. 3. Pure spastic type. This is more commonly seen as an early stage of amyotrophic

lateral sclerosis, where the spasticity of the lower extremities precedes the atrophy of the upper extremities by some months or years.

(C) Mixed bulbar and spinal forms.

Ætiology.—The earliest age incidence has been at 12 years, and several cases have been recorded which developed the disease at that age. As age advances the incidence of the malady becomes more frequent, until it attains a maximum between the ages of 30 and 40 years, after which there is a slow decline. It does not commonly commence in advanced age, but one case has come under the writers' observation which commenced at the age of 77 years. Males are affected three times as frequently as females, but in the cases occurring before the age of 25 years, the females predominate. Heredity only rarely influences the disease. The question of the relation of trauma to the causation of this disease admits of no decisive answer. In any given case it is impossible to establish a relationship, but some observers have recorded examples of a close sequence of injury and onset of the disease, and they regard the two as in the relation of cause and effect. We know of no pathological process by which a peripheral injury may set up within the central nervous system a selective neurone degeneration. If such there be, it has yet to be discovered. A series of cases of injury to the cervical spine associated with the signs of relatively minor injury to the fourth and fifth segments of the cervical cord which have been recorded by Walshe and Ross, raise the possibility that some cases of so-called progressive muscular atrophy are traumatic in origin, but are not at the outset or in their subsequent development genuine cases of motor neurone disease (cf. p. 1725, Section on Compression of Rapid Onset). Syphilis seems to be in definite causal relation with some of the cases. A positive Wassermann reaction, both in the blood and in the cerebro-spinal fluid, is found in a much larger proportion of the cases than give any history of syphilitic infection. Further, quite a number of instances of the supervention of a typical progressive muscular atrophy in cases of tabes has been observed and recorded at the National Hospital, London. It has been freely stated that progressive muscular atrophy of syphilitic origin differs from the non-syphilitic forms in its lack of symmetry and in its course; but this certainly does not hold good for very many of the cases which show a positive Wassermann reaction. In a large majority of all cases of progressive muscular atrophy, no causal factors whatever can be discovered.

Pathology.—To the naked eye, a cross-section of the spinal cord may show some diminution in size of the ventral horns. The essential lesion is a primary degeneration of the cells of the ventral horns of the spinal cord and in the homologous motor nuclei of the brain stem, namely, the hypoglossal, facial, trigeminal and oculo-motor nuclei. Coupled with the degeneration of the lower motor neurone, is a degeneration of the upper motor neurones of the pyramidal system. In the ventral horn cells the degeneration is evidenced by a gradual shrinking in size of the cells, which lose their dendrites and become oval or spherical in shape. The Nissl bodies slowly disappear, and only in rare and rapid cases is definite chromatolysis seen. The nuclei dwindle and become irregular and distorted.

The dorsal and lateral horns are almost invariably intact, but degenerative changes are sometimes seen in the cells of Clarke's column. The affection of the motor nuclei of the brain stem in the bulbar cases is in every way similar

to that of the ventral horns. The degeneration of the motor nerves which take origin from the degenerate ventral horn cells, often proceeds *pari passu* with the degeneration of the cells. But in some cases this is conspicuously and very mysteriously not the case.

The affected muscles are soft and toneless, and the muscle fibres are found irregularly degenerated, bundles of normal and of degenerating fibres, until the atrophy is complete, being found side by side. The characteristic change is shrinkage of the affected fibre to a calibre much less than normal. As is usual in all slow tissue degenerations, fibrosis and local arterial disease accompany the atrophy of the muscle fibres.

The pyramidal neurones (cells of Betz), which characterise the precentral cortex, undergo a degeneration very similar to that of the ventral horn cells, but with this difference, that the earliest structural changes are found in the most distal part of the pyramidal fibres, and that subsequently these fibres die back towards their cells of origin in the cerebral cortex. The degeneration of the upper motor neurones never proceeds to the complete destruction of anything like all the pyramidal fibres.

The pathological nature, therefore, of progressive muscular atrophy is a widely scattered degeneration of nervous elements not even confined to the motor systems, though these are in the main affected, since the afferent spino-cerebellar tracts are constantly found degenerated, from some unknown cause.

Symptoms.—The following description of the clinical features is based upon an analysis of 500 cases which have come under observation at the National Hospital, London. The onset is in most cases very gradual, but it may be more rapid, and severe incapacity may result in the course of a few months. In rarer cases, a severe degree of paralysis may develop in the course of a few days, and in such cases it is not uncommon to see the most remarkable temporary improvement. The nature of the onset, as a rule, indicates the course which the malady will pursue. A very slow onset is followed by a very slowly-advancing disease, often interrupted by long stationary periods, whereas the more rapid the commencement, the quicker will be the advance and the sooner will a fatal issue occur. Accompanying and sometimes preceding the onset, and not infrequently conspicuous during the early states of the disease, are certain sensory symptoms which, from the confusion in diagnosis they may cause and from the scant attention which has been paid them in descriptions of the malady hitherto, deserve emphasis. These symptoms are confined to the regions where the wasting first appears, and consist in a subjective feeling of stiffness and uselessness, much increased when the limb or the body is cold. Or there may be dull aching pains, intermittent neuralgic pains which may be severe, or a sensation of coldness or numbness which may be intense. Painful cramp in the muscles which are about to be affected is comparatively common.

The *muscular wasting*, which constitutes the most characteristic feature of the disease, may commence in any group of the skeletal muscles whatsoever. It may be first manifest in such rare situations as the facial muscles, intercostal muscles, muscles of the back and abdominal muscles. The commonest situation is in the muscles of the upper limb, where the distal (intrinsic muscles of the hand) or the proximal muscles (deltoids, spinati, etc.) are first affected in about an equal number of cases. In the hand, the muscles

of the thenar eminence are the first to waste, and this is followed by atrophy of the hypothenars, of the lumbricals and of the interossei with the usual flattening of the palm, exposure of the flexor tendons in the palm from loss of the bulk of the lumbricals, hollowing of the interosseal spaces and a tendency to the "griffin's paw" attitude of the hand. The *main en griffe* is never so marked in this disease as in paralysis of the ulnar nerve, syringomyelia, etc., because the wasting soon affects the long flexors of the fingers, and further contractures of the affected muscles are not well marked in progressive muscular atrophy. As the wasting spreads to the muscles of the forearm, the flexors are usually affected before the extensors.

When the upper arm is primarily affected the wasting is first seen most often in the deltoids, whence it spreads upwards, involving the spinati and the muscles attaching arm to scapula, and arm and scapula to trunk. Among these muscles some tend to escape the atrophy relatively, or to be affected much later than others, and these are the triceps, the latissimus dorsi, the lower half of the pectoralis major, the levator anguli scapulæ and especially the upper half of the trapezius, which for this reason was called "*ultimum moriens*" by Duchenne. In the limbs the wasting always commences in one limb, but soon spreads to the corresponding limb of the opposite side and tends ultimately to become symmetrical. The attention of the patient may be first drawn to his malady by the altered appearance produced by the atrophy, and this is more common when the commencement is in the hands, where the subcutaneous tissue is thin and the region constantly in view. Or the disability consequent upon the weakness may be noticed first, and this is always the case where the commencement is in the bulbar muscles, and usually also where the muscles of the legs, proximal muscles of the arms and trunk muscles are first involved. Lastly, the fibrillation may be so marked as first to attract notice.

The *loss of power*, which accompanies the muscular wasting, is, as a rule, commensurate with the wasting, and does not become absolute until the atrophy is complete. To this rule, however, there are two very important exceptions. In the first place, when the affected muscles are both tonic from the upper motor neurone lesion and atrophic from the ventral horn-cell lesion—the tonic atrophy of Gowers—the loss of power is always much greater than can be accounted for by the degree of wasting present. It is a remarkable and entirely unexplained fact that when this tonic atrophy is present the muscles never completely waste, whereas in flaccid atrophy they waste completely, if the patient survives sufficiently long. When the disease commences with initial flaccid paralysis without wasting, it is usually rapid in its course, any temporary improvements notwithstanding. This initial flaccid paralysis without wasting, especially if it improves temporarily, may give rise to great difficulty in diagnosis, for it generally occurs in one limb only, and its rapid development, and in some cases a conspicuous improvement, may give rise to the impression of a gross organic lesion of the ventral horn or ventral roots, and to hopes of recovery which are falsified later.

The disability which progressive muscular atrophy produces in the limbs is always much more marked when the limbs are cold, and conversely. There may be an appearance of vasomotor paralysis, redness, blueness and some swelling of the periphery, but this seems to occur much more as the result of the continual pendent position of the hands, when the muscles, which

flex the elbow and which raise the shoulder, are affected, than as the result of any definite vasomotor palsy. In the regions where the muscular atrophy is apparent, the fat and subcutaneous tissues also waste slowly and progressively, and in all but the rapidly progressive cases this wasting is conspicuous.

Next in order of frequency to initial wasting in the upper extremities comes the incidence of the disease upon the muscles concerned in facial expression, articulation, mastication and deglutition, and in lesser degree upon the muscles of phonation; and the disease may be confined to these muscles throughout the whole of its course. From the widely different clinical picture resulting, and from the fact that all these muscles are supplied from the brain stem and upper two segments of the spinal cord, this form of the disease has borne the name of "progressive bulbar paralysis," or "labio-glossopharyngeal paralysis." Here the wasting commences in the intrinsic muscles of the tongue and spreads thence to the orbicularis oris, to the extrinsic muscles of the tongue, pharynx and larynx, to the muscles of mastication and, eventually, but in less degree, to the facial muscles generally; but only in rare cases are the oculo-motor muscles affected.

The intrinsic muscles of the palate, the constrictors of the pharynx, the intrinsic muscles of the larynx, and the muscle of the oesophagus are little affected. This seems at first an anomalous and astonishing fact, considering how great and important are the troubles with deglutition in bulbar paralysis. But the anomaly disappears at once when one considers that the muscles which are concerned with buccal deglutition are the muscles of the tongue, those forming the floor of the mouth, including the mylohyoid and the digastric, the muscles which raise and lower the jaw, and those of the lips. Further, the muscles which are most important in pharyngeal deglutition are those which raise and lower the hyoid bone and larynx as a whole, and these are the stylohyoid and stylopharyngeus, the palatoglossus and palatopharyngeus, the geniohyoid, thyrohyoid, sternohyoid, sternothyroid and omohyoid. All these muscles are early and severely affected in bulbar paralysis; and when they fail, the intrinsic muscles of the palate are unable to shut off the naso-pharynx, the constrictors of the pharynx are entirely unable to perform the act of deglutition, and the intrinsic muscles of the larynx—though phonation is never lost—are unable, since the larynx is unfixed by the extrinsic muscles, to modulate the tone of the voice. The very active pharyngeal reflex and the well-known great difficulty in using the laryngoscope on account of spasm of the pharynx in the subjects of this disease, are very good clinical evidence that the pharyngeal constrictors are not affected.

The earliest physical sign of bulbar paralysis is the loss of the finer movements which are essential for correct articulation, and consequently a slurring dysarthria develops and increases, and the consonants become less and less distinct until they are inaudible. The failure of the palate to close upon the posterior pharyngeal wall begets a nasal element in the voice. Later, the patient becomes unable to interrupt his blast at any of the stop positions, and his utterance becomes a long, moaning, monotonous, inarticulate sound. His phonation remains, but he cannot alter its pitch nor divide it into parts of speech, except by taking a fresh breath. The orbicularis oris is early affected, and the lips lose their firmness and become thin,

and as they weaken, the unopposed retractors of the angles produce a wide, straight mouth, both at rest and in emotional action. Whistling and pursing up the lips become impossible, and ultimately there is much dribbling of saliva, for this can neither be retained by the lips nor swallowed. The tongue shows fine fibrillation, and as it wastes its point, becomes rounded, and is protruded with difficulty. Its surface becomes dimpled and faceted, and in the end consists solely of the covering mucous membrane, the glands and the fibrous tissue, and lies motionless in the floor of the mouth, resembling a crinkled mushroom. The muscles of mastication all become affected. The bite becomes feeble and the mouth cannot be opened against resistance. In the late stages the jaw drops and the mouth is constantly open. The combined weakness of tongue and buccinators makes it very difficult for the patient to keep his food between his teeth in mastication, and often he aids his disability by digital pressure upon the cheeks. Nasal regurgitation is not uncommon. The difficulty in swallowing is greatest with fluids, for these require quick action, and is next greatest with lumpy solids, for these necessitate powerful action. It is least with food of a porridge-like consistency, and this should be carefully borne in mind in feeding the patients.

The other muscles of the face are affected later and to a much less severe degree than is the orbicularis oris. It is as if there were a physiological selection on the part of the disease for the nervous mechanism subserving mastication and deglutition. Still in the majority of cases there are bilateral general facial weakness and wasting which, with the peculiar mouth and dropping jaw, produce a characteristic facies which can be instantly recognised. If the upper facial muscles are tested by raising the eyelid with the finger against resistance, invariably they will be found to be weak. Only in very rare cases does the atrophy extend to the oculo-motor muscles. As in the paralysis of the limbs, so also in bulbar paralysis, concomitant signs of both upper motor neurone and of lower motor neurone lesion may exist. When such tonic atrophy of the bulbar muscles is present, the symptomatology and clinical appearance are the same as have been above described for the simple atrophic form, with the exception that the jaw-jerk and the other muscle-jerks of the bulbar region, which are absent in the latter condition, are brisk in the tonic-atrophic form. And, further, it must be remembered that the additional element of spastic paralysis adds greatly to the degree of the paralysis as a whole.

In less common cases of progressive bulbar paralysis the upper motor neurone lesion alone is in evidence, and the bulbar paralysis is purely spastic. Here the symptomatology as regards articulation, deglutition, etc., is the same, and the facial aspect identical with that of the simple atrophic and tonic-atrophic forms. The muscle-jerks are brisk. The appearance of the tongue, however, is quite different; it is smooth, narrow, stiff and drawn into a narrow compass by the spasm of the muscles composing it. It appears too small for so large a mouth. There is no fibrillation, and the muscles are nowhere wasted.

The muscles of the back of the neck, the splenius, complexus, etc., are not uncommonly the first muscles to be affected with the wasting of progressive muscular atrophy. There is increasing difficulty in extending the head, which drops forward, causing a characteristic attitude, which is associ-

ated with a constant overaction of the frontales which raise the brows to clear the line of vision when the head is dropped forward, so giving rise to a permanently furrowed brow. The loss of substance in the muscles of the back of the neck, together with the dropping forward of the head, causes the lower cervical and upper dorsal spines to stand out in undue prominence, and to give an appearance approximating to that of an angular curvature.

Primary affection of the lower extremities is much less common than that of the upper extremities, bulbar region or neck muscles. The anterior tibial and peroneal muscles are usually attacked first, and less commonly the quadriceps. The clinical type is that of flaccid atrophy in most of the cases. Tonic atrophy, which is so common in the upper limbs and in the bulbar region, is rare in the legs. Spasticity without atrophy from the upper motor neurone lesion alone is very common in the lower extremities. It forms a characteristic part of the frequently occurring clinical type of amyotrophic lateral sclerosis, in which the upper extremities or bulbar region are affected with atrophic paralysis, and the legs with spastic paralysis. In this common combination the atrophic paralysis is usually of the tonic and much less frequently of the simple flaccid type. Spasticity from the upper motor neurone lesion may develop in the lower extremities long before there are any signs of atrophic paralysis elsewhere from the lower motor neurone lesion, and such cases present the physical signs of a primary lateral sclerosis. Therefore, it cannot be too strongly borne in mind that any case presenting the features of a primary lateral sclerosis in an adult may eventually prove to be one of progressive muscular atrophy.

Wherever the site of commencement of progressive muscular atrophy may be, it invariably spreads to other regions, sometimes slowly and with periods of arrest which may last for years, sometimes with remarkable rapidity. The manner of spread is usually in terms of the contiguity of the affected elements in the nervous system; but it is sometimes in terms of the physiological association of the muscles, as is commonly seen in the bulbar forms of the malady. When the disease is definitely installed the appearance of fibrillation, in any muscles otherwise unaffected, is a sure sign that atrophy will shortly commence in those muscles.

According to the method of advance shown by the disease, cases of progressive muscular atrophy fall into two groups which it is important to distinguish. In the first group, the atrophy spreads locally and slowly and remains confined to one region of the anatomy during most of the course of the malady. These cases are always of the simple atrophic type, and they usually survive a long time. Such cases, however, tend to become general just before the end. In contrast with the local type of the affection is the group in which the manifestations, commencing locally, spread within a comparatively short time to many parts of the anatomy, or even become universal. The spread may be very rapid, and the end may occur in a few months, or it may be slower; but it is unusual for any of the cases forming this group to survive for more than eighteen months. This group comprises (1) the generalised cases of simple flaccid atrophy; (2) all the cases of amyotrophic lateral sclerosis; and (3) most of the bulbar cases.

Fibrillation is a most important symptom of the disease, and is an associate of the muscular atrophy. It precedes the wasting of the fibres, and is a sure

herald of the advent of wasting in this disease. It ceases to occur when the muscle is completely wasted, and is not seen when the atrophy is not progressing. On account of the importance of fibrillation as a diagnostic sign of progressive muscular atrophy it is important here to consider those other conditions in which it is met with clinically. It occurs in syringomyelia and in peroneal atrophy, but only when the muscular atrophy is progressing; and, therefore, it is only an occasional symptom in either disease. It is often very marked in cases of interstitial neuritis (sciatica, etc.). It occurs in a most magnified and conspicuous form in certain conditions of gastro-enteritis, and is presumably due to an intoxication, and to this form of fibrillation the term "myokimia" has been applied. It is not met with in polyneuritis, poliomyelitis, myopathy, nor in the common gross lesions of nerve trunks, nerve roots or spinal cord.

The *electrical reactions* of the affected muscles vary according to the degree of degeneration. Since normal and degenerate fibres are stimulated side by side in the affected muscle, there will be some lowering of the response to faradism with a tendency to a polar change. This is known as the "mixed reaction," and it is common to all diseases in which muscle degenerates fibre by fibre. Faradic excitability lessens as more of the muscle fibres degenerate, and when degeneration is complete all electrical excitability is lost. The excitability of the affected muscles to direct mechanical stimuli, such as percussion, is increased so long as any living muscle remains.

Contractures are conspicuous by their absence in this disease, which is thus strongly contrasted with peroneal atrophy and some other muscular atrophies. If the atrophy becomes complete in a whole limb the end-result is that the limb is flail-like and without contracture.

Mental alterations are constantly present in the cases in which the bulbar region is affected. Emotional instability and hyperexcitability are the usual change. The patient is easily excited to tears or to laughter by trivial causes, and when so excited cannot control his expression of emotion. He himself feels little joy or grief during the paroxysms of laughing or crying.

Sphincters.—In the majority of the cases these are not affected, but every now and then dysuria in any of its forms occurs, and it may occur early in the course of the malady, and it may be severe. Loss of sexual power is very common.

Reflexes.—The superficial reflexes are modified in this disease, on the one hand by spasticity, when this is present, and, on the other, by the muscular atrophy which may prevent response in the affected muscles. The pharyngeal reflex in bulbar cases is usually brisk, notwithstanding the statement to the contrary, which most antecedent writers upon this subject have recorded; but the response is not the normal response, involving all the muscles concerned in deglutition, for these are atrophied and paralysed; it is confined to the constrictors of the pharynx and the muscles of the palate, with the feeble co-operation of such of the somatic bulbar muscles as are still able to act. The plantar reflexes are usually of the extensor type when the legs are spastic; but this does not always obtain, for there may be definite rigidity of the legs with brisk knee-jerks and foot-clonus with a persistent flexor response. Similarly, the abdominal reflexes do not disappear so constantly or so early as is the case in disseminated sclerosis, for example, and they may persist when the legs are markedly spastic and extensor plantar

responses have appeared. The muscle-jerks disappear from the affected region in simple atrophic cases *pari passu* with the wasting of the muscles. In cases of tonic atrophy they are everywhere increased, even in regions where the atrophy is severe, and in this type of the malady they never disappear. The same increase of the muscle-jerks occurs in the purely spastic cases.

Diagnosis.—The malady has to be distinguished from the many conditions in which progressive weakness and wasting of the muscles occur, from those in which muscular wasting and spasticity are conspicuous clinical features, and lastly from other diseases, in which bulbar symptoms are early evidenced. Peroneal muscular atrophy very closely resembles progressive muscular atrophy, in that slow wasting and fibrillation of the muscles are the chief clinical features. The points which distinguish the two conditions are that peroneal atrophy is often a familial disease, and is apt to commence in childhood, when it is unusual for progressive muscular atrophy to begin. The location of the atrophy is peculiar, and when well marked in the periphery of all four limbs, as is common in this disease, cannot be confused with progressive muscular atrophy since the latter disease never has this distribution. Syringomyelia is easily distinguishable by the early and striking loss of pain and temperature sensibility. Cervical rib not uncommonly produces atrophy of the intrinsic muscles of the hand, and, though this is usually confined to one hand, it may be bilateral. Further, it is exceptional for the atrophy to involve all the small hand muscles simultaneously, or equally. It picks out the *opponens pollicis* first and most severely, and is not uniform for all the hand muscles, as in progressive muscular atrophy. Pain in the distribution of the eighth cervical and first dorsal roots, and some loss of sensibility, may be present. The atrophy remains local, and is never accompanied by fibrillation. The abnormal rib is easily discoverable on radiographic examination. It must be borne in mind that cervical ribs are not uncommon, and that their presence does not necessarily prove the cause of atrophy of the hand muscles, for cervical ribs may be present in progressive muscular atrophy, in syringomyelia, and in any other disease.

Arthritic muscular atrophy occurs in the regions of joints which show easily recognisable disease. Fibrillation does not occur, nor are there alterations in the electrical excitability of the wasted muscles. Dystrophia myotonica is at once separated from progressive muscular atrophy by the myotonus, when this latter symptom is present. When myotonus is absent, the characteristic wasting of the sternomastoids, and of the muscles of the thighs, the age of the subject, and sometimes the presence of cataract should suggest the diagnosis.

Lesions of peripheral nerve trunks may be diagnosed by the history of a local cause, by the discovery of a palpable local lesion upon the course of the nerve, and by the confinement of the atrophy to the distribution of one particular nerve, while open pain and sensory loss occur in that same distribution.

Lesions of the nerve roots, and especially those produced by pachymeningitis and by neoplasm in the vertebræ may cause signs and symptoms so closely resembling those of the more rapid forms of progressive muscular atrophy, as to render correct diagnosis very difficult. Such a lesion in the cervical region, for example, may give rise to wasting of the hand and fore-

arm muscles, and a spastic condition of the legs, resembling exactly a condition of amyotrophic lateral sclerosis, without deformity or rigidity of the spine, and without pain or sensory loss. In such cases of difficulty the course of a little time will bring the advent of the conclusive symptoms of a local pressure lesion. It is important in this connection to remember that pressure upon the spinal cord results in hyperalbuminosis of the cerebro-spinal fluid, and if the lesion causing the pressure is syphilitic, there is likely also to be lymphocytosis in that fluid, neither of which conditions is found in progressive muscular atrophy.

Diagnosis is most difficult in those cases where spasticity in the limbs is the first sign of progressive muscular atrophy to appear, and where such spasticity precedes the appearance of any muscular atrophy by a long time. If it be clearly borne in mind that spastic paralysis may be the earliest, and for a time the only sign of progressive muscular atrophy, and that among the many diseases of the nervous system, which commence with the same clinical picture of spastic paralysis, a certain diagnosis cannot be made until further distinguishing signs appear, error will be avoided. The importance of the examination of the cerebro-spinal fluid in doubtful cases cannot be too strongly emphasised.

Course and Prognosis.—The nature of the disease is to progress, and to extend its area of invasion until a fatal issue is reached. The progress may be rapid, and the end may be reached in a few months, or it may be slow, and many years may elapse before death occurs. The local types of slow onset are the most gradual in their development, and these are often characterised by periods of arrest in the progress of the disease. The generalised simple atrophic type of the disease is the most rapid, especially when it commences with severe initial flaccid paralysis without atrophy.

In the bulbar types of the disease, and in amyotrophic lateral sclerosis, the course is for the most steadily progressive. Every type will show, however, upon occasion, exacerbations and remissions, and the exacerbations are the most important, and in the bulbar types may bring about the end in a few hours. Of particular interest are rapid extensions of a flaccid paralysis, which may occur in a few hours, and which resemble, and indeed are identical with, onset of the disease with initial flaccid paralysis without atrophy, which has been already described. Whatever type of the disease be present, it tends in the end to spread and to become general.

Involvement of the respiratory muscles or severe bulbar symptoms, and the pulmonary complications which may accompany either condition, may bring about the fatal issue. It is usual, however, for death to occur in a manner which is common to so many degenerative nervous diseases, a rapid increase of the paralysis is associated with an increasing lethargy, which soon deepens into a rapidly fatal coma. It is uncommon for death to occur from intercurrent maladies. The average tenure of existence after definite signs are present is under 1 year in the generalised flaccid type, and it may be as short as 2 months. Bulbar symptoms are not generally survived for more than 12 months. Localised cases of simple atrophy may live for many years. Some of the patients in whom a positive Wassermann reaction is found improve, and the disease is sometimes arrested by antisiphilitic treatment.

The progressive character of the disease renders the prognosis grave in

every case. There are some cases occurring in middle life, which are presumably cases of progressive muscular atrophy of local distribution and slow onset and course, which become finally arrested or even improve ; but in the absence of pathological verification the true nature of such cases is open to doubt.

In amyotrophic lateral sclerosis the average duration of life is not more than 2 years from the onset. When bulbar symptoms are present the average duration is under 2 years. In the generalised cases the average duration is under 1 year. Widely spread fibrillation in muscles, which are neither weak nor wasted, is the constant herald of generalisation, and renders the immediate prognosis serious. In cases where syphilis is present the prognosis is more favourable, and there is even a possibility of arrest and improvement if energetic treatment of the associated condition is provided. Rapid extension of the weakness, the advent of bulbar symptoms, involvement of all the respiratory muscles, and especially general asthenia and drowsiness are the signs which usher in the fatal result.

Complications.—By far the most common complication which is met with in cases of progressive muscular atrophy is the presence of some syphilitic lesion of the nervous system, and this may be of any nature, both local or general. *Tabes dorsalis*, associated with progressive muscular atrophy, is not uncommon. General paralysis of the insane has been noted in a few cases, as has also paralysis agitans.

Treatment.—For the most this malady seems to be entirely uninfluenced by any treatment that has hitherto been adopted. Even where syphilis is a factor in the causation, although appropriate treatment for these conditions has been applied, and improvement and even arrest may result, it is no rare thing to see no amelioration, and in some cases such treatment seems actually to hasten the progress of the disease. Recently, dramatic claims have been made in respect of vitamin E (tocopherol acetate), given in doses of 3 mgs. thrice daily. This is said to arrest wasting and weakness and in early cases to effect rapid improvement. In a brief experience since this claim was made the present writer has been wholly unable to confirm it, and it must be accepted with the greatest reserve. It remains, therefore, to secure favourable conditions of life for the patient, and to maintain the general health in as perfect a state as possible. Massage and passive movements are useful as giving bodily comfort to the patient, and satisfying him that something is being done for him. In bulbar cases, the dysphagia must be aided by avoiding liquids and solids, and by serving all the articles of diet in pulvaceous form. Salivation, which is so troublesome in this condition, may be greatly helped by the administration of hyoscine by the mouth.

PERONEAL MUSCULAR ATROPHY

Synonym.—Charcot-Marie-Tooth Type of Muscular Atrophy, Neuritic Type of Muscular Atrophy.

This is an absolutely distinct and peculiar form of muscular atrophy, with a frequent tendency to occur in several members of the same family. It usually commences in mid-childhood, and after progressing for some twenty years or less, comes to a final arrest. The atrophy always commences

in the intrinsic muscles of the feet, and is throughout strictly distal in distribution. The muscles of the face and trunk and the proximal muscles of the limbs are never affected. The atrophy leaves a peculiar elastic fibrosis in the affected muscles, so that the incapacity caused by this disease is much less than in any other form of muscular atrophy of like degree. Sensibility is often slightly affected, and there may be deep sensory loss. The essential morbid anatomy is a primary neurone atrophy of the anterior horn cells and of some of the afferent neurones in certain regions of the spinal cord.

Ætiology.—The disease usually commences between the fifth and tenth years of childhood, but it may appear as late as the fourth decade of life. Males and females are both affected. Heredity plays an important part in the incidence, although isolated sporadic cases are not uncommon. It may exhibit every type of inheritance. The malady often occurs in families, and has been traced through five generations; it may skip a generation and then reappear.

Pathology.—The anterior horn cells of the affected regions show a slowly progressive atrophy and disappearance, with corresponding atrophy of fibres in the peripheral nerves. The cells of Clarke's column show signs of degeneration, as do also some of the fibres of the posterior columns of the spinal cord, and especially those of the postero-lateral column. Slight degeneration in some of the fibres of the pyramidal tracts is usually found. The affected muscles show a simple atrophy of the muscle fibres, indistinguishable from that seen when a motor nerve is divided. There is a simple shrinking of the fibres, which stain progressively and more and more deeply with hæmatoxylin, lose their striation, and finally disappear. Secondary fibrotic changes accompany the atrophy, together with sclerosis of the arteries of the muscle.

Symptoms.—Muscular atrophy dominates the clinical picture of this malady. It is strictly distal in distribution, and this feature will serve to distinguish peroneal atrophy from any other form of muscular atrophy. This is to say it does not affect one particular muscle, but the distal ends of all the muscles below a certain level on the limb, leaving the proximal ends of the muscles normal, and it advances up the limb inch by inch, the separation of the wasted portion of the muscle from the normal portion being always transverse to its length. In other words, the muscle fibres seem to waste in terms of the length of the spinal axons which supply them. The wasting commences always in the intrinsic muscles of the feet, and hollowness of the instep and thinness of the feet, together with retraction of the toes and the difficulty which the pes cavus so produced entails in fitting boots, first draws attention to the disease. As the process advances, the lower segments of the anterior tibial, peroneal and calf muscles become affected, and the limb is subsequently involved until the lower third of the thigh is reached, at which stage the disease is invariably arrested. This slow spread of the atrophy from the distal towards the proximal portion of the limb, gives rise to a most unique and characteristic feature in the appearance of the legs at the several stages of the disease. As an example, the complete atrophy of all the muscles below the middle and a well-developed musculature in the upper half of the leg, give rise to the inverted "fat bottle" calf. When the atrophy has involved the lower third of the thigh, the lower end of the femur, bare of muscle and covered only by skin and tendons,

contrasts strongly with the well-developed muscles of the upper thigh, and causes the thigh to resemble an inverted champagne bottle.

Some years after the atrophy has become marked in the lower extremities, and in the usual run of cases just before the age of puberty, the intrinsic muscles of the hands and first those of the thenar and hypothenar group begin to waste, and this wasting may extend as high as the middle of the forearm. It must be borne in mind that the disease may become arrested at any period of its spread, and especially that the upper extremities often escape altogether. With the exception of the lower part of the thighs, the proximal segments of the limbs do not become involved, and the muscles of the head, neck and trunk remain unaffected.

The affected regions of the muscles waste absolutely, and leave a very elastic fibrous tissue. The electrical excitability in the wasted regions becomes first lowered and then lost, and, in the earlier stages, may show a mixed reaction, in which there is lowering of excitability to faradism, with a tendency to an inverted polar reaction. Fibrillation of the muscles is an important sign. It is seen only when the disease is progressing, and in the muscles which are obviously wasting. It is never general, as in some cases of progressive muscular atrophy. And since peroneal atrophy is at times advancing and at other times stationary, fibrillation may be in one case conspicuous and in another never seen. It disappears entirely when the progress of the malady becomes finally arrested, and is, therefore, useful as a clinical indication of active advance of the disease. Contractures always occur, and from the nature of the distribution of the atrophy are necessarily confined to the feet and the hands. In the feet, pes cavus with retracted toes is the rule; but sometimes, and in some stages of the disease, the feet and toes may be dropped and the feet inverted. The sphincters are unaffected. The ankle-jerks are diminished or lost in proportion to the wasting of the calf muscles. In the final arrested stage they are usually lost. The knee-jerk is always retained and is usually brisk. The plantar reflexes are usually lost early so far as any response in the foot is concerned, but some response in the upper thigh muscles, upon stimulating the plantar region, often remains. Pain, tenderness and cramp are entirely absent. Conspicuous loss of sensibility is uncommon, but slight loss of deep sensibility, loss of the vibration sense and relative tactile loss, may often be detected upon careful examination: but in rarer cases all forms of sensibility may be severely affected, or even entirely lost. Perforating ulcers may be met with upon the soles of the feet, and are explained by the thinness of the feet and their deformity, which, coupled with the clumsiness of the use of the feet, lead to the formation of severe corns which break down into perforating ulcers. Loss of sensibility also is a factor in their production.

The most striking of all the clinical features of peroneal atrophy is the comparatively slight disability which the wasting of the muscles and consequent paralysis, and even the sensory loss, when present, cause.

Course.—The course is irregularly progressive for a number of years only, and the advance of the disease ceases usually in the third decade of life. Exacerbations of the weakness are likely to be followed in every case by considerable improvement, owing to the secondary fibrosis in the muscles.

Diagnosis.—Peroneal atrophy in the early stages is easily confused with progressive muscular atrophy, in that wasting of muscles and fibrillation

are the conspicuous features. The onset usually in childhood and the fact that the feet are affected first, the peculiar distal distribution and the presence of any familial incidence, are important. But the only distinction which is absolute is the distribution, for progressive muscular atrophy may begin in childhood and peroneal atrophy may not appear till after middle life, and often familial relations are absent in the latter malady. In the course of time the diagnosis always becomes clear, for progressive muscular atrophy never keeps to the classic distribution, nor is it followed by the peculiar fibrosis which characterises peroneal atrophy.

Dystrophia myotonica when commencing in the peroneal muscles may for a time closely simulate peroneal atrophy. The presence of the least sign of myotonia, the involvement of the face and the atrophy of the sterno-mastoids, will establish the diagnosis.

The usual forms of myopathy are at once separated from peroneal atrophy by the distribution of the muscular weakness and wasting, which in the former group of maladies is conspicuously upon the face, trunk and proximal muscles of the limbs, and in the latter upon the distal muscles. Peripheral neuritis is more rapid in its onset, and is apt to be associated with marked sensory disturbances, both objective and subjective, and the paralysis is in terms of individual muscles, which is not the case in peroneal atrophy.

Treatment.—The general health should be carefully maintained, and the nutrition of the affected muscles aided by the application of massage. Care must be taken, on the one hand, to avoid over-fatigue of the affected muscles, and, on the other, to ensure such regular exercise as is compatible with their capacity. Bicycling, for example, since it employs chiefly the thigh muscles, is a better form of exercise for these patients than is walking. In no circumstances should tenotomies be performed for the deformity of the feet, for such measures tend to destroy the effect of the conservative fibrosis, so essential to the production of a useful limb. The use of heavy mechanical supports is to be avoided above all things. Light, well-fitting boots, so as to interfere as little as possible with the exercise of the damaged muscles, are essential.

PROGRESSIVE SPINAL MUSCULAR ATROPHY OF CHILDREN

Synonym.—The Werdnig-Hoffmann Disease.

This is a malady of the first year of infancy, often incident upon several children of the same parents, and characterised by the gradual development of progressive muscular weakness and atrophy, which affects the proximal muscles first and most, increases to a complete paralysis of trunk and limbs, and finally affects the bulbar muscles. The disease is invariably fatal in from a few weeks to several months. The most striking pathological changes are a progressive degeneration and disappearance of the ventral horn cells of the spinal cord, and of their analogues in the brain stem.

Ætiology.—In some of the cases the paralysis is noticeable at the time of birth, and the disease is obviously of pre-natal development. In others the children are quite healthy at birth, and the disease develops some time during the first year of life, and most frequently within 8 weeks of birth. Though sporadic cases may be met with, yet in the majority of instances

several children of the same mother are affected. Both the pre-natal cases and the post-natal cases may be met with among the children of the same mother. The sexes seem to be equally affected. No maternal ill-health during pregnancy has been noticed, and nothing is known about any other aetiological factor.

Pathology.—The most extensive changes are found in the ventral horn cells throughout the spinal cord and brain stem, and at many levels no normal cells whatever are to be seen. Tigrolysis, swelling and glassiness of the cells, extrusion of the nuclei, disappearance of the dendrites, shrinking of the cells and final disappearance is the sequence of the changes. Degeneration of the anterior roots and of the peripheral motor nerve fibres consequently occurs. These changes are not confined to the lower motor neurones, for in our cases examination by the Marchi method showed extensive degeneration throughout the posterior columns of the cord, indicating that lower sensory neurones were also considerably affected.

The muscles show intense degeneration with hypertrophy of some fibres and atrophy of most of the fibres, waving, moniliform shape, hypernucleation of the spindles, general nuclear increase and fibrosis.

Symptoms.—In the cases which are pre-natal, the malady is noticed at the time of birth on account of the tonelessness, flaccidity and the pooriness of movement in the trunk and proximal muscles of the limbs. In the post-natal cases there is a gradual onset of similar weakness and flaccidity in the trunk first, and in the limbs afterwards, which usually commences within six weeks of birth, but which may not appear until towards the end of the first year of life. The weakness seems always to be least marked in the periphery of the limbs, where curious, slow, involuntary movements of the fingers and toes have been noted in a good many of the cases. The paralysis is followed by a rapid and extensive wasting of the muscles, accompanied by occasional fibrillary twitchings. Since these children are not only well nourished, but often put on much fat during the illness, wasting of the muscles may not be apparent on inspection or palpation. It can, however, immediately be detected by radiography, which distinguishes sharply between fat and muscle.

As the malady progresses the trunk muscles become completely paralysed, the intercostal muscles being always paralysed before the diaphragm. The limbs become progressively weaker, and, lastly, bulbar paralysis supervenes in those cases where death has not already occurred from respiratory paralysis. The reaction of degeneration is present in the affected muscles. Sensibility may be unimpaired; but in several of my cases there has been conspicuous loss of pain sensibility over the limbs and trunk. The sphincters are unimpaired until the very last stages of the disease. The superficial and deep reflexes are lost. The ocular muscles have not been affected, and intelligence is preserved throughout.

Diagnosis.—The peculiar and striking features of the disease make the diagnosis easy, if the symptomatology be known. Amyotonia congenita presents the same helplessness and flaccidity of trunk and limbs as does the Werdnig-Hoffmann disease, and further resembles it in being sometimes congenital, and sometimes having an onset very early in life. In amyotonia congenita, however, the paralysis is not complete, and it tends to improvement and not to progressive increase. Contractures also occur, which are

not found in the Werdnig-Hoffmann disease, and, lastly, the definite spinal cord changes of the latter malady are not found in the former.

Course and Prognosis—The course is invariably progressive, and is more rapid the earlier in life the disease commences, and it is most rapid of all in the pre-natal cases, which are usually fatal within a few weeks. With an onset some weeks after birth, life is usually continued for several months, and a few cases have been reported with an onset towards the end of the first year, in which death has been delayed until the third or fourth year.

Treatment.—No treatment is known to influence the course of the malady.

LESIONS OF THE PERIPHERAL NERVES

LOCAL LESIONS OF NERVE ROOTS AND NERVE TRUNKS

PHRENIC NERVE.—This nerve supplies the diaphragm. Paralysis results most often from disease of the spinal cord, but the roots may be implicated in disease of the spine, and the trunk may be injured, in its course through the neck and thorax, by wounds or tumours. Bilateral paralysis occurs in lesions of the cord and spine, and in alcoholic, diphtheritic, saturnine and other forms of peripheral neuritis. Other causes usually affect one side only. When the diaphragm is completely paralysed, the normal inspiratory protrusion of the upper part of the abdomen disappears, or is replaced by retraction of this part with each inspiration. During rest, so long as the lungs are healthy, the respiratory rate does not increase, but if bronchitis or pneumonia arises as a complication, or if the patient exerts himself, the diminished reserve of respiratory power is seriously felt. When one nerve only is affected the diaphragm does not descend on that side. This is rarely detected by observation of the abdominal movements, but is easily seen on the X-ray screen. It produces no discomfort.

THE LONG THORACIC NERVE.—This nerve supplies the serratus magnus muscle. When all the fibres of this muscle contract, the scapula moves upwards, forwards and outwards. It contracts with the pectoralis major in the action of pushing forward the point of the shoulder and in the rapid-thrust movement. It also assists the deltoid in raising the arm. When it is paralysed alone, the position of the scapula at rest is unaltered, but if the trapezius and the rhomboids are paralysed as well the scapula drops, and its lower angle is displaced inwards. Paralysis of the serratus magnus is best demonstrated by causing the patient to hold the arms outstretched before him. The arm is not raised so high on the affected as on the normal side, because the scapula is not fixed and the deltoid works at a disadvantage. Viewed from behind the deformity is characteristic. The vertebral border of the scapula stands out prominently and the hand can be pushed between this bone and the thorax—"winged scapula." On raising the arm from the side, there is difficulty in attaining the horizontal position, but the winging of the scapula is less apparent.

The nerve may be damaged by carrying heavy weights on the shoulder, by falls or blows on the shoulder, and by continued muscular effort with the

raised arm. The nerve may be injured alone in gunshot wounds, but as a rule it is associated with lesion of the brachial plexus. In addition, a serratus magnus palsy may develop suddenly in an otherwise healthy person after exposure to cold, or as part of a rare reaction to the administration of serum or antitoxin. In the cases caused by compression, severe neuralgic pains in the neck precede the onset of paralysis. Recovery is always very slow and the defect may be permanent.

BRACHIAL PLEXUS.—The brachial plexus may be injured by stabs in the neck, by penetrating missiles, by dislocation of the shoulder or fracture of the clavicle, or by pressure of a tumour, aneurysm or cervical rib. Further, the nerves may be torn by forcible dragging on the arm in accidents or during delivery. In most cases the lesion is partial and the symptoms conform in the main to one of the following types.

Upper plexus paralysis (Erb's palsy).—This results from an injury to the fifth and sixth cervical roots. The muscles paralysed are: biceps, deltoid, brachialis anticus, supinator longus, supraspinatus, infraspinatus, rhomboideus, subscapularis, clavicular portion of pectoralis major, serratus magnus, latissimus dorsi, teres major. The arm cannot be flexed at the elbow (flexors of forearm), nor raised and abducted (deltoid). The movements of the wrist and fingers are not impaired. Adduction of the arm is weak (pectoralis major), and rotation is feeble or absent (spinati). On attempting to oppose the shoulders, the scapula on the affected side passes farther from the middle line (rhomboideus). The hand of the affected side cannot be placed on the buttock of the sound side (latissimus dorsi).

The reaction of degeneration is often complete in the deltoid and flexors of the forearm and nearly so in the spinati. It is usually incomplete in the other muscles. Sensation is diminished or lost along the outer border of the whole limb immediately after the injury, but improvement sets in rapidly. For some time the patient experiences pins and needles and burning sensations in the affected area, which last longest in the thumb and index finger. The biceps reflex is lost. In this form the tendency to complete recovery is great. As a rule all the symptoms disappear completely in from 6 months to 2 or 3 years. Weakness persists longest in the deltoid and supinator longus.

Lower plexus paralysis (Klumpke's palsy).—This results from a lesion of the eighth cervical and first dorsal roots, or of the common trunk of the median and ulnar nerves. The intrinsic muscles of the hand and the flexors of the wrist and fingers are paralysed, and the inner border of the forearm and hand is anæsthetic. When the roots are damaged, sympathetic fibres may be implicated with the production of myosis, narrowing of the palpebral aperture, enophthalmos and alterations in sweating on the face, neck, arm and upper part of the chest, on the affected side.

Middle plexus paralysis.—This form of paralysis is a common result of gunshot injuries of the plexus. It affects the muscles supplied by the musculospiral and circumflex nerves—posterior cord. As the nerve to the latissimus dorsi arises from the same trunk, this muscle is often paralysed as well. In addition to these simple types, more complicated paralyses occur, in which various parts of the plexus are injured together.

In *paralysis of the inner cord of the plexus*, atrophy is confined to the intrinsic hand muscles, and the sensory loss is confined to the hand.

Lesions of the brachial plexus show a remarkable tendency to spon-

aneous recovery. In many cases recovery is complete in 6 months to 2 years, in others it is partial, and some muscles remain paralysed.

THE MUSCULO-SPIRAL NERVE.—Owing to its long course, its position in relation to the humerus, and its peculiar vulnerability to compression, paralysis of the musculo-spiral nerve is one of the commonest peripheral palsies; although it is a mixed nerve, containing sensory, motor and vasomotor fibres, the symptoms of an injury are almost entirely motor. In the upper arm the nerve supplies the triceps and the anconeus, in the forearm the supinators, the extensors of the wrist and fingers, and the extensors and long abductor of the thumb.

Symptoms.—Injury to the nerve is followed by dropping of the wrist and fingers. The wrist and the first phalanges are flexed. The flexion is limp and easily reducible.

When the lesion is in the axilla the whole of the *triceps* is paralysed, and extension at the elbow is lost. Occasionally in wounds of the posterior aspect of the arm the nerves to the triceps are injured, whilst the main trunk escapes. The patient is then able to extend the arm powerfully by means of the anconeus, but if he is made to raise the elbow as high as possible with his fingers on the point of the shoulder, extension of the bent forearm is impossible.

In most cases the nerve is injured in the middle third of the arm and the triceps escapes, but the supinator longus and all the extensor muscles in the forearm are paralysed. Partial paralyses, such as are seen in lesions of the median and ulnar nerves, are very rare. The *supinator longus*, so-called, is not a supinator. Its action is to flex the forearm, whilst the hand is in a position intermediate between pronation and supination. Paralysis of this muscle is detected by the absence of contraction when the pronated forearm is flexed against resistance. Owing to paralysis of the *supinator brevis* supination is abolished. During the movement of flexion of the forearm the biceps acts as a supinator, during extension the external rotators of the shoulder, but feebly.

Paralysis of the *extensors of the carpus* abolishes both extension and lateral movement at the wrist. The flexors of the carpus play no part in lateral movements. The *extensors of the fingers* extend the first phalanges only. Extension at the distal joints is carried out by the lumbricals and interossei. Paralysis of the *extensors and long abductor of the thumb* renders abduction of the thumb and extension of the phalanges impossible. On attempting to abduct the thumb, it passes no farther than the radial border of the hand. In some cases, the second phalanx of the thumb can be feebly extended by the muscles of the thenar eminence.

Many muscles not supplied by the musculo-spiral work at a disadvantage when the extensors are paralysed. These defects must not be mistaken for signs of injury to other nerves. Owing to the flexed position of the hand the grasp is feeble, but if the wrist is extended passively the grasp is improved. The patient cannot make a fist properly, as the thumb does not oppose the index finger and the fingers cannot be flexed into the palm, until the thumb has been moved aside by the sound hand. The movements of the interossei in abducting and adducting the fingers are also feeble while the wrist is flexed, but are much stronger when the hand is resting flat on a table with the wrist and fingers extended. The complete reaction of degeneration is

often found in all the paralysed muscles from the onset. Atrophy becomes obvious in a month or two. Its extent and severity give important evidence for prognosis.

Sensory disturbances.—Subjective symptoms are rare. In a few cases, paræsthesiæ are felt on the posterior aspect of the forearm and on the dorsal aspect of the thumb. They are of brief duration, and are commoner with partial than with complete lesions. Severe causalgias are almost never seen in lesions of this nerve. Sensibility to light touch, superficial pain and temperature is impaired over a small area on the radial border of the hand, including the proximal joints of the thumb and first two fingers. The defect is often very slight, and is only discovered on very careful examination. Deep sensibility is rarely affected. Considering the extensive distribution of the external cutaneous branch of the musculo-spiral nerve, it is rather surprising that the sensory disturbances are so slight, when the nerve is injured above the origin of this branch.

Recovery.—It might be thought that recovery would take place in the order of the length of the branches to the various muscles. This, however, is not the case. As a rule the extensors of the wrist recover first, then the extensors of the middle, ring, little and index fingers in this order, next the supinator longus, and the extensors and abductors of the thumb last of all. On palpation of the muscles during attempted extension, contractions can be felt before any movement is produced. Other signs of impending recovery are the disappearance of automatic pronation and of the flail-like drop of the hand, also diminution of automatic flexion of the fingers after passive extension. Recovery of movement is complete when the patient is able to extend the wrist and all the fingers simultaneously or separately. After this becomes possible, restoration of power is rapid.

THE MEDIAN NERVE.—Whilst the clinical individuality of the musculo-spiral nerve is shown in the preponderance of motor symptoms and in the uniform completeness of the paralysis that follows an injury, that of the median is seen in the frequency of partial and especially of painful lesions. Isolated palsy of this nerve is infrequent except as a result of gunshot wounds and other injuries. It may be damaged by repeated violent contractions of the pronator radii teres, as in one of the forms of "tennis elbow."

Total paralysis.—The muscles paralysed are the pronators, the radial flexor of the wrist, the flexors of the fingers except the ulnar half of the deep flexor, most of the muscles of the thenar eminence (opponens, abductor brevis and outer head of the flexor brevis pollicis) and the two radial lumbricals. Stated briefly the symptoms are : inability to flex the phalanges of the index finger and the second phalanx of the thumb ; difficulty in flexing the phalanges of the middle finger ; defective opposition of the thumb. The appearance of the hand in total lesions is fairly constant. The hand inclines to the ulnar side, the index and middle fingers are more extended than is normal, and the thumb lies on a level with the fingers—the ape-hand.

Pronation is incomplete and defective. The patient tries to overcome the defect by rotating the whole limb at the shoulder. Paralysis of the *flexors of the wrist* is seen when an attempt is made to flex against resistance. The tendon of the ulnar flexor alone stands out, and the hand is drawn towards the ulnar side. Even at rest, the flexor tendons are more prominent on the sound than on the affected side.

Flexion of the fingers is good in the two ulnar fingers, though weaker than normal. The index cannot be flexed at all, and the third finger only incompletely. Flexion at the proximal joint is usually good in all the fingers including the index, and flexion at this joint with extension at the last two joints is usually well done by the interossei and lumbricals. If the proximal phalanx of the thumb is immobilised, it will be seen that flexion of the terminal phalanx is abolished, owing to paralysis of the *flexor longus pollicis*.

Paralysis of the *thenar muscles* renders opposition and abduction of the thumb defective. By means of the adductor the thumb can be drawn into the palm, but as the radial fingers cannot be flexed nor the thumb opposed, it is impossible to place the tip of the thumb on the tips of the fingers. Atrophy of the muscles becomes obvious in a few weeks. The outer part of the thenar eminence is flattened, and the bulk of the muscles arising from the internal condyle is greatly diminished.

Sensory disturbances.—In almost every case there is complete anæsthesia to all forms of sensation in the two terminal phalanges of the index and middle fingers. The skin outside this area may be unaffected even in complete lesions, but in most cases sensibility is diminished in the terminal phalanx of the thumb, and to a less extent over the remainder of the radial half of the palm, including the radial side of the ring finger. The stereognostic sense is lost in the outer fingers. This defect, together with the loss of power, renders the thumb and index finger useless, and makes paralysis of the median the most serious single nerve lesion of the upper limb.

Vasomotor and trophic changes.—In many cases the skin in the distribution of the median nerve is red, dry and chapped, and the nails white or purple. It is possible that these changes are due to an associated vascular lesion.

Recovery is extremely slow and is rarely complete. Sensation begins to return before power, but the stereognostic sense is often defective, long after movement in the fingers has returned. The pronator and the flexors of the wrist recover first, then the flexors of the thumb and middle finger. Flexion of the index finger and opposition of the thumb, if it is regained at all, remains defective for several years. In searching for signs of recovery, care must be taken lest some "trick-movement," due to contractions of healthy muscles, is misconstrued. For example, when told to flex the terminal phalanx of the thumb, the patient first over-extends and abducts, and then relaxes suddenly. The terminal phalanx then makes a slight passive movement of flexion, which may be mistaken for true active flexion. Recovery is complete when the patient is able to make a good fist with the fingers flexed well into the palm, and the thumb pressed firmly upon the dorsal aspect of the second phalanx of the middle finger.

PARTIAL LESIONS.—Partial paralysis of the median nerve is much commoner than the complete form.

Motor symptoms.—Flexion of the index finger and opposition of the thumb are most impaired. The flexors of the middle finger and of the terminal phalanx of the thumb may suffer also, but to a less degree, whilst the pronators and the flexors of the wrist often escape entirely.

Sensory symptoms.—Apart from the painful lesions to be mentioned later, sensory troubles are usually slight in partial lesions. Anæsthesia is rare, but sensibility to all forms may be diminished in the areas mentioned under complete lesions.

Vasomotor symptoms.—The skin is often cyanosed in the distribution of the injured nerve, and it may perspire more freely than in healthy parts. These changes are more distinct when the paralysis is complicated by a vascular lesion.

Recovery is naturally more rapid than in complete lesions. The order in which the muscles recover and the tests for complete return of function have been mentioned above.

PAINFUL LESIONS OF THE MEDIAN NERVE.—*Causalgia.*—In many cases the most prominent symptom of injury to the median nerve is *pain*.

Motor disturbances are always present, but are usually slight, the weakness affecting mainly the flexors of the index finger and the thenar muscles.

Vasomotor changes are a feature of this type. In many cases perspiration is diminished over the radial half of the palm, and the skin becomes dry and scaly. In others, perspiration is increased over the median area.

Sensory disturbances.—Pain comes on about a month after the injury, at first as tingling or pricking in the finger-tips and palm, later as a constant severe smarting, dragging or *burning* pain—hence the name *causalgia*. Added to the constant pain, which never ceases day or night, paroxysms occur, in which the pain increases suddenly in intensity. The application of cold water gives temporary relief, and patients often wear bandages or gloves which they keep constantly moistened. Many develop a phobia of dryness. They will not touch dry objects, even with the healthy hand, the sight of another person handling a dry object increases the pain, and any rustling or crackling sound, suggestive of dryness, may bring on a paroxysm.

In severe cases the limb is held flexed at the elbow and wrist, with the hand constantly raised and the fingers extended or hyper-extended. The whole hand atrophies, and irreducible ankylosis occurs with the limb in this position. The skin of the hand is thin and dry. The fingers taper, and the nails are long, brittle, blackened and striated longitudinally. The pain reaches its acme 4 or 5 months after the injury, and then slowly declines, but the limb remains useless. Even in slighter cases, without much deformity, recovery of function is extremely slow, and is rarely complete.

THE ULNAR NERVE.—The ulnar nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the hypothenar eminence, the interossei, the two inner lumbricals, and the adductor and inner head of the short flexor of the thumb. Its sensory area is the ulnar border of the hand, the little finger and the inner half of the ring finger.

Total paralysis.—Paralysis of the *flexor carpi ulnaris* may be detected by palpating the tendons when the wrists are flexed against resistance. The limpness on the affected side contrasts strongly with the firmness on the sound side. Lateral movements of the hand are unaffected, as these are carried out by the extensors.

Paralysis of the ulnar portion of the *flexor profundus digitorum*. In making a fist, flexion of the index finger is perfect and that of the middle finger good, whilst in the ring and little finger it is absent or very feeble. This weakness is best seen when flexion is attempted with the index and middle fingers extended. Even when the fingers can be flexed by the action of the flexor sublimis, the power of resisting passive extension is completely lost in the terminal phalanx of the two ulnar fingers. Paralysis of the *hypothenar* muscles abolishes lateral movements of the little finger, and

diminishes the power of flexion at the proximal joint. Paralysis of the interossei and of the inner two lumbricals leads to the production of the "claw-hand."

The action of these muscles is to flex the fingers at the proximal joints with the distal joints extended. In the "claw-hand" the posture of the fingers is just the opposite of this, namely, extension at the proximal joint with flexion of the distal joints. Although all the interossei are paralysed, the defect is only seen in the ulnar fingers, as the radial lumbricals supplied by the median are still healthy. It is produced by the action of the long extensors, which being now unopposed, over-extend the proximal joints, and by the flexor sublimis which flexes the second joint and draws the distal joint down with it. The clawing of the fingers is greatly accentuated when the nerve is paralysed below the point of origin of the fibres to the long flexors of the fingers. Other features of the "ulnar hand" are atrophy of the interossei and of the hypothenar eminence and persistent abduction of the little and ring fingers. The movements of abduction and adduction are lost in the inner two fingers, and often in the middle finger. Further, these fingers cannot be flexed at the distal joint, whilst the proximal joints are extended.

Paralysis of the *adductor pollicis* and of the inner head of the *flexor brevis pollicis* produces peculiar disturbances in prehensile movements. If the patient is asked to grasp a folded paper between his thumb and index finger, and to resist efforts to remove it by pulling, it will be found that this movement, which is normally very powerful, is grossly defective. He cannot grasp the object beneath the thumb with the second phalanx extended; but presses the tip of the flexed thumb against the outer margin of the index finger.

Sensory disturbances.—In complete lesions, all forms of sensation are abolished in the little finger, and along the ulnar border of the hand. Beyond this there is usually diminished sensibility on the ulnar side of the ring finger, and over a narrow area towards the centre of the hand on both aspects. Spontaneous pains are rare, and vasomotor changes are usually slight.

Partial paralysis.—In partial lesions the same symptoms are found in a less degree. The small muscles of the hand suffer most. Clawing may be slight or absent. Neuralgic pains may be felt in the distribution of the ulnar nerve; but causalgia is never seen in lesions of this nerve alone.

Recovery of sensation is usually complete before movement is regained. The flexor carpi ulnaris recovers first, then the long flexors of the fingers, and last the small muscles of the hand. In these recovery is extremely slow. When recovery of movement is complete the patient can abduct and adduct the middle finger with the palm flat on a table, and he can also scratch the table with the nail of the little finger without moving his wrist.

THE MUSCULO-CUTANEOUS NERVE is rarely affected alone, but is often implicated with the brachial plexus. It supplies the biceps, coraco-brachialis and brachialis anticus. Flexion of the forearm can still be carried out by the supinator longus; but the power of flexion is greatly diminished. Sensation may be diminished or lost along the radial border of the forearm.

THE CIRCUMFLEX NERVE supplies the deltoid and teres minor, and the skin over the deltoid. It may be injured alone in injuries to the shoulder and by pressure of a crutch. The chief symptom is paralysis of the deltoid with almost complete inability to raise the arm.

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INTERNAL CUTANEOUS NERVE—Division of this nerve produces a narrow area of anæsthesia on the inner side of the forearm.

THE LESSER INTERNAL CUTANEOUS supplies the skin on the inner aspect of the upper arm.

In war injuries lesions of the nerves of the lower limb are very frequent; but in civil practice, apart from sciatica, local lesions of these nerves are rare.

THE LUMBO-SACRAL PLEXUS.—The *lumbar plexus* may be damaged by abdominal tumours, and its roots by new-growth or other disease of the vertebræ. In a certain number of cases signs of inflammation of the lumbar plexus are found in association with sciatica or neuritis of the *sacral plexus*.

ANTERIOR CRURAL NERVE (L_2, L_3, L_4).—This is the largest branch of the lumbar plexus. It supplies the psoas, iliacus, pectineus, sartorius, adductor longus and quadriceps femoris. It may be injured alone by fractures of the pelvis or of the femur, by dislocations of the hip, or by implication in wounds, psoas abscesses or new growths.

The most prominent symptoms are loss of power to extend the knee, loss of the knee-jerk, wasting of the quadriceps, and sensory disturbances over the anterior surface of the thigh and inner surface of the leg. The psoas always escapes, unless the plexus itself is also damaged; but flexion at the hip may be imperfect through paralysis of the iliacus. Owing to the rapid dispersion of the branches in the thigh, wounds in this part often cause partial lesions. In these the *nerve to the quadriceps* is most often injured. The resulting paralysis causes serious disability in walking as the knee gives way at every step, especially in going down stairs, and lameness lasts for a long time after complete return of voluntary movement.

OBTURATOR NERVE (L_2, L_3, L_4).—This nerve is rarely damaged alone. It supplies the three adductor muscles, the obturator externus and the gracilis. The symptoms are weakness of adduction and internal rotation at the hip.

EXTERNAL CUTANEOUS NERVE (L_2, L_3).—This nerve supplies an area of skin on the buttock, and through its femoral branch the skin on the antero-lateral aspect of the thigh. As a result of injury, but more often without obvious cause, the skin in the territory of this nerve may show peculiar sensory disturbances, which have been described under the name of *meralgia paræsthetica*. Most cases occur in men. In women it is usually associated with pregnancy. The nerve is tender on pressure at the point where it passes from under Poupart's ligament, and neuralgic pain or numbness and tingling is felt in the skin, which may be slightly insensitive on objective examination or extremely hyperæsthetic, so that the slightest touch causes pain. The symptoms, which are always unilateral, are made worse by walking, and may cause serious incapacity by their persistence and severity. In severe cases the nerve should be excised.

The *sacral plexus* may be damaged by growths or inflammation in the pelvis, by compression during parturition, and by penetrating missiles. It is also often the seat of spontaneous neuritis.

THE GREAT SCIATIC NERVE (L^4, L^5, S^1, S^2).—This nerve supplies the flexors of the leg and all the muscles below the knee. It may be involved in pelvic new growths, or injured by fractures of the pelvis or femur. Next

to the musculo-spiral it suffers in gunshot wounds more often than any other nerve.

Total paralysis.—The foot drops, and the toes point downwards. Walking is possible, but the patient cannot stand on the heel or toes of the paralysed foot. The knee is raised high, but the steppage is not so marked in total lesions as when the external popliteal alone is paralysed. All movement below the knee is abolished. When the wound is in the buttocks flexion of the knee is very weak. The foot becomes œdematous if allowed to hang down. Sweating is often absent on the sole and dorsum of the foot, but is normal on the inner side of the foot, which is supplied by the anterior crural. The skin is dry and thin, and may be scaly. Hyperkeratosis of the sole is common. Subjective sensibility is rarely affected. The skin is completely anæsthetic over the entire foot, except the inner border of the sole and around the internal malleolus. The anæsthesia extends upwards on the postero-external aspect of the calf in its lower two-thirds, embracing the tendo Achillis and external malleolus. Beyond this area of complete anæsthesia there is a wide zone in which sensibility is diminished. The sense of position and passive movement is abolished in the foot and toes. The knee-jerk is present. The ankle-jerk is always lost. Stimulation of the sole may produce a contraction in the tensor of the fascia lata; but there is no response in the foot.

Partial paralysis.—In wounds of the sciatic nerve it often happens that the fibres of the external popliteal alone are wounded, since the sciatic trunk often divides into the internal and external popliteal branches as high as the great sacro-sciatic notch. The symptoms are described below under paralysis of this nerve. In other cases, the fibres of the internal popliteal are damaged either alone, or with some of the fibres of the external popliteal. In this case the outstanding clinical feature is pain of the same nature as that already described in lesions of the median nerve.

EXTERNAL SCIATIC NERVE.—This nerve may be injured as it winds round the fibula by wounds or fractures or by compression of a tight bandage. The paralysis is usually severe, all the muscles being equally affected. The foot is dropped and inverted, and the toes are slightly flexed. Dorsal flexion of the foot, extension of the proximal phalanges of the toes, and abduction of the foot are impossible. The patient can walk, and he can stand on tip-toe, but he cannot run, and walking is made difficult by the foot-drop. Subjective sensory disturbances are usually absent. The skin is anæsthetic over a narrow band which extends from the outer surface of the leg in its middle third, downwards beside the outer border of the tibia, and along the middle of the dorsal aspect of the foot as far as the base of the toes. For an inch or so, on both sides of this band, the sensibility of the skin is diminished. The knee-jerk and ankle-jerk are present. The plantar response is always flexor. Vasomotor changes are slight, and trophic changes are absent.

INTERNAL SCIATIC NERVE.—This nerve is rarely injured alone. It supplies the popliteus, the calf muscles, the flexors of the toes and the intrinsic muscles of the foot. When it is paralysed, the patient is unable to stand on tip-toe, or to extend or invert the ankle, or to flex his toes. Paralysis of the interossei leads to a claw-like deformity of the foot, associated with lowering of the heel and raising of the metatarsus—talipes calcaneo-valgus.

The calf muscles are flabby and the ankle-jerk is abolished. Sensation is lost on the sole, except along its inner border, on the outer border of the foot, and on the plantar surface of the toes. Causalgia, similar to that in paralysis of the median, is very often present.

POSTERIOR TIBIAL NERVE.—This nerve may be injured by a penetrating missile or a deep wound in the calf. Movements of the ankle are unaffected, and anæsthesia is confined to the sole of the foot and heel, or merely to its inner half. The paralysis of the intrinsic muscles of the foot may escape detection, and the lesion may easily be overlooked, especially when the nerve is injured below the origin of branches supplying the flexor longus hallucis and the flexor longus digitorum. The symptoms then are pain in the sole of the foot, anæsthesia on the sole, and paralysis of the plantar muscles.

Treatment of Local Nerve Lesions.—Treatment must depend on the nature and degree of the lesion. During the long period which elapses between the onset of paralysis and the first signs of recovery, even in cases of simple physiological interruption of the nerves, every effort must be made to prevent degeneration of the muscles, to keep the circulation of the limb active, and to prevent the occurrence of contractures and deformities. Massage, movements, electrotherapy and suitable appliances all have their uses. With regard to operative treatment, it must be remembered that more than half the cases of nerve injuries undergo spontaneous cure. It is advisable, therefore, to wait three or four months before an operation is undertaken. If, at the end of this time, the wound is soundly healed and all signs of sepsis have disappeared, and if, as a result of repeated examinations, no sign of recovery has been detected, no harm can be done by exposing the nerve. If it is found to be divided completely, the ends should be "freshened" and sutured end to end. If the nerve is notched laterally, the edges of the notch should be pared and sutured, care being taken to preserve the bridge of uninjured tissue. Sometimes the nerve at the site of the lesion appears as a fibrous, flattened band between two swellings on the nerve. In most of such cases the nerve is completely divided, and the condition calls for resection of this fibrous tissue and end-to-end suture. Another common finding, when the nerve is exposed, is a nodule or cicatricial swelling in the course of a nerve which has maintained its continuity. In these cases the continuity of the nerve should not be interrupted. It should be freed from adhesions, and incised in the long axis of the swelling. All operations which involve grafting of nerves are futile. For an account of the great advances in the technique of the surgical treatment of nerve injuries which have been made as a result of experience gained in the Great War, special treatises must be consulted.

The treatment of painful forms of nerve lesions is extremely difficult. In severe cases external applications and internal medication entirely fail. Simple freeing of the nerve sometimes gives relief. Where this fails, it may be advisable to practise complete division followed by immediate suture. Alcoholisation of the nerve trunk often gives immediate and lasting relief. Under general anæsthesia the nerve is freed, and then injected with 1 c.c. of weak alcohol at a point two or more centimetres above the lesion. This, of course, is followed by motor paralysis; but recovery occurs in about six months.

INTERSTITIAL NEURITIS

Synonym.—Neuro-fibrositis.

Definition.—A malady which commonly attacks the large nerve plexuses or nerve trunks, but which may affect any peripheral nerve trunk, and which is characterised anatomically by an inflammation of the connective tissues which surround and bind together the nerve fibres into the nerve trunks. This fibrositis, which may be local or diffuse in the affected nerves, is the result of the causes of fibrositis in general, and is frequently associated with fibrositis elsewhere, as, for example, when sciatica is associated with lumbago. The symptoms are those of irritation of the nerve fibres, namely, pain in the distribution of the nerve trunk, tenderness of the nerve trunk, muscular fibrillation and cramp. Loss of function of the nerve fibres in the way of loss of sensibility or muscular paralysis is the rarest of events in interstitial neuritis, and is seen only as the result of terminal cicatrization in severe cases. Muscular wasting may occur in severe or in longstanding cases, but it is a general wasting of muscles of the painful region, not confined to the distribution of the nerve involved, and therefore resembling the muscular wasting which is seen in joint disease.

Pathology.—The malady is met with soon after puberty, and is incident chiefly upon the first half of adult life, being unknown in childhood and rare in old age. It is often associated with other forms of fibrositis such as lumbago. Often it arises spontaneously, without external cause; but exposure to cold may directly cause it, as also may injury such as stretching, bruising or wounding of the nerve trunk. Gout and diabetes are well-known clinical associations.

The morbid anatomy is well seen when the nerve is exposed during operative procedures for the relief of the condition. The affected nerve trunk is swollen and pink in colour; the sheath is distended, and droplets of fluid exude when it is incised, and sometimes the nerve is adherent to the surrounding tissues. This inflammatory condition may be local and appear as a pink bulbous enlargement of the nerve trunk, or it may spread widely over a long stretch of the nerve trunk and its branches. When the inflammatory process subsides there may be cicatrization of the peri- and endoneurium. Only in the rarest cases does the morbid process become so severe as to interfere with the more important functions of the nerve trunk with the production of motor and sensory paralysis, and even in these cases complete ultimate recovery is the rule. The local inflammatory condition causes a slight shortening of the nerve trunk, and this causes the affected limb to be held in that position which will keep the nerve trunk most relaxed. It is also the cause of the severe pain which occurs on any movement which stretches the nerve trunk.

Interstitial neuritis is sometimes an associate of arthritis. For example, in arthritis of the shoulder-joint it is not uncommon to meet with definite involvement of the brachial nerves, and again in chronic arthritis of the hip-joint the inflammatory process may extend from the capsule of the hip-joint directly into the contiguous sciatic nerve.

The nerves may affect any of the nerve roots or nerve trunks, and sometimes several of these may be co-involved. When the nerve roots

are affected "radicular neuritis" results. The sciatic nerve is by far the most common seat of the disease, producing the condition known as "sciatica." Next in order of frequency comes the brachial plexus, causing "brachial neuritis," the anterior crural nerve causing "anterior crural neuritis," the upper part of the cervical plexus producing "cervico-occipital neuritis," and the intercostal nerves producing the so-called "intercostal neuralgia."

Symptoms.—These are the same whatever nerve is affected, and consist in—(1) Pain radiating in the area of distribution of the affected nerve, of a dull, aching character with acute exacerbations and often very long-lasting. (2) Tenderness of the affected nerve to pressure and stretching. (3) Subjective peripheral sensations such as tingling, burning or numbness. (4) General wasting of the muscles of the surrounding region with marked hypotonus, not confined to the muscles supplied by the affected nerve and akin to arthritic muscular atrophy. This wasting may reach a very remarkable degree. (5) Increase of the deep reflexes of the limb. (6) Diminution or loss of the deep reflex in the supply of the affected nerve. This is a valuable indication of the severity of the lesion. In a case of sciatica, for example, all the muscles of thigh and leg are wasted, the knee-jerk and the adductor-jerks are markedly brisk, whereas the ankle-jerk, which is in the sciatic supply, is diminished in slight cases and lost in severe cases. (7) The affected limb is held in a characteristic position to avoid stretching of the nerve, and the gait is similarly modified. (8) Trophic and vasomotor changes are not uncommon. (9) Fibrillation is often present.

Diagnosis.—There is sometimes considerable difficulty in the diagnosis of cases of interstitial neuritis on account of the almost identical clinical picture which may occur in the early stages of pressure upon nerve roots or nerves by tumours. The following points are of value in distinguishing the two conditions: The pain of pressure lesions is rarely so severe as that of interstitial neuritis. Tenderness on pressure or stretching of the nerve trunks is absent in pressure lesions. Signs of loss of function—paralysis and sensory loss—come on early in pressure lesions. The most careful search should be made in every case for any possible cause for local pressure, such as primary and secondary neoplasms, spinal tumours, spinal caries and diabetes. To make a diagnosis of interstitial neuritis in the presence of a mammary or testicular carcinoma, removed or not, is to advocate the highly improbable, whatever the symptoms may be.

BRACHIAL NEURITIS.—This form of interstitial neuritis is somewhat rare, and is met with chiefly in women over the age of 35 years. Sometimes it follows injury to the brachial plexus from any violence causing undue separation of head and shoulder. More often it arises spontaneously. The pain, which is often of sudden onset, may be of great severity, and may be at first referred to the region of the plexus itself, the back of the scapula, the axilla, the forearm or the hand, is at first intermittent, but it soon becomes continuous and spreads over the whole upper limb. Tingling and numbness in the hand and trophic changes in the skin and nails of the fingers are the rule.

One of the great difficulties in this malady is that in the upright position the weight of the arm and shoulder carry the shoulder downwards and stretch the inflamed plexus, adding greatly to the pain. Therefore it should be treated with the recumbent position upon the back in bed. Further,

every movement of the hand or arm tends to increase the pain. Splints which keep the arm in the abducted position and the shoulder raised so as to prevent tension upon the plexus are invaluable.

There is little difficulty in diagnosis, the only confusable conditions being arthritis of the shoulder and cervical rib, in neither of which conditions is there any tenderness of the nerve trunks of the plexus.

CERVICO-OCIPITAL NEURITIS.—This condition, which is by no means rare, is characterised by pain in the upper part of one side of the neck, radiating over the branches of the upper cervical plexus, the great occipital being the most common, and the supra-sternal, supra-clavicular and supra-acromial branches less common seats for the pain. The fibrositis not infrequently co-involves the fibrous structures in the region of the articular and transverse processes, giving rise to pain and stiffness of the neck on movement. When the pain is confined to the great occipital distribution alcohol injection is sometimes most efficacious.

SCIATICA.—In few common and familiar maladies is as much difference of opinion as to ætiology, pathology and treatment as in the case of sciatica. The classification suggested by Barnes Burt is probably the most useful. He recognises three types: (i) root sciatica, sometimes called radiculitis; (ii) trunk sciatica; and (iii) referred sciatica, and believes that the ætiology of the three is different in each case. Root sciatica is commonly due to spondylitis, or strain of the lumbar vertebra; trunk sciatica depends upon interstitial neuritis of the nerve sheath; and referred sciatica results from inflammation of some structure (muscle, bursa, joint) supplied by the sciatic plexus. He finds in his own series of cases that root sciatica accounts for 36 per cent. of the total, trunk sciatica for 18 per cent., and referred sciatica for 46 per cent. In previous editions of this book, the description was confined to root trunk sciatica. This account now follows, questions of differential diagnosis being discussed subsequently. It has been said that true sciatica as here defined is never bilateral, and that bilateral sciatic pain is always the result of gross lesions involving the nerve. It is important that this error, which has crept into so many textbooks, should be contradicted, for sciatica is occasionally bilateral, and the sciatica which occurs in glycosurics is usually bilateral.

The malady is not met with in childhood, but it begins to be common soon after puberty, and its incidence is greatest upon early middle life. In the majority of the cases it arises without assignable cause, sometimes injury to the nerve of any nature, as from a twist of the leg, a bruise or a fall is responsible. Only in rare cases does exposure to cold and wet seem to have excited the onset. It is important that the urine should be tested in every case of sciatic pain, for glycosuria is more often found in cases of sciatica than is usually believed. In the case of an apparent sciatica, the possibility of rupture of an intervertebral disk should be borne in mind (cf. page 1773).

Symptoms.—The chief symptom is pain along the course of the nerve or of its branches, and since the sciatic nerve often divides within the pelvis into the great internal and external sciatic branches, the pain may be confined to the distribution of one of these alone. One feature of the pain valuable for diagnostic purposes is that it never reaches above the crest of the ilium, but in this connection it must be borne in mind that fibrositis of the back (lumbago) not infrequently precedes or accompanies the onset of sciatica.

The pain may be partly intra-pelvic, for the sciatic nerve is formed within the pelvis. Where the interstitial neuritis is entirely intra-pelvic, tenderness of the nerve trunk to digital pressure in the thigh and buttock fails as a physical sign. The onset is occasionally sudden, and associated with slight pyrexia and constitutional disturbances as in other forms of fibrositis; but, as a rule, the malady sets in gradually with pain in the buttock, back of the thigh or leg, in movements and in postures which make the nerve tense, or cause pressure upon it. The pain gradually increases in severity. It may be both gnawing and burning and sharp and darting in character. It is usually continuous, with occasional severe exacerbations which occur spontaneously or are excited by movement. Its intensity generally increases at night. The seat of the pain often varies from day to day.

Extreme tenderness of the nerve on pressure is rarely absent, except in those cases where the neuritis is intra-pelvic, and the tender region usually indicates the situation of the lesion of the nerve trunk. Stretching the nerve by extending the knee with the thigh flexed is productive of great pain which may be lasting. The best method of testing the sensitivity of the nerve to stretching is to put gentle pressure with the thumb on to the popliteal space as the patient sits in a chair with the knee bent at a right angle. Sometimes there is considerable tenderness of the muscles. The muscles waste not only in the sciatic supply, but throughout the whole lower limb and buttock, surely from reflex irritation as in arthritis atrophy. Cramp in the leg and reflex spasm are common, and muscular fibrillation is often seen. Paræsthesia in the form of tingling, burning and numbness is the rule; but loss of sensibility only occurs in the rarest and most severe cases, and its presence should always suggest the presence of a pressure lesion. Barnes Burt states that in root sciatica (radiculitis) there is also some rigidity of the lumbar spine, tenderness along the lateral aspects of the spine in this region, and also pain along the distribution of the lateral cutaneous nerve of the leg.

Peculiarities of stance, gait and position arise from the tenderness of the nerve to stretching, that position being assumed by the patient which keeps the nerve slackest. In standing, the weight of the body is placed upon the sound limb, and the other limb is flexed at hip and knee and extended at the ankle, with the toes only resting on the ground. In walking, the patient limps in this same position without straightening his knee or extending his ankle, and in bed he lies with knee flexed and ankle extended.

The knee-jerk is sometimes markedly increased notwithstanding the wasting of the quadriceps, the only exception being when sciatica is complicated with anterior crural neuritis. The ankle-jerk being in the sciatic supply tends to be diminished in proportion to the severity of the neuritis, and in severe cases it is always lost. It may not return after recovery from the original attack.

Slight trophic and vasomotor changes in the periphery of the limb are commonly seen.

In very rare cases the cicatrization which follows the inflammatory process may cause motor and sensory paralysis of all the region below the knee. Such a case came under our care 2 years after the onset, and was explored by Sargent, who found the nerve in the gluteal region densely cicatrised and widely adherent to the muscles. The nerve was freed, and

incised longitudinally in many places, and this patient made a complete recovery. Every degree of severity may be met with from the mildest to the most acute, and from the most rapid lasting but a few weeks to the most chronic lasting 2 or more years.

It is a most remarkable fact that severe sciatica never occurs twice in the same limb. One severe attack seems to free the affected nerve from subsequent liability to the affection, and it is comforting to be able to assure the patient that he will never have the trouble again in the same limb. In a very large experience we have never met with an exception to this rule.

Diagnosis.—Root and trunk sciatica must be distinguished from “referred sciatica,” and from pains of sciatic distribution due to involvement of the nerve or its roots by growth.

In sacro-iliac disease pain may be referred along the sciatic nerve, but the nerve trunk is not tender to pressure or to stretching, while the joint is tender, and radiography may reveal a lesion in it. In osteo-arthritis of the hip the signs of sciatica are absent, while there is limitation of active and passive movement of the joint, and pain on movement. In this condition it is more common for pain to be referred down the front of the thigh than along the sciatic distribution. Fibrositis of gluteus medius and minimus may also produce pain of sciatic distribution of some severity.

In new growths involving the nerve, the clinical picture is not really that of a true sciatica. The pain differs in character, is frequently accompanied by muscular weakness and wasting and by sensory loss, and the patient's general condition is cachectic and suggestive of malignant disease. There may also be a history of primary carcinoma. The presence of bilateral sciatic pain is, as has been said, not pathognomonic of involvement of the nerve roots by growth. Indeed this involvement is as commonly unilateral as not.

The pains of tabes and other nervous diseases when confined to the sciatic distribution are distinguished by the presence of other physical signs of those diseases.

Course and Prognosis.—Sciatica commencing acutely tends in the course of time to lessen in severity and become chronic, but some acute cases cure rapidly. When the malady has a slow commencement it usually lasts longer than does an acute case, and it is much more liable to exacerbations than when commencing acutely. The traumatic cases show no essential difference from the spontaneous, except that in the former adhesions of the nerve to the muscles from bruising is likely to prolong the duration of symptoms. The prognosis is always absolutely good as regards recovery, and in severe cases there is no likelihood of relapse. In slight cases, however, subsequent attacks are not uncommon.

Treatment.—The first essential in the treatment of all recent cases is to secure rest and to avoid all those things which excite or increase the pain. The duration of the attack is often considerably prolonged by neglect of this most essential element in treatment. Sometimes the fixation of the limb in a semiflexed position by means of Liston's or Macintyre's splint gives great relief. The use of the bed-pan is advisable to avoid flexion of the hip and stretching or pressure upon the nerve in the act of defecation. On the other hand, towards the end of a chronic case, active exercise with massage and passive movements are necessary to restore the shortening of

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the nerve. The application of heat in the form of hot-water bottles, poultices and radiant heat is invaluable for the relief of pain, and for curative purposes. Counter-irritation is very useful, and is best used in the form of the strong tincture of iodine which should be painted in a broad strip over the course of the sciatic nerve, from sciatic notch to heel, daily until the skin becomes inflamed, just short of blistering. Massive injections of from 4 to 8 ounces of sterilised normal saline solution, made slowly into the region of the affected nerve at a temperature of 104° F., are said to be valuable both in acute and chronic cases. Acupuncture of the nerve with a series of specially designed needles is a useful and ancient remedy which acts by puncturing the sheath of the nerve, and allowing the escape of inflammatory exudation. Attempts to stretch the nerve by flexion of the thigh and extension of the knee may do great harm, and rarely do good. In chronic cases of sciatica or brachial neuritis and in fibrositis of the glutei, iodine given in the following manner is sometimes useful:

℞ Iodi . . .	gr. 1
Pot. iodidi . . .	gr. $\frac{3}{4}$
Sp. rect. . . .	mun. 12

Sig. min. 12 to be dropped into half a teacupful of water, and to be taken after being allowed to stand for 15 minutes. This may be administered thrice daily, and continued for 3 weeks. Some patients tolerate the doubling of this dose after the first week. The salicylates are of great service, especially in the form of aspirin, which may be given liberally. Urodonal in doses of 60 grains thrice daily is also often valuable. In the more chronic cases sulphur and guaiacum are serviceable. For the relief of pain heat is generally beneficial, but in some of the most acute cases the application of heat increases the pain, and in these an ice-bag will sometimes give great relief. All the analgesics of the coal-tar series, acetanilide, phenacetin, etc., are valuable adjuvants to relieve pain, and these may be conveniently prescribed with aspirin, or if sleep be difficult with barbitone or carbromal. When pain is very severe and rebellious to the above-mentioned remedies, opium or morphine is indicated. It is essential from the exhausting quality of the pain that the patient should be well fed, and alcohol is often of service.

RUPTURE OF THE INTERVERTEBRAL DISK

Synonym.—Herniation of the nucleus pulposus.

Definition.—A condition of pressure upon the spinal cord or on the roots of the cauda equina resulting from the extrusion into the vertebral canal of the nucleus pulposus of the intervertebral disk. It is traumatic in origin.

Ætiology.—The majority of cases give a history of injury at, or shortly before, the onset of symptoms. The injury is commonly of the variety known as a strain of the back, due to sudden bending, the lifting of heavy weights, or sudden movements of the back, as when striving to avoid a fall. Males are commonly affected.

Pathology.—Formerly the condition under discussion was one recognised on laminectomy, and spoken of as endochondroma of the disk. Actually in the circumstances enumerated above, the disk ruptures and its nucleus

(nucleus pulposus) herniates into the canal. The common seat of such rupture is the lumbar spine, commonly between the fourth and fifth lumbar, or between the fifth lumbar and the sacrum. Multiple ruptures have been recorded.

Symptoms.—The outstanding feature is pain, starting in the small of the back and radiating down one thigh, the outer side of the leg and the ankle. The pain is severe and lancinating, aggravated by stooping and relieved by lying down. There is some flattening and limitation of movement of the lumbar spine. Flexion of the extended leg at the hip may be painful. In some 60 per cent. of cases the ankle jerk is lost, other abnormal signs being absent. Occasionally more severe signs of root compression are present, and in rupture of dorsal disks some signs of compression paraplegia, mild in degree, may be found. The most typical picture is one of pain in the small of the back radiating down one leg. In other words, the symptoms usually mimic those of sciatica.

Diagnosis.—In addition to the history and signs mentioned, radiography of the spine may reveal narrowing of the intervertebral space at the level of the damaged disk, but the intrathecal injection of lipiodol may be necessary to confirm diagnosis.

Treatment.—Attention having been drawn to the condition, there is a not unnatural tendency to diagnose it somewhat too frequently, and to submit patients unnecessarily to lipiodol injection. Careful consideration of all the facts, and conservative modes of treatment, *e.g.* rest, massage, etc., should first be tried. The operative procedure for the removal of the extruded portion of the disk involves laminectomy and is a severe ordeal. For this reason it should not be undertaken unless the diagnostic indications are clear and unequivocal.

CERVICAL RIBS

Ætiology.—The development of the ribs at the thoracic inlet depends on the mode of formation of the brachial plexus, for the nerves are large structure in the embryo at a time when the ribs are soft and pliable. When the plexus is "normal," a well-formed first rib springs from the first dorsal vertebra. If, however, the plexus is "post-fixed," that is, when the contribution to the plexus from the fourth and fifth cervical segments is small and the fibres from the first and second dorsal segments form a powerful cord, this cord in rising over the first dorsal rib may compress and deform it to such an extent that it presents the characters of a rudimentary rib. On the other hand, and this is more frequent, when the plexus is pre-fixed, that is, when the contribution from the upper cervical segments is relatively large and that from the dorsal segments is small, a supernumerary rib is allowed to develop from the seventh cervical vertebra. When this pre-fixation is pronounced, the seventh cervical rib is often very large and is easily felt in the neck. In these cases symptoms are usually absent. In a certain number of cases in which the abnormality is intermediate in degree, symptoms are caused by compression of the lower cord of the plexus as it passes over the supernumerary rib, or over the deformed first rib. This compression may be exercised by the bony portion of the extra rib, but more often the nerves are damaged by a fibrous prolongation of the rudimentary rib which connects it with the first rib.

But these abnormalities in the ribs only cause symptoms in some 10 per cent. of the cases in which they are present. Further, the symptoms are often unilateral with bilateral supernumerary ribs, and the symptoms are often most prominent on the side of the smaller extra rib. Again, the onset of symptoms is usually delayed until adult life is reached. It is clear, therefore, that some contributory cause must come into play. This is found in the dropping of the shoulder girdle, which is normal in adolescents, and is often excessive in persons whose muscular tone is low. In a child the clavicle rises boldly as it passes outwards. In a normal adult male the clavicle is almost horizontal, in women it droops slightly, and in those who develop symptoms of pressure on the nerves, the outer is usually distinctly lower than the inner end. In the latter, the lowest cord of the plexus is submitted to constant rubbing against the extra rib which rises and falls during respiration, and it is compressed by any movement of the arm which depresses the shoulder girdle. Relief is obtained by raising the shoulders, and patients soon learn to support the limb and to assume attitudes in which pressure on the nerves is relieved.

Women suffer most often, the right arm being affected more often than the left. The onset is usually gradual, but occasionally it comes on suddenly after childbirth, or on lifting a heavy weight.

Symptoms.—These may be sensory, motor, or vasomotor, either singly or in combination. Subjective sensory disturbances are most frequent. They take the form of numbness and tingling or neuralgic pains. Paræsthesiæ are most often unilateral, and are frequently confined to the ulnar or to the radial side of the hand and fingers. It is rare for all the fingers to be affected. Pain, when present, is usually felt below the elbow. It is often neuralgic, darting down the arm, and again confining itself to one border of the limb. It hardly ever radiates from the neck.

Objective sensory disturbances are usually slight or absent. They may be found over the ulnar or radial border of the distal portion of the limb in an indefinite area, which does not conform to the distribution either of the ulnar or radial nerve.

Muscular atrophy is not so frequent as subjective sensory disturbance. In the "median type," wasting is confined at first to the abductor and opponens pollicis muscles, and the outer part of the thenar eminence shows a remarkable reduction in size, which contrasts strongly with the inner part, which retains its normal bulk. In the "ulnar type," wasting appears first in the small muscles of the hand supplied by the ulnar nerve. In some cases all the muscles of the hand and, to a less degree, the flexors in the forearm show considerable wasting. The atrophy is frequently bilateral and symmetrical.

Vasomotor disturbances are very common. The hands feel hot or cold, they may be œdematous or discoloured, and the changes may suggest Raynaud's disease. Pressure on the subclavian artery sometimes causes inequality of the pulse. This disappears when the arm is raised.

Diagnosis.—The presence of pain, paræsthesiæ or vasomotor disturbances in the upper limbs, or wasting in the muscles of the hands, should always arouse the suspicion of supernumerary or rudimentary ribs. When pain is the only symptom, its distribution along one border of the arm or hand, and the patient's account of the manner in which it may be increased or diminished by raising the shoulder girdle or performing movements which

depress it, usually direct attention to the cause. Symmetrical atrophy in the hands may suggest progressive muscular atrophy of spinal origin, but this diagnosis is usually rendered untenable by the association of sensory troubles or vasomotor phenomena, or by the findings on X-ray examination of the neck. For the differential diagnosis from syringomyelia, see page 1732.

Treatment.—Pain may be relieved by rest with the arms suitably supported. Atrophy calls for immediate operation to remove the offending rib. Pain is always relieved by operation, either immediately or after an interval of some months. The progress of atrophy is always retarded, and complete recovery may occur if an operation is undertaken early.

OBSTETRICAL PARALYSIS

It is important and useful to group together under this heading all those conditions of paralysis occurring, either in mother or child, which are the result of the processes of labour in the passage of the foetal head through the pelvis. Autopsies upon the still-born, and upon children who have survived birth for a few days only, have shown that hæmorrhage into the meninges is of common occurrence, and it has been argued that such meningeal hæmorrhages are the cause of many of the conditions of cerebral paralysis which are present immediately after birth, or which appear during the first year of life, and especially the cause of cerebral diplegia. The pathological conditions found in the brain in cases of cerebral diplegia, however, are such as make it absolutely impossible that they could be caused by meningeal hæmorrhage, for no sign of old hæmorrhage is ever found, nor could hæmorrhage cause a general cell atrophy of the brain without signs of any local lesion. It seems clear, then, that though meningeal hæmorrhage may be of common occurrence during birth, and may be the cause of still-birth, yet there is no clinical or pathological evidence to show that it gives rise to any lasting cerebral defect.

The following condition may occur : (1) In the child : facial paralysis ; hemiplegia from laceration of the brain substance ; fracture-dislocation of the spine with transverse lesion of the spinal cord ; injury to the brachial plexus from the separation of head and shoulder in traction ; and injury to peripheral nerve trunks at the elbow, axilla or groin, in using traction with the finger.

(2) In the mother : paralysis of the supply of the lumbo-sacral cord and obturator nerve from prolonged pressure of the head against the sacrum and pelvis.

Facial paralysis.—This is usually caused by the pressure of the forceps upon the facial nerve as it crosses the ramus of the jaw, but it has been known to occur where instruments have not been used. When unilateral, as is the common event, it gives rise to little or no difficulty with sucking, and is evidenced by the unsightly deformity of the face, which is drawn over to the sound side. When bilateral, it is one of the causes of complete inability to suck, and on account of the flaccid symmetry of the face may easily be overlooked. It necessitates spoon feeding for a considerable time. Obstetrical facial paralysis invariably recovers within a few weeks and does not give

rise to after-contraction. Gentle stretching and massage of the face with the finger is the only treatment required.

Hemiplegia from laceration of the brain may occur during delivery in contracted pelvis from the pressure upon the sacral promontory, and has been caused by the use of forceps. It is exceedingly rare, and is generally rapidly fatal from the associated hæmorrhage. It may occasionally be survived, with an irreparable hemiplegic condition.

Fracture-dislocation of the spine is produced by traction upon the after-coming head by pulling upon the trunk. We have seen it associated with injury to the brachial plexus. It occurs most often in the lower cervical region, and the transverse lesion of the spinal cord is usually complete.

Injury to the brachial plexus may occur in traction either upon the head, or upon the trunk, if the head is aftercoming, and is caused by an undue separation of head and shoulder on one side rupturing or straining the brachial plexus. The paralysis is usually of the upper arm or Erb type, the fifth and sixth roots being most affected, and the deltoid, biceps and supinator longus muscles being paralysed, but the whole plexus may be involved and even torn completely across. Traction upon a prolapsed arm has caused lower arm or Klumpke type of paralysis, in which the first dorsal and eighth cervical roots are most affected, and the intrinsic hand muscles and the flexors of the forearm are paralysed. The obstetrical lesions of the brachial plexus are for the most part serious lesions, many of the cases making no motor recovery at all, though sensibility is usually regained. The prognosis depends upon the severity of the damage to the plexus, as to whether the roots are actually torn or only bruised. The slight cases recover well enough.

Injury to the peripheral nerves from pressure or traction upon the flexures is seldom severe enough to prevent a rapid and complete recovery.

Paralysis of the lumbo-sacral cord and of the obturator nerves in the mother, immediately after parturition, is an exceedingly interesting clinical condition. In the first place, the lumbo-sacral cord is in a much more exposed position as regards the foetal head engaging the pelvis than are the other nerves of the sacral plexus, and may be subjected to such severe pressure as causes paralysis, and in the second place, the obturator nerve actually crosses the brim of the pelvis and must of necessity be pressed upon by any large foetal head which passes the pelvic brim. The lumbo-sacral cord paralysis is evidenced by dropped foot and paralysis of the anterior tibial and peroneal muscles and if it is severe, by loss of sensibility over the distribution of the fourth and fifth lumbar roots. Sometimes the third lumbar root area is affected. The obturator nerve involvement is shown by weakness or paralysis of the muscles supplied by the obturator nerve, namely, all the adductor muscles of the thigh. The paralysis may be noticed directly after parturition, or when the patient begins to get about upon her legs. The lumbo-sacral paralysis is usually unilateral, and is nearly always upon the right side. The obturator paralysis is not uncommonly bilateral, and both forms of the paralysis may coexist. There may be numbness, but no pain. This condition nearly always occurs with a first delivery, and often the child's head has been unduly large. It may recur with subsequent deliveries, but this is not a common event.

The prognosis is absolutely favourable, every case making a complete

recovery in from a few weeks to a few months. The treatment is rest in the first place, with gentle massage and passive movements, and when power begins to return the patient may commence to get about.

POLYNEURITIS

Synonym.—Multiple Peripheral Neuritis.

The clinico-pathological condition we know as polyneuritis, and seen in its most typical forms in diphtheritic paralysis or in alcoholic neuritis, represents a very striking and uniform reaction of the nervous system. Invariably associated with it is a reaction of the myocardium, so that there is in cases of polyneuritis a recognised liability to sudden fatal heart failure. It is in the case of diphtheritic paralysis and of beri-beri, another form of polyneuritis, that this mode of fatal termination is most often seen. Indeed, beri-beri may appear as a rapidly fatal cardiac illness before any signs of involvement of the nervous system have had time to develop.

Ætiology.—At first sight the factors that give rise to polyneuritis fall into three groups: (i) certain chemical poisons, (ii) the toxins of certain bacteria, and (iii) certain disorders of metabolism. Widely differing as these three causative factors may seem to be, there is reason to believe that a common underlying factor which is immediately responsible for polyneuritis may underlie them all. It is probable that in the case of groups (i) and (ii) the pathogenic substance gives rise to a disorder of metabolism in the course of which a toxic metabolite is produced in the body, this acting as the direct poison for nervous system and heart muscle. In the metabolic group (iii) the same process is in action.

Thus, in beri-beri, for example, the illness ensues upon the ingestion of a diet deficient in vitamin Bi. In the absence of this substance carbohydrate metabolism is disordered and a toxic metabolite is produced. Thus, beri-beri is not, as the biochemists formerly insisted, a starvation-degeneration of the nervous system, but an intoxication strictly comparable with that obtaining in other varieties, ætiologically considered, of polyneuritis.

The final and complete proof of this unity of causation of polyneuritis, in whatever circumstances it is seen, is not yet available, but there is an increasing body of evidence in favour of it.

Returning for the moment to the ordinary ætiological classifications of polyneuritis, we see that in the case of alcoholic or arsenical polyneuritis the poison is taken by the mouth, and presumably the final common toxic substance reaches the nervous system by the blood stream. In the case of diphtheritic paralysis, on the other hand, the exotoxins are produced locally at the site of the diphtheritic ulceration, whether on the fauces or, as in extra-faucial diphtheria, at some other local site on the body surface. This unique channel of entry gives rise in diphtheritic paralysis to a group of symptoms not found in other ætiological varieties of polyneuritis. This group includes palatal and accommodation paralyses, which precede the appearance of polyneuritis.

It is noteworthy that in the case of extra-faucial diphtheria this initial paralysis is not palatal, but is anatomically related to the site of the diphtheritic lesion (skin ulceration or wound). Yet the paralysis of accommodation

may occur whatever be the site of the diphtheritic lesion. It is believed, therefore, that the exotoxins gain access to the nervous system by conduction from the seat of the lesion via the axones of the nerves which innervate this region. They pass upwards in the axis cylinders to the central nervous system and produce their toxic action directly there, this action being reflected peripherally again as a motor and sensory paralysis of the muscles and skin (or mucosa) in the region of the lesion. Thus, a diphtheritic ulcer on a finger may be followed by a local paralysis of that part before polyneuritis develops. The subsequently developing polyneuritis is then probably produced in the manner described above, while the accommodation paralysis may indicate a specific action of the toxin upon the nervous mechanism concerned. We thus have a local, a specific and a general group of symptoms. The analogy of the local, specific and general phases of tetanus will occur to the reader.

Many of the intoxications of the nervous system commonly included under the heading of polyneuritis are associated with lesions and clinical manifestations which are not those of polyneuritis. Such substances, to name but a few, are lead, mercury, copper, carbon disulphide and carbon monoxide, and it would be erroneous to regard these as causes of polyneuritis. Many infectious fevers are stated to be not rarely followed by polyneuritis, namely, enteric, malaria and dysentery, but the present writer who spent four years with the Egyptian Expeditionary Force in 1915-19 where the two last named of these infections were common saw no case of true polyneuritis associated with them.

Finally, the categories of toxic polyneuritis, acute febrile polyneuritis and Landry's paralysis have no known causal factors. They make their appearance in apparently healthy persons, adequately nourished and free from all discoverable signs of infection, and it is extremely difficult in the present state of knowledge to account for them on any hypothesis of avitaminosis, or to suggest any possible mode of intoxication. In short, the pathogenesis of polyneuritis presents many unsolved problems.

Clinically, polyneuritis is a lower motor neurone type of paralysis accompanied by a varying intensity of sensory loss of much the same type of distribution as the paralysis, and, as already mentioned, by signs of myocardial poisoning.

1. ARSENICAL NEURITIS

Peripheral neuritis may be caused by a single large dose of arsenic, or it may result from prolonged use of the drug in the treatment of such diseases as Hodgkin's disease, chorea and severe anæmia. It is a rare malady, and the likelihood of its appearing under the last-named conditions is negligible. The toxic action of arsenic with alcohol seemed to be greater than that of either alone.

The description given below of alcoholic neuritis applies to this form as well. Hyperæsthesia of the skin and tenderness of the muscles are more constant and more severe in the arsenical form, and paralysis and atrophy of the muscles are often more widespread and more rapid in their progress. Hyperkeratosis of the soles and pigmentation of the skin are characteristic of arsenical poisoning. In a suspected case, the diagnosis can be confirmed

by the discovery of abnormal quantities of arsenic in the urine or in the hair and skin.

The mental changes described in connection with alcoholic neuritis under the heading of Korsakow's psychosis may be present, especially when repeated poisonous doses of arsenic have been taken.

2. ALCOHOLIC NEURITIS

In former years alcoholism was perhaps the commonest cause of severe peripheral neuritis. At present it is a rare disease. It occurs most often in women, especially in those who take small amounts of alcohol frequently. It has often been the first indication of secret drinking.

Pathology.—The changes in the nerves are those of parenchymatous neuritis. They are most intense in the small branches supplying the skin and muscles, and they diminish in severity as the larger branches are approached. They are best seen in the terminal branches of the musculo-spiral and anterior tibial nerves. The wasted muscles often show a reduction in the size of their fibres, and an increase of connective tissue—fibrous myositis. The spinal cord may be healthy, but in almost all cases examination by modern methods shows changes in the nerve cells and degeneration in the tract fibres derived from the posterior roots.

Symptoms.—The onset is insidious, and in most cases premonitory symptoms, such as numbness and tingling in the extremities or cramps in the muscles of the lower limbs, are present for several months before actual weakness occurs. Subjective sensory troubles are a marked feature, even in the early stages. Besides numbness and tingling, the patients complain of feelings of excessive heat or of coldness in the limbs, or of severe aching or cutting pains in the legs. Painful cramp in the calf muscles is a common symptom. It is often worst at night, and may interfere seriously with sleep. Objective examination usually reveals sensory loss, in which the various elements of sensation are affected in a manner which is almost pathognomonic.

Stated briefly, there is anæsthesia of the skin with hyperæsthesia of the deeper structures. Light touches are not appreciated at all or many are missed, the temperature sense is defective, and the prick of a pin causes no pain, whereas even moderate compression of the muscles may cause the patient to cry out. The sensory loss is greatest in the feet and hands and diminishes towards the knees and elbows. Muscular tenderness is usually greatest in the calves. The soles of the feet are also unduly tender. Hyperalgesia is often well marked before anæsthesia of the skin appears. To the disability caused by pains and spasms, weakness of the muscles is added in all but the slightest cases. The arms may suffer first, but in most cases the extensors of the toes, the dorsiflexors of the ankle, and the extensors of the fingers and wrists are attacked in progression, and double foot-drop and wrist-drop result. To overcome the foot-drop, the knees are raised high in walking. This gives to the gait the "steppage" character which is common to all forms of peripheral neuritis. In most cases the distal flexor muscles are also affected, but to a slighter degree. In severe cases, weakness extends to the proximal muscles and even to the muscles of the trunk. The affected muscles become soft and diminish rapidly in bulk. Unless precautions are taken, contrac-

tures occur in the flexor muscles and produce deformities of the limbs, which add greatly to the difficulties of treatment.

At the onset the knee-jerks are exaggerated, but in most cases by the time the patient comes under observation all the tendon reflexes are absent. The cutaneous reflexes may be unaltered, diminished or absent. Sphincter control is retained. Slight bilateral weakness of the face is often present but severe paralysis is rare. Ptosis, nystagmus, and weakness of the external ocular muscles have been observed.

Trophic and vasomotor disturbances in the extremities are common. The hands and feet often perspire freely, and they may be white and cold or red and hot. In some cases oedema of the hands or lower extremities is present. In chronic cases the skin of the hands and fingers is thin, smooth and shiny, and the nails are ridged and brittle.

In almost every case of alcoholic neuritis there is some *psychical defect*. One form—Korsakoff's psychosis—is characteristic of and almost peculiar to this disease. The most prominent feature is failure of memory and loss of appreciation of time and place. A patient who has been bedridden in a hospital for nervous diseases for several weeks, when visited by the resident physician who has attended her daily, will "recognise" him at once as Dr. X, whom she has not seen since he brought her first child into the world some years ago. She is now, she says, in a lying-in hospital which she entered yesterday, and has just been confined with her second baby, who is in bed beside her. She also "recognises" strangers at her bedside, and connects them with events of long ago. Everything is related in the most circumstantial manner, and if the facts were not known her tale might well be accepted as truth. In most cases the mental defects are not so gross. There is merely a failure of memory, to which is added moroseness and irritability, caused by withdrawal of alcohol.

3. DIPHThERITIC PARALYSIS

The exotoxin of diphtheria is highly selective for nervous tissues, and some form of paralysis occurs in a very high proportion of the cases. The intensity of the paralysis bears no constant relation to the severity of the local infection, for cases, in which the original disease has passed unnoticed, may be followed by serious damage to the nervous system. Walshe has classified the nervous manifestations of diphtheria into three distinct groups, namely, the local, the specific and the generalised paralyses.

Local paralysis occurs in parts related anatomically by nervous connections to the site of the diphtheritic lesion. In faucial diphtheria, the local palsy appears in the palate. In extra-faucial diphtheria, *e.g.* infected sores on the limbs, the local palsy appears in the muscles supplied by the segments of the cord to which afferent nerves from the infected focus pass. The reason for this is, that toxins elaborated by the diphtheria bacillus ascend from the primary focus to the cord or the medulla. Having reached the central structures, they diffuse to neighbouring motor cells and, by injuring them, cause paralysis of the muscles they supply. Paralysis of the palate therefore does not occur except in faucial diphtheria.

The *specific* manifestation of diphtheria is paralysis of accommodation. Like trismus in tetanus, it is not due to a local lesion, but occurs in many

cases, whatever the site of origin of the toxins. It is present in cases of both faucial and extra-faucial diphtheria, and is the local effect of exotoxin accepted from the ingeneral blood stream.

The third or *generalised* form of diphtheritic paralysis is multiple neuritis. It follows extra-faucial as well as faucial diphtheria, and is also a result of the action of exotoxin circulating in the blood.

Symptoms.—As faucial diphtheria is the commonest form, the most frequent nervous symptom is *paralysis of the soft palate*. It is shown by the nasal quality of the voice and by the regurgitation of fluids through the nose. As a rule, the weakness is bilateral and equal, but in some cases it is greater on the side on which the local lesion is more severe. It makes its appearance in most instances about the end of the second week, but may come on as early as the fourth day, and as late as the sixth week. The soft palate is relaxed, and its movement on phonation is diminished. The palate may be insensitive, and its reflex is often diminished or lost. Recovery usually occurs in a few weeks. In rare instances the muscles of the pharynx and the vocal cords are paralysed. Together with palatal palsy, it is common to find marked weakness and tenderness of the sternomastoid muscles and masseters. These are also local effects.

Paralysis of accommodation appears about the same time as the palatal palsy, perhaps a few days sooner. The reaction of the pupils to accommodation as well as to light, can almost always be obtained. The trouble is subjective, and is shown by defects of near vision—for example, by inability to read small print. Hypermetropes suffer great inconvenience. In myopes it may pass unnoticed. Paralysis of any of the extrinsic ocular muscles with strabismus and diplopia may occur, and this may be either nuclear or peripheral in type.

Multiple neuritis usually comes on three to six weeks after recovery from the throat infection. Its presence is often detected when patients begin to exert themselves during convalescence. Weakness and aching pains in the legs, unsteadiness in walking, clumsiness in performing fine movements with the hands, feelings of pins and needles in the extremities—all these are common early symptoms. Weakness affects in varying degree the muscles of the neck, trunk and limbs. It is generally slight in degree, greater in the lower than in the upper extremities, and greater in the extensor muscles than in the flexors. Marked local atrophy is uncommon. In severe cases, life may be endangered by paralysis of the intercostals and of the diaphragm, but fortunately one set of muscles has usually begun to recover before the other is seriously affected. The small muscles of the hands and feet and the muscles of the calves and forearms are almost always tender on pressure. They are soft and flabby, and often show a partial reaction of degeneration.

Sensory ataxy is almost always present, and is often severe when the paralysis is trivial. It causes the patient great inconvenience, as it interferes seriously with walking and with the finer movements of the hands. Objective examination reveals sensory impairment of the "glove-and-stock-ing" type. On the hands and feet, the loss to light tactile stimuli is often complete, pain and temperature being less affected. As the limb is accented, sensation gradually becomes normal. Even when the sensibility of the skin is but little diminished, the sensations of position and of passive movement

in the extremities are often seriously impaired, and the sense of vibration is often lost.

In the early stages and for a few days the tendon jerks are exaggerated, but are lost later in every case. Their return is often long delayed, and it is common to see patients months after recovery of normal power, in whom the knee-jerks are still absent. It is common also to find them absent many months after an attack of diphtheria in patients who give no history of nervous symptoms during the attack. The skin reflexes are usually retained, and stimulation of the sole gives a normal response.

Cardiac failure is a grave but uncommon complication. It is of myocardial origin. Vasomotor paralysees and disturbances in the nutrition of the skin, which occur so often in other forms of peripheral neuritis, are never seen in diphtheria. In those that survive the attack, complete recovery from the nervous troubles always occurs.

4. DIABETIC NEURITIS

In many patients with glycosuria, symptoms are present which point to changes in the peripheral nerves, or in the fibres of the posterior roots. In many respects they resemble *tabes* rather than peripheral neuritis; but as the exact pathology is still unknown, it is convenient to describe them here.

Pathology.—Degenerative changes have been found in the peripheral nerves in some cases, in others these were healthy, whilst the intramedullary portion of the posterior roots showed degeneration, similar to that found in *tabes*.

Symptoms.—In some cases the only symptom is neuralgic pain in the distribution of one or more peripheral nerves. This is commonest in the lower limbs, where it simulates sciatica, and sugar is found in the urine in the absence of any other sign of diabetes.

In severe cases of diabetes the knee-jerks and ankle-jerks are diminished or lost in more than half the cases. This may accompany subjective sensory troubles in the lower limbs, or it may appear as an isolated symptom. The muscles are very often tender and the vibration sense of the feet is frequently absent. To objective examination, the sensibility of the skin is usually intact. Perforating ulcers of the feet have been observed.

Diagnosis.—The diagnosis of multiple neuritis from other diseases rarely presents any difficulty. It is made from the combination of symmetrical flaccid paralysis with sensory loss of the "glove-and-stocking" type, and tenderness of the muscles and nerves, confined to or most intense in the distal parts of the limbs. When sensory disturbances and diminished tendon reflexes are prominent symptoms and muscular weakness is slight, *tabes* may be suggested, and the resemblance is still greater when ataxia is present. Difficulty usually arises when the distinction has to be made between *tabes* and alcoholic neuritis, in a patient who has courted both diseases. In most instances the diagnosis can be made from the nature and distribution of the sensory changes. The lightning pains of *tabes* cannot be mistaken by any one who is familiar with their peculiar characters. Anæsthesia of the extremities is common to both diseases, but diminished sensibility around the

nose and across the chest is peculiar to tabes and is present in almost every case. In neuritis the calf muscles and nerve trunks are tender, whereas in tabes the sensibility of these structures is usually greatly diminished. Hyperæsthesia to touch and temperature, and great exaggeration of the abdominal reflexes, also suggest tabes.

5. ACUTE FEBRILE POLYNEURITIS

Synonym.—Acute Infective Polyneuritis.

At various times small epidemics of a form of polyneuritis characterised by a febrile onset and by the involvement of the facial nerves have been described (Osler, Gordon Holmes, Rose Bradford and others). Nothing is known of its ætiology and it is probably not essentially different from Landry's paralysis in nature, though less fulminating in onset and not so liable to grave involvement of the trunk muscles.

Symptoms.—The onset is with slight fever, headache and malaise, pains in back and limbs, and such general symptoms as a coryza or gastro-intestinal irritation. The fever persists for 2 or 3 days only. A few days then elapse before the signs and symptoms of polyneuritis develop. It is said that the proximal limb muscles are more severely involved than the distal muscles, a point of distinction from other forms of polyneuritis, but this relative incidence of weakness is not invariable and has probably been over-stressed. The trunk muscles do not escape, and the face is usually bilaterally paralysed. As in other forms the paralysis is of the lower motor neurone type, flaccid, atrophic and with loss of tendon jerks. As in Landry's paralysis sensory loss is very slight, and there is relatively slight muscular tenderness. The cerebro-spinal fluid may show a high rise in the protein content, but is otherwise normal.

The clinical course is variable, and sometimes fluctuating in the individual case. Death may ensue from paralysis of the respiratory muscles, and recovery in the majority of cases is fairly rapid. There is the usual tachycardia of polyneuritis.

Treatment.—The first essential in treatment is to remove the patient from the influence of the existing cause. In alcoholic cases, rigid precautions are necessary to prevent secret access to alcohol. To attain this, treatment in an institution is almost a necessity. In most instances when the cause, whatever it may be, is removed, gradual improvement sets in and complete recovery ensues, in a time that varies with the severity of the symptoms. The administration of vitamin B₁ preparations has now a great vogue in the treatment of both multiple (parenchymatous) and interstitial neuritis. In the latter its use has no theoretical justification or practical value, and even in the former, though its use is rational, it yet remains to be proved that the course of the malady is materially influenced. This may be due to inadequacy of dosage in the past, and the parenteral injection for from 2 to 4 weeks of 500 to 1000 units daily is the minimum dosage likely to be efficacious. During this time the physician's most important duty is to prevent the occurrence of deformities and contractures. From the beginning, each joint in the affected limbs should be moved passively to its full range several times each day, and care should be taken to ensure that the attitude of the limbs during rest is a suitable one, especially that the feet are main-

tained at right angles to the legs by the use of appropriate apparatus. Drawing up of the heel must be prevented at all costs.

Gentle massage is soothing in the acute stage. Later, more vigorous rubbing may be given, and the patient should be encouraged to move the limbs voluntarily. Electricity is of no value. Analgesic drugs and soothing applications may be needed at the onset. Thereafter, local treatment to the limbs is combined with measures to improve the patient's general condition. The possible rôle of B avitaminosis in the production of many, if not all, varieties of polyneuritis has naturally led to the administration of the B₁ complex as an important element in treatment. Not less than 1000 to 2000 units daily and given over a period of two or three weeks is adequate, but even with this dosage it is not yet clear that the course of any ætiological variety of polyneuritis is materially influenced, and extravagant claims should be treated with great reserve.

6. LANDRY'S PARALYSIS

In the year 1859 Landry applied the name "acute ascending paralysis" to a case in which acute flaccid paralysis with loss of reflexes and without sensory disturbances commenced in the periphery of the lower limbs, and rapidly spread upwards. The arms were next involved, first in the periphery, and later the trunk, respiratory muscles, neck, and lastly the cranial muscles were involved, and death occurred from respiratory failure. He made a careful microscopic examination of the spinal cord with the methods then at his disposal, and failed to detect in it any morbid changes. He subsequently described this symptom complex, which has since borne his name, from an analysis of 10 cases.

Since this time a large number of cases have been recorded which, from the acute nature of the onset, and from the spreading nature of the paralysis, have been described as cases of Landry's paralysis. This name should be restricted to those cases of acute spreading paralysis, in which disorders of sensibility and sphincter trouble are absent or little marked, and in which recovery is complete if the patient survives, and in which no gross lesion is found within the nervous system after death.

Acute poliomyelitis may also, in rare cases, give rise to a spreading paralysis, and cause much difficulty in diagnosis; but it is invariable that some permanent paralysis remains upon recovery, and, further, the lesions of poliomyelitis are both gross and characteristic.

The majority of authors who have written upon this subject have made the attempt to separate Landry's paralysis from the group of "acute toxic polyneuritis," both on pathological and on clinical grounds. Such a separation would appear to be entirely unsupported by the evidence. In both these conditions all the nervous pathological changes which have been discovered are confined to these lower neurones, motor and sensory, and are often in polyneuritis confined to the lower motor neurones. The clinical separation of Landry's paralysis and polyneuritis is equally artificial and impossible, though much stress has been laid upon the presence of disturbances of sensibility, and the strictly peripheral distribution of the paralysis in polyneuritis. As regards sensory disturbance, this clinical feature is dependent upon the peculiar selective capacity of the poison. The following

description of this malady is based upon the personal observation of 10 cases with 4 autopsies which have come under our observation at the National Hospital and at St. George's Hospital.

Ætiology.—What is known of the causation of the disease in general resembles very closely that of acute polyneuritis. It affects males much more frequently than females, and occurs chiefly in adult life between the ages of 16 and 54 years. The cases which have been reported in children were probably examples of the spreading type of poliomyelitis.

Pathology.—Slight hyperæmia of the spinal cord, and especially of the grey matter, with a few punctiform hæmorrhages, is the only change noticeable upon naked-eye examination. Very definite histological changes are found upon microscopic examination in the anterior horn cells and in the cells of Clarke's column, where any degree of change may be found, from an early pericentral chromatolysis to a complete loss of the chromatin granules and concentration of nuclei.

The cerebro-spinal fluid is in excess, and clear. In two of the cases under our care, it presented no abnormality either as regards cell or albumin content. In other cases there is an excess of albumin, and in this respect it resembles the cerebro-spinal fluid of polyneuritis, which is usually albuminous, and sometimes so highly so as to clot spontaneously.

Symptoms.—The onset is in some cases abrupt, with the appearance of the characteristic spreading paralysis. Much more frequently the paralysis is preceded by certain premonitory symptoms, which may last from a few hours to days or weeks. These symptoms may consist in malaise, headache, lassitude, insomnia, anorexia, constipation, gastralgia, vomiting and diarrhœa, and there is not infrequently slight elevation of temperature. More characteristic still among the prodromal signs are subjective disturbances of sensibility. Pains in the back and limbs are common, and may be of a dull aching nature, or they may be sharp and shooting in character. Numbness, tingling, "pins and needles" and other paræsthesias may occur over any part of the body, and are most commonly complained of in the periphery of the limbs. The muscles may be locally tender during this prodromal stage.

It is not uncommon for the paralysis to commence in the periphery of the lower extremities, to ascend rapidly, and to involve the muscles in the order of their innervation from the spinal cord, the trunk becoming affected before the upper extremities, and the intercostal muscles before the diaphragm. The muscular weakness may commence in any group of muscles, as, for example, in the face, neck, upper extremities or trunk, and when so commencing the spread of the paralysis is downwards, constituting a descending type of paralysis.

In Landry's paralysis, as in acute polyneuritis, the innervation of the respiratory muscles seems to be peculiarly resistant to the toxin.

In those cases which recover the advance of the paralysis ceases, and those muscles which have been most recently affected begin to show some recovery quickly.

When the disease does not prove fatal either from respiratory failure, pulmonary complications or sudden syncope, the paralysis ceases to spread, and the patient enters upon the stage of recovery, which presents many features of interest. The flaccid muscles show a moderate degree of wasting within 2 or 3 weeks on the onset, this wasting being much less in those cases

which recover rapidly. It is a general atrophy, and is not limited to particular groups of muscles. In rare cases, though fair power is regained, yet the muscles remain conspicuously small for life; but generally the muscles recover their bulk and tone completely. The paralysed muscles retain their excitability to faradism throughout, though there may be some slight diminution of faradic excitability in proportion to the general wasting of the muscles. Contractures and deformities do not occur.

The paræsthesias, which have been described with the onset, often persist, and there may be cramp-like pains. Not uncommonly the muscles are tender upon deep pressure; but there is never that severe degree of tenderness met with in some forms of peripheral neuritis as, for example, in alcoholic neuritis. There is exceptionally blunting of sensibility, most marked in the periphery; but this is never deep, and is rapidly transient.

Though from the general weakness of the trunk muscles there may be some difficulty in emptying the bladder and rectum during the first few days, and even retention with overflow incontinence that may require catheterisation from the same cause, yet these last but a few days. The deep and superficial reflexes disappear early with the onset of the first signs of the paralysis in the affected regions. The psychic functions remain unimpaired throughout.

Diagnosis.—The rapidly spreading character of the paralysis in Landry's disease is so striking as to necessitate distinction only from those few maladies in which a similar rapidly spreading paralysis may occur, and these are acute spreading myelitis, intrathecal hæmorrhage, acute poliomyelitis (spreading type) and acute polyneuritis. Acute spreading myelitis is at once distinguished from Landry's paralysis by the severe sensory loss and sphincter paralysis, which in the former condition develop *pari-passu* with the motor paralysis and, further, if the myelitis does not involve the lumbo-sacral enlargement of the spinal cord, an extensor plantar reflex will be observed.

The rare, spreading form of poliomyelitis presents difficulty in diagnosis, especially in the acute stage. The general symptoms and the pyrexia are apt to be more severe in poliomyelitis. An onset in childhood is more suggestive of poliomyelitis than of Landry's paralysis. A fairly high polymorpho-nuclear leucocytosis in the blood, and a lymphocytosis in the cerebro-spinal fluid, are in favour of poliomyelitis. The persistence of local atrophic palsy on convalescence is absolute evidence of poliomyelitis. The distinction of Landry's paralysis from acute polyneuritis is held by the writers of this article to be entirely artificial, since they argue that *Landry's disease is merely a striking type of acute polyneuritis*.

Prognosis.—In about one-half of the cases the paralysis advances until the respiratory and bulbar muscles are involved, and death occurs from respiratory failure, usually on the third or fourth day, but sometimes not until ten days or more have elapsed. So long as the paralysis is extending, and especially when the respiratory and bulbar muscles are failing, the prognosis is very grave. The extension of the paralysis may, however, cease at any stage, and when this occurs the prognosis at once becomes favourable, even though there be considerable involvement of the respiratory and bulbar muscles.

Treatment.—The patient must be placed at complete rest, and the discomfort and panic which are likely to arise from the utter inability to move must be assiduously relieved by frequent changes of posture. The greatest

care must be taken that the patient is adequately fed with nutritious and light food. Stimulants are usually indicated. A mercurial aperient should be administered early and the bowels regularly relieved, for in some cases obstinate constipation occurs. The bladder should be catheterised, if there is a difficulty in micturition. Both pain and pyrexia may be relieved by the administration of salicylates or aspirin.

Atropine tends to check accumulation of secretion within the bronchi. Oxygen may be administered where cyanosis occurs. When once the patient has shown signs that the malady has passed its height, and that recovery is commencing, little treatment is required except careful nursing and feeding. Gentle massage may then be employed.

7. PROGRESSIVE HYPERTROPHIC POLYNEURITIS OF DEJERINE AND SOTTA

Definition.—An extremely rare progressive form of polyneuritis, sometimes developing in infancy, showing an heredo-familial incidence, and characterised by thickening of the nerve trunks due to hypertrophy of the sheaths of Schwann.

Pathology.—The thickening of the nerves may be palpable during life, but is not invariably so. Microscopically this thickening is found to be due to masses of non-nucleated tissue arising from the sheath of Schwann.

Symptoms.—The malady develops and progresses very slowly with weakness, muscular wasting, sensory loss, loss of tendon jerks. There may be noted, in addition, kyphoscoliosis, nystagmus and ataxy of movement. It was formerly thought that the Argyll Robertson pupil was an integral part of the symptom-complex, but this is not the case.

Prognosis.—Death ultimately ensues from intercurrent disease.

Treatment.—There is no known treatment.

NEUROFIBROMATOSIS

Synonym.—Recklinghausen's Disease.

Definition.—A rare and complex disease characterised by multiple benign tumours on the skin and on the nerves and the ganglia of the somatic and autonomic nervous systems. Cutaneous pigmentation, and multiple tumours on brain and spinal cord may also be present.

Ætiology.—The malady is probably developmental in origin, and heredo-familial incidence is not uncommon.

Pathology.—The skin presents spots of varying size and number that have the colour of freckles. When these are extensive they are known as café-au-lait patches. They tend—like the cutaneous tumours—to increase in size and number with age. The skin is adorned with numerous soft fibromatous tumours, some sessile and some pedunculated. In some cases the trunk may be thickly sewn with these. Large plexiform neuromas on cutaneous nerves may form large masses on the skin, flattened and of irregular outline, and when this aspect of the disease is prominent it is given the name of *molluscum fibrosum*.

On the nerve trunks are more or less numerous, fairly firm nodular tumours of varying size. The cranial nerves may also bear similar tumours, especially the fifth and eighth nerves. Marked kyphoscoliosis is common.

Symptoms.—The cutaneous pigmentation and tumours are readily visible and tend to increase throughout life. Sometimes the neurofibromata on the nerves give rise to symptoms of irritation (pain in the distribution of the nerve) or of paralysis. Intrathecal tumours may give rise to spinal cord compression, while the symptoms of an auditory nerve tumour (unilateral or bilateral) may first bring the patient to notice.

Treatment consists in the removal of fibromata on the nerves when these give rise to irritative or paralytic symptoms.

LEAD PALSY

The effects of lead are confined almost entirely to motor neurones. Subjective sensory disturbances are often slight or absent, and in most instances there is no objective sensory loss.

Pathology.—Aub in 1923 showed that the first event was the local concentration of lead in those muscles which were about to be paralysed and that the paralysis was a muscular event primarily, and that, secondarily, the lead ascends along the motor axons and may finally cause the death of the ventral horn cell. The degenerative changes in the nerves are confined almost entirely to the motor fibres, and are most intense in the intramuscular twigs supplying muscles of the extensor groups. Normal and degenerated fibres are found side by side, the former becoming more numerous as the nerve is traced upwards. Degenerative changes due to the action of lead are also found in the affected muscles.

Symptoms.—In most cases of the common *antebrachial* or *wrist-drop type*, paralysis is limited to the extensor muscles of the fingers and wrists—that is, to the muscles supplied by the musculo-spiral nerve. But the supinator longus and the extensor ossis metacarpi pollicis, also supplied by this nerve, usually escape. Inability to extend the first phalanges of the two middle fingers, owing to weakness of the common extensor, is usually the first difficulty. The special extensors of the index and little fingers, the long extensors of the thumb and the extensors of the wrist are next attacked, and the characteristic wrist-drop appears. As a rule the paralysis becomes severe about a week after it is first noticed. By this time it is usually bilateral and symmetrical, but for several days, or even for several weeks, it may be confined to one side. The affected muscles waste rapidly and the back of the forearm becomes flattened, thus rendering the intact supinator longus more prominent. In this form, loss of power always precedes atrophy, and some muscles may show weakness without any wasting. Recovery is almost always complete. Simple weakness without atrophy usually passes off in a few weeks. If the wasting is moderate and the muscles still react to faradism, recovery may be expected in a few months. When the atrophy is severe, a year or more may elapse before recovery is complete.

Occasionally the deltoid, biceps, brachialis anticus and supinator longus muscles are affected, either alone or in company with the forearm muscles—*upper arm* or *brachial type*. Less often paralysis occurs in the legs, the muscles supplied by the peroneal nerve, namely, the long extensors of the toes and the peronei, being chiefly involved—*peroneal type*. Like the supinator longus in the arm, the tibialis anticus, although supplied by the peroneal

nerve, usually escapes. This type is usually associated with paralysis of the forearm muscles, and runs the same course.

In the form of paralysis described above the features are those of a traumatic lesion to a nerve. Loss of power precedes, and may be more extensive than wasting, faradic irritability of the muscles is lost or diminished while the reaction to galvanism is retained, and recovery is usually complete. It is therefore called the degenerative form. In the second form, the paralysis has the characters of progressive muscular atrophy. Weakness and wasting come on together, faradic and galvanic irritability of the muscles are both diminished in proportion to the wasting, and the paralysis is often permanent. This is known as the primary atrophic form. It occurs especially in the small muscles of the hand—*Aran-Duchenne type*—but is sometimes irregular in its distribution and affects many muscles in all four limbs. It is often associated with the first form, but may occur alone. Wasting comes on slowly, and accompanies the loss of power, instead of succeeding it. It is much more intractable than the degenerative form, and often persists after muscles showing the first form of paralysis have recovered. (See also Lead Encephalopathy, p. 366.)

MUSCULAR DISEASES

MYOTONIA CONGENITA

Synonym.—Thomsen's Disease.

Definition.—A very rare malady, commencing in early childhood, which is hereditary and familial, and characterised by a striking slowness in the relaxation of the muscle after voluntary effort. The muscles pass into a spasm on voluntary contraction, which relaxes very slowly, resembling the contraction of the veratrinised frog's muscle, and its subsequent slow relaxation. Peculiar changes in the electrical excitability of the muscle and hypertrophy of the muscle fibres are constant.

Ætiology.—Beyond the facts that the malady is usually hereditary and familial, only a few sporadic cases occurring, and its incidence in early childhood, nothing is known of the causes. Cold, heat, fatigue and hunger conspicuously increase the symptoms.

Pathology.—The affected muscles are actually hypertrophied, and are always firmer to the feel than normal muscles, while sometimes they show a board-like hardness. The individual fibres show considerable hypertrophy.

Symptoms.—The presence of the disease first becomes evident from slowness, clumsiness and awkwardness of movement, with a great tendency to fall if the balance is upset. This is often most noticeable after rest, when, on attempting to move, the limbs seem glued down and move very slowly. Often the patient is able with exercise to work the stiffness off, and the myotonia lessens in the muscles which are being used; but if he is suddenly called upon to put another set of muscles into action, as, for example, by losing his balance, he is at once caught up by the myotonia and so is apt to fall. In other

cases the myotonia increases or is uninfluenced by exertion. The muscles of the legs are as a rule most affected, but sometimes all the muscles of the body may be involved.

Passive movement does not reveal the presence of any rigidity except that following voluntary contraction. The abnormality affects the voluntary contractions and relaxations of the muscles only, and the peculiarities of these are—(1) their slowness, (2) their tonic character, and (3) the continuance of the contraction after voluntary impulses have ceased. The peculiarities of electrical excitability bear the name of the “myotonic reaction” of Erb. The contraction, either on faradic or galvanic stimulation, lasts much longer than the normal and relaxes very slowly, and this is more marked the stronger the current used; with the stable application of galvanism, slow wave-like contractions of the muscle are seen to proceed slowly from the cathode to the anode. There is no pain, no sensory disturbances or loss, and the sphincters and reflexes are unaffected.

Diagnosis.—The only malady which can be confused with Thomsen’s disease is myotonia atrophica, in which the myotonic symptoms and signs are identical. In the latter malady, the onset is at a much later age, the incidence of the spasm is upon local groups of muscles, and the characteristic weakness of the facial muscles and atrophy of the sternomastoids, etc., at once distinguish it.

Course and Prognosis.—Thomsen’s disease has no tendency to shorten and destroy life. It tends to become more marked from infancy to puberty, and then less marked again as age increases. It has never been cured.

Treatment.—This is entirely unavailing, except in the way of the avoidance of those conditions, such as fatigue, cold and hunger, which are known to increase the condition. Thomsen himself, who was afflicted with the disease, was always better with free exercise.

DYSTROPHIA MYOTONICA

Synonym.—Myotonia atrophica.

Definition.—A disease of familial incidence, which begins usually in the third and fourth decades of life, and which is characterised by muscular atrophy of peculiar distribution and unlike that of any other disease. This atrophy occurs first and most in the sterno-mastoids and facial muscles, next in the muscles of the forearms, and may also be found in the muscles of mastication, in the vasti, and in the dorsiflexors of the feet and peronei. Associated with this wasting, but not commensurate with it, nor necessarily occurring in the same muscles, is a peculiar difficulty in relaxing the muscles after effort, called “myotonia,” which gives to this malady an especial feature which at once separates it from all other forms of muscular atrophy. Signs of bodily dyscrasia are often present, the most important of which are cataract, premature baldness, atrophy of testicles, loss of sexual power and general bodily wasting. This disease was first placed upon a firm clinical basis by Batten and Gibb, and Steinert in 1909. Curschmann in 1912 adopted the term *Dystrophia Myotonica* as being more correctly descriptive.

Ætiology.—This condition is probably always familial, and the heredity is homologous—that is, it tends to appear in the same child-rank, in a number

of apparently unconnected families at a common distance from one and the same ancestor, and often it seems to be entirely confined to one child-rank. The descent of the latent tendency is equally through the males and females, but the males more frequently transmit. The presence of the heredo-familial disease in earlier generations is often betrayed by other signs, such as cataract, frequent celibacy, childless marriages, high infant mortality, and a dying out of certain branches of the family. The malady has been observed at the age of 10 years, but usually the onset occurs between the ages of 20 and 35 years. A large number of the patients have been unusually gifted and proficient in athletics prior to the onset. Both sexes may be affected. No exciting causal factors are known.

Pathology.—No definite changes have been found in the nervous system. The muscles presenting the myotonia have repeatedly been examined and found normal. In the atrophic muscles the morbid process singles out certain fibres especially, so that quick and thin fibres are found lying together. There is increase of the muscle nuclei round thick and thin fibres alike, though some atrophic fibres may be found with no increase of nuclei. Recent biochemical and electrographic studies by Brown and Harvey of a form of congenital myotonia in goats suggest that there is no functional disorder of neuromuscular transmission of the motor impulse, but that the disorder is in the muscles themselves.

Symptoms.—The onset is gradual and the course extremely slow. The first symptom to call attention may be, either the difficulty in relaxing after muscular effort—the clinging of the hand to the tool which has been grasped, the smile that is so slow to disappear—or the weakness and wasting of the muscles. The two chief signs of the disease—the myotonia and the wasting—seem to have no connection the one with the other, either as regards coincidence in time or place. The myotonia may appear years before there is any obvious wasting. Moreover, the muscles which show the most conspicuous myotonia are often those which are not wasted, and finally those muscles which waste greatly tend to lose any sign of myotonia which they may have had. The extent and the intensity of the muscular atrophy and of the myotonia show great variations. The atrophy may be widely spread, and many muscles may be myotonic, or the former may be severe and the myotonia slight, or both may be present in minor degree only. Lastly, there are cases in which only the atrophy or only the myotonia is present. The myotonia consists in an inability to relax a muscle immediately after it has been put into voluntary contraction, and the greater the effort used in contracting the muscle, the greater the difficulty with relaxation. The patient grasps one by the hand, and is unable to disengage the hand, but pulls it away still grasping, and it may take seconds to relax. He smiles quickly to a suitable stimulus, and the face remains fixed at the height of the smile for long after the emotion has vanished. In eating, his jaw becomes fixed, he is unable to perform any alternate movements in the muscles which are affected, but at a very slow rate. When the myotonia is severe and general, he is liable to fall like a log when walking, from inability to relax muscles which have been put into contraction. The myotonia is seen most often and to a greater extent in the flexor muscles of the forearm and in those of the face, but it may be quite general. In the same patient it may be very marked at one time and absent at another.

The muscular weakness and wasting usually have a most typical distribution, involving the sterno-mastoids and other muscles of the neck, the facial and masticatory muscles—giving rise to the sad “myopathic” face, the vasti of the thighs, and the dorsiflexors of the feet, and this is the usual order in which the muscles are affected. It is always in one or other of these groups that the wasting commences, but sometimes the sequence of muscles attacked is quite different. Fibrillation does not accompany the atrophy. The electrical reactions show a reduction both to faradic and to galvanic stimuli, with a tendency to a polar change. Some modification of the “myotonic reaction” is often superadded in those muscles which are wasted, and this usually is present in the muscles which are myotonic and are not wasted. This “myotonic reaction” consists in a very long, lasting contraction when the muscles are stimulated with every form of stimulus, and if the latter be strong it may last as long as 30 seconds.

The affection of the muscles of the face and jaw entails some alteration of articulation and phonation. The voice is low, it lacks intonation, and has a definite nasal quality. Sensibility is not affected.

The rule is for the muscle-jerks to be diminished or lost, and it is very rare for all the jerks to be present in any case.

Apart from symptoms and signs connected with the muscles, the most important sign of the dystrophy is cataract, which occurs in more than half of the cases. This cataract is often met with in otherwise healthy brothers and sisters of those who have the muscular changes, and in otherwise healthy members of earlier generations in the afflicted families. In succeeding generations after its first appearance, the age of occurrence of this cataract shows remarkable “anticipation”—that is, commencing at first as senile cataract, it appears at an earlier and earlier age with each successive generation, until with fully developed myotonia atrophica it appears in youth. The presenile cataract of the dystrophic generation begins as a star-shaped opacity, first in the posterior and later in the anterior cortical lamellæ, sometimes with fine point-like opacities scattered through the lens. It ripens quickly to a total soft cataract, with a small central nucleus.

The genital organs remain infantile in some cases; celibacy and childless marriages are common. More often sexuality is normal until the onset of definite symptoms, after which desire and power disappear. Early baldness is the rule. A general wasting of all the tissues of the body is seen in many cases. Ultimate atrophy of the testicles is usual.

Diagnosis.—There is no difficulty in the diagnosis when the distribution of the muscular atrophy is typical and when myotonia is obvious; it simply involves a recognition of the unique characteristics of the disease. When the myotonia precedes the wasting, the age of onset will distinguish this malady from Thomsen's disease, or myotonia congenita, and the oncoming of any sign of facial weakness or muscular wasting will make the diagnosis certain. When the myotonia does not appear until long after the wasting is apparent, the diagnosis is much more difficult.

Course and Prognosis.—This malady usually progresses very slowly, but occasionally very extensive and incapacitating wasting of muscles and weakness may develop within a year of the first symptom. Some cases seem to remain stationary for very long periods. The tenure of life is certainly short in all cases, and does not appear to be prolonged beyond the middle of

the fifth decade. The oldest patient reported in the records as still living was aged 50 years.

Treatment.—It has been found that the administration of quinine, grs. 10 to 15 daily, lessens the myotonia considerably. Neither electrical treatment nor massage has the slightest effect in altering the course of the disease.

JAMES COLLIER.

Revised by F. M. R. WALSHE.

MYASTHENIA GRAVIS

Definition.—A chronic malady of adult life characterised by—(1) a variable paralysis of muscles which is produced or rapidly increased by exertion, and which tends to disappear slowly during rest; (2) a permanent paralysis which shows no improvement with rest, and which succeeds the variable paralysis. This permanent paralysis may be very local in distribution, and may be associated with atrophy of the muscles; and (3) the affected muscles on strong faradisation soon cease to respond to faradism, but remain excitable by galvanism.

Ætiology.—The malady seems to be much more prevalent in England during the past 20 years than formerly. Rarely occurring before puberty, it commences most commonly in the third decade of life, and affects the sexes equally. Nothing is known of any causal factors either immediate or remote. The one clinical association which cannot be ignored is with exophthalmic goitre, for not only may myasthenia follow that malady, but the ophthalmoplegias which occur in Graves's disease bear no small resemblance to those of myasthenia.

Pathology.—The only changes found within the nervous system are slight atrophy of those nerve cells which supply long paralysed muscles, and these changes are certainly not primary. In a certain number of the cases a large persistent thymus gland, showing proliferative and degenerative changes, or thymic rests showing similar changes, have been found. The view expressed in earlier editions of this book that the seat of the disorder of function responsible for myasthenia gravis is at the myo-neural junction has recently received confirmation from observations made with physostigmine and the synthetic substance "prostigmine." It is believed that the normal transmission of impulses from nerve to muscle through the motor nerve endings depends upon the liberation in the endings of acetyl-choline. In myasthenia the failure of effective innervation may be due either to premature destruction of acetyl-choline, or to a failure in its liberation. The administration of physostigmine temporarily delays the destruction of the acetyl-choline by the choline esterase normally present in the blood, and during its period of activity renders muscular contraction normal.

Symptoms.—The first sign of myasthenia is the variable paralysis which may commence in any of the voluntary muscles. It may be unilateral at first, but soon becomes symmetrical. The paralysis appears first upon exertion and fatigue. The schoolmaster finds that towards the end of the day's work he cannot raise his arm readily to write upon the blackboard, or that his voice fails him in speaking. The housemaid finds that her broom

slips in her hands, because of an ever-weakening grasp. The theatre-goer, towards the end of the performance, finds himself tilting his head farther and farther back to escape from an oncoming ptosis, or he develops diplopia. Next morning these symptoms are gone with the night's rest, to reappear with fresh exertion perhaps earlier each day, until work becomes impossible. The affected region may be narrowly confined, the eyes alone, the face, the muscles supplied by the trigeminus, or the larynx alone may be affected, or it may be the muscles of the upper extremities, or of the lower extremities, or of the back which may be solely involved. Lastly, the myasthenia may be quite universal, though never in the same degree in all the affected regions.

The incidence is greatest upon the muscles innervated from the brain stem, next upon the upper extremities and trunk, and least upon the legs. When the initial incidence of the malady is upon the eye muscles, diplopia and ptosis are the first symptoms. The paralysis is usually of the nuclear type, involving both eyes in terms of the conjugate movements; but inequality in the paralysis upon the two sides usually gives rise to decided strabismus and diplopia, and we have observed one case in which the initial paralysis was confined to one external rectus. When the permanent paralysis sets in, the axes usually become parallel, and the diplopia ceases. Until this event occurs, the great feature of myasthenic ocular paralysis is its variability, and its increase with fatigue. An ocular paralysis which is well each morning on waking, and which develops in the course of each day, is always due to myasthenia. Short, very quick, jerky movements of the eyes on attempted voluntary movements are often characteristic, and are quite different from the movements of nystagmus. Though ptosis is the rule, sometimes there is retraction of the lids, and both von Graefe's and Stellwag's signs may be present. Permanent nuclear ocular paralysis follows the variable paralysis in nearly every case, though it varies in degree. When the face is affected, epiphora, dysarthria and lack of facial expression with a peculiar weak "nasal smile" are conspicuous. The facies of myasthenia, with inability to close or pucker the eyes and mouth, the motionless and slightly dysarthric speech and peculiar smile are unmistakable.

Involvement of the muscles supplied by the fifth nerve causes trouble with mastication, and when the palate is affected there may be nasal speech and regurgitation of liquids. We have seen one case in which total unilateral paralysis of the larynx preceded the onset of typical symptoms by twelve months. When the tongue is affected it usually shows some wasting, especially of the linguales, and dysarthria results. Sometimes a widely spread involvement of this region causes severe dysphagia and dysarthria, and the former may be so great as to necessitate nasal feeding. The permanent paralyses are rarely seen, except in the muscles supplied by the nuclei of the brain stem. The neck muscles are usually affected when the malady extends on to the trunk, and it is a common thing to see the patients sitting at rest, either with the head supported by the hands, or resting upon the table. In the extremities the variable paralysis appears more marked at the proximal joints in most cases, and the test for myasthenia in the upper extremity is to ask the patient to extend his upper limbs level with the shoulder, when they will be seen to fall slowly down from the increasing fatigue paralysis.

Involvement of the respiratory muscles is common, and constitutes the gravest danger in the disease, since any effort, and especially an emotional

outburst, may in a few seconds fatigue the respiratory muscles into a complete and fatal paralysis. Myasthenia often remains long confined to one region, and subsequently spreads rapidly. Wasting of the muscles occurs only when there is marked permanent paralysis, and may be seen in the muscles of the tongue, face and in the masticatory muscles. In one fatal case under our care there was marked wasting of the intrinsic hand muscles on both sides.

The myasthenic reaction is only seen in those muscles which are showing conspicuous fatigue paralysis. If a strong interrupted faradic current is applied to such muscles, there is at first a strong contraction, but this is not maintained as in normal muscles, and it rapidly decreases until there is no response. If the stimulation is discontinued for a few minutes, and again applied, there is a further response, which tires more rapidly than the first. After exhaustion of the muscles by faradism, some volitional contraction remains. Exhaustion of the muscles does not occur from galvanic stimulation. Sensibility and sphincter control are not affected. The reflexes are normal in all but the rarest cases. The knee-jerk is not abolished when the quadriceps is exhausted, either by voluntary exertion or by faradisation.

Diagnosis.—This is never a matter of any difficulty if the variable paralysis, increasing with fatigue and lessening with rest, is conspicuous, for this phenomenon occurs in no other disease. When permanent paralysis only is present, the diagnosis requires care. It must be remembered that any unilateral or bilateral palsy of muscles supplied by the brain stem may be myasthenic. Here the history of a slow onset with variable paralysis and fatigue phenomena can nearly always be obtained, and the absence of the usual signs of gross lesions of the brain-stem nuclei, or progressive diseases affecting the latter, should avoid confusion. When, as sometimes happens, myasthenia begins with a unilateral ophthalmoplegia or laryngoplegia, the diagnosis may really be difficult. The possibility of such a commencement should be borne in mind, and a careful watch kept for the appearance of conclusive evidence. Other forms of nuclear ophthalmoplegia do not show a long history of variability and fatigue phenomena.

Course and Prognosis.—Myasthenia is always a very dangerous disease, as the term "gravis" implies. Some of the cases, and especially those in which the brain-stem region escapes, recover completely; but no records are as yet available to prove in what proportion of the cases this event occurs. The disease has proved fatal within a fortnight of the onset of symptoms, and, on the other hand, in our two cases here recorded, strenuous work was followed for 24 years after the development of permanent ophthalmoplegia. Improvement and relapses are very common. Frequently a patient will get rapidly worse, and become bedridden, in spite of careful treatment; to recover completely for the time being, when treatment has been abandoned as useless. Sometimes a patient with severe myasthenia will live for many years, if life be carefully protected. We have had one patient under observation in this state for 20 years, and she is not materially worse.

Sudden death is very liable to occur in any of the cases, but especially in those with bulbar paralysis and implication of the muscles of respiration. Death has been attributed to respiratory failure and asphyxia; but some of the patients die much too quickly for any such explanation. Two of our patients, seated at a table with their heads supported by their hands, and

engaged in pleasant, quiet conversation, smiled, lowered their heads on to the table, and were dead without the slightest sign of distress or reaction, as if from syncope.

Treatment.—The life must be so ordered as to exclude all fatigue. Massage, electrotherapy, endocrine therapy and strychnine medication are ineffective when not harmful. Claims have been made for many different drugs and preparations, but they are based upon an ignorance of the fluctuating course of the malady and of its occasional spontaneous cure. Few things in clinical medicine can be more dramatic than the rapid and complete disappearance of all weakness and disability upon the hypodermic injection of prostigmin 2 to 4 c.c. (1 to 2 mgms.) combined with atrophine sulphate, gr. $\frac{1}{100}$. This abeyance of symptoms endures for some four or five hours only. When the effect passes off the patient lapses into his original state, or may even be weaker than before. In severe cases, this possibility of increased weakness after physostigmine or prostigmin administration has its dangers. It is not yet possible to use these drugs by repeated injection as a mode of treatment for myasthenia gravis, though their occasional administration may enable the subject to meet special calls from time to time. The drug may be given orally in tablets of 15 mgms., four to eight daily.

A few patients maintain an improved level of power in the affected muscles when given ephedrine sulphate or chloride (grs. $\frac{1}{2}$ to 1, t.d.s.).

FAMILIAL PERIODIC PARALYSIS

Definition.—A flaccid paralysis affecting the muscles of the trunk and of the extremities, associated with loss of the deep reflexes and diminution or loss of faradic excitability in the muscles. The paralysis is temporary in character, though it may be fatal during the attack, and it recurs at intervals. It is a rare malady, some 200 cases having been reported in the literature.

Ætiology.—It has been noted as early as the fifth year, and as late as the thirtieth year; but usually it appears about the age of puberty. Most of the cases occur in the male sex. Heredity is very marked, and the malady has been traced through five generations. Transmission may occur either through the male or through the female, and not infrequently a generation is skipped. Several members of the same family are usually affected.

Pathology.—Several cases have come to autopsy, but no lesion which could be associated with the symptoms was found. Biopsy of the muscles has given entirely negative results.

Symptoms.—The clinical picture is so striking as to be almost dramatic. The patient retires to bed feeling perfectly well, and awakens in the morning without pain or malaise, but with a flaccid motor paralysis, which always involves all four extremities, and which may reach all the muscles of the body, except those of the organs of speech, deglutition and respiration, and even these are often partially involved. Severe involvement of these vital muscles during an attack has caused death. The bladder and rectal functions are retained, and it is unusual for the patient either to void urine or feces during the attack. The paralysis is usually at its height on waking; but it may subsequently increase. After lasting for a variable time, from a few hours to

a few days, it passes off, sometimes gradually, sometimes rapidly. In the family under our care it was astonishing how the patients on waking in an attack could judge invariably how long the particular attack would last. They could judge with unfailing certainty when ability would return, and were in the habit of arranging their business accordingly. Most of the patients in addition to the severe attacks of paralysis suffer from what they call "morning weakness," temporary inability to grip with the hands, and slight disability with the feet on waking. It is curious that similar morning weakness, lasting a few minutes, is not very uncommon in normal children. The paralysis in periodic paralysis is flaccid, and there is loss or marked diminution of response to faradism during the paralysis. The deep and superficial reflexes are lost in the paralysed region. Objective sensation is not affected; but there may be subjective sensations of tingling and numbness, and the muscles may be a little sore and stiff after the attack. We have noticed flushing of the surface and sweating during the attack. There is an invariable tendency for the attacks to diminish in frequency and severity after middle life is reached.

Diagnosis.—This must be evident to any one acquainted with the symptoms of the disease.

Treatment.—Potassium chloride in large doses (up to 30 or 40 grs.) will avert or cut short an attack. No other remedial measure is known.

MUSCULAR DYSTROPHY; MYOPATHIC ATROPHY

Synonym.—The myopathies.

Under this heading, a disease is described in which the voluntary muscles undergo primary degeneration, independent of detectable disease in other parts. To facilitate description, a number of clinical types have been distinguished according to the age at which the disease appears, to the group of muscles first attacked, to the presence or absence of pseudo-hypertrophy, or to the prominence of the hereditary factor. The chief of these are—(1) the pseudo-hypertrophic type; (2) the juvenile type of Erb; (3) the facio-scapulo-humeral type of Landouzy and Dejerine; (4) the distal type.

The first type is fairly constant, but there is in reality no sharp division between the different forms. That the others do not represent separate diseases is proved by the appearance of more than one of them in members of the same family. The disease is familial, and it is also hereditary in the sense that it may appear in some or all the members of a family through several generations.

The changes in the muscles in the myopathies are the same as those which occur when muscles degenerate from any other cause, namely, a slow and progressive atrophy of the contractile elements, with a concurrent increase of fat and fibrous tissue. In the pseudo-hypertrophic form the connective-tissue hyperplasia is excessive in some of the affected muscles and their bulk is increased. In the other forms of the disease, and in those muscles in the pseudo-hypertrophic form which become weak without any increase in size, the overgrowth of connective tissue may balance the loss of bulk due to atrophy of the contractile tissues, and the diseased muscles retain their normal size; or atrophy may proceed faster than hyperplasia, and the muscles waste from the beginning.

1. PSEUDO-HYPERTROPHIC PARALYSIS

Ætiology.—The cause of the disease is unknown. In many instances no antecedent cases can be traced in the family. In others, a family history is obtained, always on the mother's side. Isolated cases occur, but more often several children are attacked in each generation. Boys suffer more frequently than girls in a proportion of about 5 to 1. Sometimes one sex alone suffers, sometimes both. It is rare for all the children to be attacked. The males who escape beget healthy children, whilst the females, who appear to have escaped, may transmit the disease to some of their offspring.

Symptoms.—The symptoms appear in early childhood. The onset is often delayed to the fourth or fifth year, rarely until towards puberty, and very rarely until as late as the twentieth year. In cases of late onset, enlargement of the calves has usually been present for many years. Weakness appears first in the muscles of the pelvic girdle. The child, who usually looks fat and strong, begins to walk late, he falls easily, and rises again with difficulty. He does not romp as other children do. He cannot skip or jump, and he has great difficulty in mounting stairs. At first the muscles may be normal in size, but, as a rule, some show obvious enlargement before the fifth year is reached. The enlargement is most conspicuous in the calves, the buttocks and the infraspinati. The erector spinæ, the quadriceps in whole or part, the deltoid, the supraspinatus and the triceps often show considerable hypertrophy. Occasionally the masseters are enlarged. At the same time other groups of muscles atrophy. This is most severe and most frequent in the latissimus dorsi and in the lower part of the pectoralis major. Later it extends to other muscles, and ultimately to those which were at first hypertrophied. The neck and face are spared. There is no exact correlation between the size of the diseased muscles and their power, but weakness is usually greatest in those which show most atrophy. The defects are greater in the proximal muscles, and diminish distally. The hands often retain good power to the end. This distribution of paralysis gives rise to certain characteristic defects of attitude and movement.

In standing the legs are placed far apart, and the upper part of the trunk is thrown back, so that a plumb-line from the most prominent vertebra falls behind the sacrum. This attitude compensates for the forward tilting of the pelvis, resulting from weakness of the glutei, which normally raise the anterior border of the pelvis by lowering its posterior border. In the sitting posture the lordosis disappears, for now the attachments of the flexors of the hip are approximated, and these muscles no longer lower the anterior border of the pelvis. On lying down the lordosis appears again, but can be abolished by relaxing the flexors of the hip-joint, that is, by flexing the hips passively. In walking, the feet are widely separated, and to clear the ground with the advancing foot the body is inclined first to one side and then to the other. This "waddling" produces a gait like that seen in congenital dislocation of the hip. The early preponderance of weakness in the extensors of the hip and knee is betrayed by the great difficulty experienced in mounting stairs.

The manner in which the child rises from the supine to the erect position is almost pathognomonic of the disease. He first tries to sit up, but fails. He then rolls over on his belly, and raises himself first on his knees and elbows, and then on his hands and feet. Next he places his hands on his

knees, and as it is impossible for him to raise the trunk actively owing to weakness of the extensors of the hip, he literally climbs up his thighs, pushing the trunk passively almost to the erect position. The remaining power in the extensors may be enough to enable him to complete the movement. If not, he jerks the shoulders back suddenly and gains the erect posture by a writhing movement, whose details are difficult to follow. To climb the thighs successfully a certain amount of power is necessary to hold the knees slightly flexed. When this power is lost he is no longer able to rise. The arms are also used to assist the weak legs in sitting down and in getting up from a chair.

As time goes on the weakness increases, and invades all the muscles of the trunk and limbs. Some of the muscles become shortened, and distortions are produced by permanent alterations in the position of the joints. The knees and elbows become flexed, the feet take up the attitude of talipes equinus, the spine becomes curved, and the whole body is grossly deformed.

The deep reflexes and the electrical excitability of the muscles diminish gradually as the wasting increases. Sensation is unaffected. The sphincters are not involved. The mental condition shows no abnormality.

Diagnosis.—The diagnosis is usually simple if a few of the outstanding features of the disease are known. The defects of attitude and movement, especially the mode of rising from the supine position, together with the characteristic association of enlargement of the infraspinati and calves with atrophy of the latissimus dorsi, form an unmistakable combination.

Prognosis.—This is most grave. Few patients reach adult life, and most die within 10 years of the onset of the disease.

Treatment.—Drugs have no beneficial influence. Massage and passive movement are useful in the prevention of contractures, and the efficiency of the muscles may be prolonged by suitable exercises. Walking should be practised daily, until it becomes impossible. Very often this is lost owing to contractions of the calf muscles, and is regained after tenotomy.

2. OTHER TYPES OF MUSCULAR DYSTROPHY

Ætiology.—The separation of the remaining types of myopathy from the pseudo-hypertrophic form is not an absolute one, as isolated cases are occasionally met with which seem to form a connecting link between the several varieties. The varieties, however, are habitually separate in occurrence, and in families in which numerous cases conforming to the types to be described hereunder have occurred throughout several generations, no cases presented the peculiar features of the pseudo-hypertrophic form. Moreover, the sex incidence as well as the period of onset is different in the two varieties, and it is possible that there is some essential pathological difference between them, and that they are separate diseases. With regard to the types of myopathy unassociated with pseudo-hypertrophy, no doubt exists as to their fundamental unity. They are merely varieties of one disease.

The influence of heredity is much more prominent than in the pseudo-hypertrophic form. Isolated cases occur, but they are rare. In most instances several members of a family are affected in the same and in succeeding generations.

The sexes suffer equally. The time of onset varies within wide limits—

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from infancy to old age. When the wasting begins in the face (*facio-scapulo-humeral type*) the disease frequently begins in childhood; but sometimes it begins there late in life. In the cases where it is first noticed in the muscles of the shoulder and pelvic girdle the onset is most frequent between the ages of 15 and 35 (*Erb's juvenile type*); but here, again, it may begin in childhood or early old age, and the term *juvenile* is hardly applicable to it. The same variations in the age of onset are noticeable in cases where the atrophy begins in the forearms and legs (*distal type*).

The various types may be exemplified in members of the same family, and in the same family the age of onset may show extreme variation.

The cause of the disease is quite unknown.

Symptoms.—In the so-called *juvenile form* weakness and wasting come on simultaneously. In most cases they are first noticed in the arms; but in some families the legs suffer first. Of the arm muscles the biceps, triceps, and supinator longus are most often first affected. The lower part of the pectoralis major, the latissimus dorsi, trapezius and rhomboids are attacked in most cases. Atrophy of the serratus magnus is common; but it may escape even in severe cases. The deltoid, supraspinatus, infraspinatus and subscapularis usually escape. Atrophy of the forearm and hand muscles is rare.

In the legs, the flexors of the hip, the extensors of the knee and the glutei are most frequently affected. The muscles below the knee often escape entirely.

In the face the zygomatic muscles and the orbicularis are attacked. The face is dull and expressionless, the naso-labial fold is obliterated, the lips are habitually separated and the lower lip projects—*myopathic facies*. The face does not light up in conversation, in blinking the eyes are incompletely closed, and the articulation of labial consonants is defective. In smiling the mouth forms a straight line, instead of its angles being drawn upwards and outwards by the zygomatici. The power of whistling is lost. When the patient closes his eyes, or compresses his lips as forcibly as he can, they can be forced open with great ease. The buccinators are often affected, the tongue and the masticatory muscles never. The spinal muscles often atrophy, and in a few cases the abdominal muscles have been involved. The excitability of the muscles to faradic and galvanic stimulation usually diminishes in proportion to the wasting. The muscles never show fibrillary tremors. Sensibility is unaffected, and all the other functions of the nervous system are normal. Deformities are neither so common nor so severe as in the *pseudo-hypertrophic form*.

Diagnosis.—When a family history of atrophy is obtained, *myotonia atrophica* and *peroneal muscular atrophy* must be excluded. *Myotonia atrophica* is distinguished by the peculiar prolonged response of some of the muscles to voluntary, electrical and mechanical stimulation, and by the distribution of the wasting. Atrophy of the sternomastoids, which is constant and severe in *myotonia atrophica*, is never seen in the forms of *myopathy* now under consideration. In *peroneal muscular atrophy* the combination of atrophy in the lower limbs and small muscles of the hands, together with sensory disturbances in the lower limbs, is distinctive. In an early case, when the hand muscles are still normal and sensory changes are absent, the differentiation from *myopathy* may be impossible for a time.

In isolated cases myopathy is suggested by the appearance of muscular atrophy in childhood or youth. The diagnosis of myopathy is based on the distribution of the wasting in the absence of any sign of disease of the nervous system.

Prognosis.—The disease shows wide variations in its course and duration. The atrophy may remain confined to the group of muscles in which it begins, or extension may take place after an interval of several years. It rarely extends beyond the muscles mentioned above. In most cases, even in those that begin in childhood, progress is extremely slow, and as no symptom of the disease is necessarily fatal, death usually results from other maladies unconnected with the disease.

Treatment.—Owing to the variable course of the disease, it is impossible to estimate the value of any treatment that may be employed. Massage, and especially voluntary exercises designed to bring the weakened muscles into play, seem sometimes to retard the progress of the disease.

AMYOTONIA CONGENITA

Synonyms. — Oppenheim's Disease ; Myatonia Congenita.

Definition.—A malady of early childhood, usually congenital and sometimes familial, characterised by extreme flaccidity, smallness and weakness of the muscles, which are not actually paralysed, by lowering of the faradic excitability of the muscles, by loss of the tendon jerks, and by contractures in the region affected.

Ætiology.—In most cases the disease is present at the time of birth ; in a few cases it has appeared during the first year of life in an apparently healthy child, and sometimes following an acute illness, such as bronchitis or diarrhœa. Usually sporadic, it has occurred in several children of the same parents.

Pathology.—The chief morbid changes are found in the muscles. In these very conspicuous pathological conditions are present, closely resembling those found in the myopathies. The three most striking conditions are—(1) the minute size of the majority of the muscle fibres, from 7μ to 12μ ; (2) the presence of a few very large or "giant" fibres reaching 140μ in diameter, and larger than any fibre occurring in normal muscle ; (3) marked regressive changes are seen in the giant fibres. There is increase of the connective tissue between the muscle bundles and a notable determination of fat. Reduction in numbers of the ventral horn cells of the spinal cord occurs, and the ventral roots are small and poorly myelinated.

Symptoms.—The extreme flaccidity of the affected muscles is noticed from the time of birth. They are small and weak, and though there is no muscular wasting and no absolute paralysis, yet in many cases the limbs cannot be raised against the action of gravity, nor can the head be held up. The great relaxation of the muscles and ligaments allows of the most fantastic attitudes being assumed without pain. When the child gets older, he is unable to sit up, but when placed in the sitting position the spine bunches up from absence of any muscular support, and he is unable to support his weight upon the weak legs. The amyotonia is symmetrical, and affects the legs always, the trunk often, the arms not infrequently, but never the face.

Notwithstanding the flaccidity, some degree of flexor contracture is usually present. The faradic excitability of the muscles is much lowered, but not lost. Sensibility and the sphincters are not affected. The superficial reflexes are normal, but the deep reflexes are invariably absent in the affected regions. The children are usually intelligent, with good bodily development, and growth proceeds normally.

Diagnosis.—This presents no difficulty on account of the presence of the flaccidity at birth, the absence of the deep reflexes, and the tendency slowly to improve. It has to be separated from those maladies to which it bears a superficial resemblance, namely, the myopathies, rickety weakness, obstetrical, infantile and diphtherial palsies.

Course and Prognosis.—Some of the children succumb during the early and severe stages of the disease, but the tendency of the disease is to improve slowly in the course of years, and in some cases almost complete recovery with return of the knee-jerks occurs.

Treatment.—This consists in aiding the natural tendency to improve with massage, passive movements and exercises, in treating contractures with tenotomy, and in attending to the general health and nutrition.

JAMES COLLIER.

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SECTION XXI

PSYCHOLOGICAL MEDICINE

INTRODUCTION

PSYCHIATRY is concerned with forms of illness as widespread and diverse as those of somatic medicine. There are almost as many beds in mental hospitals as in all other hospitals put together ; and there is an undoubtedly large, if unnumbered, part of the population who have mild mental disorder not needing mental hospital care : hysteria, obsessional neurosis, hypochondria, chronic depression, paranoid states, and so forth. The diversity of this widespread group of illnesses depends on their being disorders of mind—disorders, that is, of the human function which comprehends and sums up all other functions of the organism, serves to relate a human being to his complex environment, and is the chief token that he is an individual, and not a sample. Mental disorders are therefore varied, as are the people who suffer from them. It is only by ignoring most of what is individual in these illnesses that a few common types or categories can be recognised, comparable to the “diseases” of somatic medicine. Such a procedure is necessary for practical ends ; material must be classified. Moreover, a biological foundation may be assumed for the syndromes with which psychiatry works. They stand for the main ways in which a human being can become mentally unhealthy. There are only a few such ways, and they are determined by the structural and functional patterns inherent in the organism. Diversity arises through their becoming manifest under the influence of each individual's special environment and in combination with his other inherited tendencies. Diversity, therefore, can be due to a combination of single hereditary causes and to the effect of each individual's environment throughout his life upon his development and behaviour. There is always interplay between inheritance and environment. Part of the psychiatrist's business is to discover how this interplay has led to the present illness. The interplay, moreover, is sufficiently varied in the course of each patient's life to make prognosis and the effect of treatment a matter of individual study, rather than of summary inference from the diagnosis, once made.

Treatment is only another special instance of the environment acting on the patient ; its power and limitations for him must be studied by seeing what effects this or that experience has had on his previous life. Consequently the psychiatrist, even more than the general physician, must study illness in two ways : first, as showing some typical pattern of morbid behaviour, and tending to run along well-known lines ; and secondly, as a patch of personal biography, something to be understood, rather than classified in terms of psychology and biology. The two methods are complementary,

though, in a brief text-book presentation, the former must be the more prominent.

There is no dividing line between somatic medicine and psychiatry. Psychiatry, although it has to work in part with social and psychological conceptions of which general medicine has felt little need, suffers greatly when it limits itself to this way of regarding mental phenomena. It cannot safely ignore the relationship between bodily happenings and the patient's state of mind. Crude instances of this relationship are the delirium that accompanies an acute fever and the irritable fatigue (*neurasthenia*) that may follow it; the insanity that is due to cerebral tumour or general paralysis of the insane; the obsessional neurosis that follows *encephalitis lethargica*; and the hysterical symptoms of disseminated sclerosis. There is no mental disorder, mild or severe, in the causation of which bodily disease may not play an important part. Moreover, it is not only in crude instances of structural or chemical disease that the relationship between bodily and mental illness may be recognised. A human being does not exist as a rarefied mind united with a solid body; he is an organism all of whose subsidiary functions contribute to this highest function—his mind—which brings him not only consciousness, but also an integrated behaviour in relation to his surroundings. Disturbances, transient or permanent, of these part-functions (for example, in the sensory apparatus or the circulatory system) will have some effect on his state of mind. Changes in the central nervous system are the most obvious instance of this, but the endocrine glands, the autonomic nervous system, and the metabolic processes are often of notable significance in the various maladjustments summed up as mental disorder. A human being is constantly responding to, and influencing, his surroundings; but his doing so is conditioned by the various parts of his body and the way they are working.

Before the categories and clinical features of mental illness are described, the principles of psychopathology, prognosis, and treatment call for some very brief consideration, since without them psychological medicine written down is a repellent catalogue of details. Though the principles set forth may seem trite or too obvious to be worth stating, it is unfortunately the case that they are seldom applied as fully as they might be to the clinical study and treatment of mental disorders.

Psychopathology.—**INTRINSIC CAUSES.**—The intrinsic causes of mental disorder are those which depend on heredity and on normal phases of development, *e.g.* puberty or the climacteric. Extrinsic causes, which come from the environment, are either mental experiences or physical damage. The distinction between intrinsic and environmental, like that between physical and mental, is convenient but artificial; a long sequence of related happenings both within and without the patient's body goes to the causation of any mental illness. It is, of course, possible in many instances to discover some indispensable link in this chain of causes—an intoxication with alcohol, for example, a syphilitic infection of the brain, an inherited predisposition to periodic insanity, a bereavement—which may legitimately be singled out as the chief cause and classified as intrinsic or extrinsic, but this is more valuable for formal and didactic purposes than clinically. Actual cases usually show a complicated *ætiology*. Thus, a man whose parents had both been subject to melancholia became himself profoundly depressed after the death of

his wife, and attempted suicide by drowning. He survived, but during the resulting pneumonia he was delirious and threw himself from an upper window, crying out that he must go to his wife. The causes of the mental disturbance in this case were many and obvious; numerous they always are, but not always obvious. One cause may, of course, be prepotent.

The more detailed the analysis of a patient's endowment and experiences, the more entangled physical and psychological, internal and external factors seem to be.

Heredity and constitution.—The hereditary factor is not a general neuropathic taint; there are specific predispositions to one or other anomaly. These predispositions are transmitted in accordance with familiar genetic principles, summed up in the modern gene-theory of inheritance. Studies of families and of twins have proved the importance of the hereditary factor in the major non-organic psychoses, though they have not yet sufficed to reveal the number of genes concerned in the transmission of the hereditary types of morbid reaction.

Among the main reasons for this incompleteness in our knowledge is the impossibility of concluding that an inherited trait is not present, merely because it is not manifest in some recognisable form. Other inherited factors and, most of all, the environment, will in many cases determine whether an individual predisposition is to become evident or not. Thus a man may have an inherited tendency to melancholia which remains latent until a financial reverse or disease of the cerebral arteries provides the conditions necessary for its manifestation. It is true that some inherited predispositions, *e.g.* to Huntington's chorea, are almost independent of the environment in this respect, but such are exceptional.

Since the Mendelian and other conceptions of modern genetics are held to obtain for the heredity of mental disorders, it can be understood that more than one type of proneness to mental disorder can be inherited by the same person. He may, for example, be prone not only to periodic insanity, but also to schizophrenia. Mingled proclivities of this kind account for anomalous clinical pictures, frequently met with and difficult to classify as either one syndrome or another. The "either-or" kind of diagnosis is often out of place or misleading in psychiatry because of the commonness with which more than one constitutionally rooted type of illness may be found in the same patient. Syndromes are frequently combined; to grasp their clinical meaning it may be indispensable that one investigate the patient's family not only as to mental disorder, but as to normal characteristics too.

The signs of a transmissible tendency to some mental disorder may not be actual illness, but only a special kind of personality. There are certain varieties of personality which show some or all of the essential features that characterize certain types of mental illness; the differences between personality and illness seem then to be of degree rather than of kind. Moreover, those who manifest one or other type of illness are often found to have had the type of personality that is functionally similar to it. So close may the similarity be that it is difficult to decide when the illness has begun, because there was no sharp dividing line, in time or in form, between the more or less normal previous personality and the actual disorder. This frequency of association and similarity of form

between the normal state and the illness points to the constitutional background of mental illness, and shows how hereditary tendencies can express themselves in more or less normal ways in personality before the catastrophe of an obvious illness has directed attention to them. Nor is it only in the personality that inherent proclivities may be revealed; certain types of bodily structure, too, occur much more frequently in those with a particular mental constitution or mental illness than in the rest of the population. The most striking instance of this is the frequency with which a "pyknic" bodily habit and a "syntonic" personality are found among those who have periodic attacks of mania or melancholia (see p. 1855). Such constitutional features, whether mental or physical, indicate that inherited tendencies can body themselves forth in normal physical and psychological structure before morbid exaggerations of them make an appearance. The varieties are sometimes called by appropriate names, *e.g.* schizoid, cyclothymic, syntonic, obsessional, hysterical, paranoid. The relationship is not a simple one. There are very many people with these types of personality who never fall mentally ill.

A pronounced personality, belonging to one or other of these types, does not indicate that the person who exhibits it is likely to have a mental illness, but only that if he should have a mental illness, it will probably be of the corresponding type. As with all inherited anomalies of which the crude manifestation is delayed until adult life, there may be for many years none or only mitigated signs of the proclivity; these may be indistinguishable from what occurs in normal people. The more pronounced the anomaly of personality, the more likely that it portends a mental illness, or, at any rate, a proclivity to the mental illness in specially adverse circumstances.

In the foregoing, personality and constitution have been spoken of as though they were static, innate attributes of the human organism. Neither of these epithets, however, is appropriate, not even in respect of bodily constitution. Responsiveness and plasticity are essential to human development of every kind; there is a constant interplay of personality with the outer world, modification of it and by it. The main pattern of development is doubtless determined by innate, inherited factors—bodily structures grow, instincts come into play, and the general direction of functional activity is predetermined. But general directions and main patterns mean little unless they are given body and content by individual experience. Nutrition, for example, can deflect the body from its ordained pattern or enable its fulfilment; all sorts of physical interference can maim it or improve it: the same is profoundly, if obviously, true of the mental side of human growth and maturity. Consequently, each patient's personality is not only to be assessed as conforming to a frozen artificial type, but as a complex of dynamic functions, changing in outward form, sometimes in unstable equilibrium, and none the less powerful for being subterranean. Here, as was said earlier of psychiatry in general, there must be two ways of viewing the data: in classes, and as individual living biographies to be understood rather than schematised. Both methods are necessary to any complete psychopathology.

Phases of development.—A concrete instance of the foregoing is the change that occurs at certain turning-points, such as puberty, pregnancy, or the climacteric. Endocrine and other physical changes at these epochs are

accompanied by psychological disturbances, the severity and form of which may bring them under the notice of the psychiatrist. They are dramatic episodes in a lifelong and universal process of growth, maturity, and involution or decay, which is marked by plasticity and development of varied functions in the first stage, stability and differentiated adaptation in the second, emotional lability and suspicion, intellectual narrowing of interests, rigidity, failing grasp and memory, in the last. The mental disturbances which may occur at different ages are much influenced by these intrinsic factors and tendencies.

EXTRINSIC CAUSES.—The outer world impinges on human beings from the day of their birth, or even their conception, in more and more complicated ways, as they themselves become more complicated. In other words, the environment is, for the individual, as complicated as he can make it; and that will depend on how far he has himself developed hitherto. Human beings deal selectively, not merely passively, with experience. At each stage of their growth, previous experience helps to determine what they will select from their environment, and how they will use this and integrate it, to serve in its turn as the partial determinant of further growth and integration (the other determinants being innate ones). "Experience" is here being used in the widest sense to denote the response of a human being to the impact of the outer world upon him, whether it be consciously recognised as such or not at the time.

It is, therefore, impossible to give adequate consideration to any aspect, including the psychological, of a human being's way of dealing with the outer world unless one pays regard to his previous experience, mental and physical, and to the present state of his whole organism, mental and physical. The cultural milieu in which he has grown up must be taken into account. Too partial a regard for subsidiary functions, whether physiological or psychological, may lead one away from the living human being, who is an integrated organism, not a collection of disparate mental and physical systems; similarly, too concentrated a gaze on this latter aspect, *i.e.* on the socially organised person, to the neglect of part-functions, may make one see only a disembodied spirit, as remote from medicine as from daily life.

Physical experiences.—Some external happenings influence the mental state chiefly by way of the body: infection, physical trauma, intoxication, and metabolic and endocrine disturbances due, wholly or in part, to environmental influences may result in mental disorder. In many of these instances, the mental change is mediated by way of some cerebral damage, and the clinical picture is of the organic neurogenic kind, *e.g.* dementia. It would be wrong to attribute the whole of the mental disorder to the cerebral damage; but to it is referable the core of the psychosis. Some diseases have an incidence on special functions and parts of the central nervous system, which determines characteristic features in the mental picture, *e.g.* the anxious fidgetiness of the patient who has had chorea, the stiff mind and obsessional thoughts and movements of the post-encephalitic, the hysterical phenomena of the elderly arterio-sclerotic patient or the man poisoned with carbon monoxide, the aphasia and apraxia of the post-apoplectic, the silly "moria" of the cerebral tumour. In the main, however, it is not possible to correlate mental symptoms with special areas or kinds of cerebral damage—partly because the brain is not the only structure concerned, partly because it acts as a whole,

and also because the presumptive changes in it are too evanescent and delicate to be accessible to our crude methods of examination.

To limit oneself to the brain in studying the somatic correlates or basis of mental phenomena would be an error. In the physical accompaniments of emotion, the whole body participates through the mediation of the vegetative nervous system and the endocrine glands. This is significant, because emotional upset is one of the most important phenomena of mental disorder. The sequence of psycho-physical happenings of which an emotional upset is the climax and the outward sign, may be started not only by some mental happening, but also by physical experiences—intoxication with a drug, or a circulatory disturbance, or a metabolic upheaval such as acute hypoglycæmia. Whether, for example, this hypoglycæmia comes from outside, as an injection of insulin, or arises (as it rarely may) from within the body, as a “spontaneous” deficiency, is of little consequence in its bearing on the mental disturbance engendered. The chief emphasis lies on the physical apparatus through which so widespread an affection of the whole organism can be evoked, just as in other circumstances the emphasis would lie on the psychical apparatus which serves the same end. This applies more widely than to emotional disturbances alone. Where a symptom is, on the face of it, definitely physical or definitely mental, its causation may not be inferred to be exclusively of the same order; the chief cause of, say, an anorexia may be a series of mental experiences or an attack of migraine or a uræmia or a pituitary disease. Study of the anorexia alone cannot serve to discriminate them; not even study of the psychological state alone, or of the physical state alone may suffice. Very often the physical and psychological factors in causation are mingled almost inextricably—they represent, in fact, different facets of the same series of phenomena.

Mental experiences.—Mental growth is dependent on daily experience for its material. Experience can be subdivided into perceptual, emotional, and other kinds, but such a division is fictitious. The means by which daily experience is incorporated with our mental equipment and acquires an influence over our subsequent behaviour in all respects can only be understood if we avoid thinking of emotions, instincts, perceptions and other abstractions as real entities, as distinct and separately operative forces. Memory, for example, is not merely an intellectual function by which we recall a happening into consciousness in more or less verbal terms, but a device, or function, by which past happenings are able to influence subsequent behaviour; the ways in which they do so, and the form in which the earlier experience is reproduced into consciousness, will be greatly influenced by its original emotional, as well as more purely perceptual, aspects, and by other physical and mental experiences—a distressing repetition of the experience, for example, or a physical happening like concussion or cortical atrophy.

There are general tendencies in mental life—instinctual tendencies—which bring us into relation with our immediate surroundings, direct us to feed ourselves, maintain our lives, reproduce, and aim at other ends, which are variously formulated by philosophers, saints and psychologists. These biological forces, however denominated or classified, are not peculiar to human beings, but in respect of human beings are so much more accessible to minute inquiry along verbal lines, that an unduly complex conceptual system has been built up to describe them. Comparative, behaviouristic and experimental

psychology have partly corrected unreal refinements of a verbal kind, as also can physiology and a truly medical psychology. Metaphysical subtleties and speculations, *e.g.* psycho-analytical ones, cannot meet medical needs, nor enable one to understand the human being as a whole organism in the way that the doctor must. The influence of previous experience on all subsequent behaviour is as evident in physiological happenings as in the mental field; the special language and formulations and hypotheses of psychology are not to be taken as wholly and permanently separate from those suitable to less highly integrated functions, though something must be conceded to the special complexity of psychological phenomena. Such terms as projection, sublimation, conversion, symbolism, identification, repression, amnesia, perseveration, displacement of affect, cover special instances of the general relationship between inherent tendencies of the organism and their material substrate, and the influence of past on present experience and behaviour.

In dealing with the multifarious world about him, a human being is constantly obliged to select what he will perceive, and in what form he will perceive it; pure "objective" perception never occurs. To perceive things at all, he must give them meaning by relating them to himself and to his previous experience. Unless he can do this, not necessarily consciously, he is at the mercy of his environment, as a new-born baby is. Perception is therefore an active process; it has instinctual and emotional, as well as cognitive aspects. It depends partly upon memory for being able to give meaning to what it perceives; such memory need not be conscious. Consciousness, it is well to bear in mind, is only an attribute of psychological happenings, not their essence or their criterion; mental life goes on with varying degrees of consciousness attaching to it. There is no sharp division between conscious and unconscious mental life: no region called "the Unconscious" with its own rules and contents. Many of the psychological happenings most significant for psychiatry go on without clear consciousness of them, but in appropriate conditions they may be accompanied by much more, or by full, consciousness. Biologically and psychologically regarded, consciousness is an attribute, like memory or movement, immensely important for us human beings, but not a "present-or-absent" factor decisive for our mode of mental conduct.

Perception being thus an active process, which makes use of past experience, it not only selects its material and invests it with meaning, but in doing so may distort it, and give it a special "false" meaning. Unwelcome emotions may be thus projected on to external objects or happenings, which are then regarded as hostile or contemptuous, or in some other way significantly related to oneself. This is not remote from the process in visual perception, whereby one projects the image on one's retina into the external world, and is convinced of its reality there; the further process of clothing it with emotional significance depends on one's inherent tendencies and one's previous experiences. Paranoid symptoms, ideas of reference, grandiose and self-reproachful delusions exemplify this. Hallucinations and kindred phenomena are a special instance of the interplay between material substrate (*e.g.* in cocaine poisoning), inherent tendencies (*e.g.* visual fantasies of children), and past experience (*e.g.* hallucinations of homosexual abuse or divine commands). Similarly, by fantasy and imagination the outer world can be manipulated or denied according to the heart's desire, just as by body-images

of proposed movement the way is prepared for purposive muscular action. In giving meaning to present things, personal connections between them and earlier experiences are established; whether normal or morbid, this ascription of "symbolic" meaning to every-day objects is indispensable to thought, and is most striking in our use of spoken or written language, where sounds and shapes are conventional symbols for the most diverse experiences. Some of our words are personal to ourselves, and are used in an individual way; in morbidly heightened form, this process may issue in schizophrenic neologisms, or oddities of expression. Similarly an obsessional patient may feel towards some word or object a superficially incomprehensible mixture of attraction and repulsion, which is due to this word or object being the symbol of some earlier experiences that have been of great moment in his life. To see how it has come to be such a symbol calls for minute study of his earlier experiences. Physical happenings in one's own body may symbolise present emotions or earlier experience of a momentous and emotionally painful kind. A gesture of disgust may normally be evoked unconsciously by a banal happening, which has somehow become emotionally coloured by past experience. A headache may embody our dissatisfaction with a present situation. So hysterical "conversion" symptoms may reflect and symbolise an inner emotional struggle, as may also some obsessional movement, schizophrenic stereotypy or hypochondriacal fear. The body, with all its functions, is the background of psychic life, and resonates to it.

What experiences will be important in determining the form of mental symptoms, depends much on the emotional disturbance they originally provoke, and this, in turn, on the instinctual drives which they touch on and disturb. Instincts may conflict, and the emotion accompanying the conflict prove so disturbing that it cannot be borne in its naked form; "repression" serves the end of making this more or less tolerable, through disguising or distributing it. So emotion may be shifted from one object to another, and paradoxical or unexpected emotions be thus aroused by objects on to which the affect has been displaced. Or energy mainly directed to plain ends, *e.g.* sexual love, may be diverted into less obvious channels, and when thus "sublimated" and mingled with features derived from other instinctual sources, its origins may be hard to recognise. Sexual instincts so often conflict with others that many of the most powerful motives for the production of mental symptoms come from the struggle.

To describe the whole of instinctual life, however, in terms of sex and aggression, as is sometimes done, is only possible if one strains the meaning of these words out of all knowledge. It is as unwise to make the sexual paramount in explaining psychogenesis as to burke it.

The patient's present symptoms, it is clear, must be examined in the light of his earlier experience. Thus one elucidates in detail the content of his illness and some of the causes of its occurrence. In doing so it is not necessary to push back one's inquiries to a supposedly crucial stage of early childhood. The experiences of the first two or three years of life are, like all subsequent experience, contributory to mental development, and they show certain sequences of phenomena characteristic of such development. Moreover, their relative simplicity makes it possible to recognise in these early reactions the instinctual drives, or (more correctly) the "inherited functions" which become manifest when the environment supplies the

necessary material, though, of course, it cannot supply the necessary energy and direction ; these last must come from within. On the other hand, the functions recognisable in the relatively simple reactions of early childhood are not the same as those which may be seen in later years when the organism is more fully grown, any more than an infant's physical structure and functions are identical with those of the more differentiated adult. The obvious continuity of the actual happenings in a human being's lifetime does not justify one in trying to analyse and reduce all adult mental phenomena into terms of supposed child psychology, nor does clinical practice usually require it.

The effect of war upon the incidence of mental illness has obvious importance. The psychiatric disorders which occur in war do not differ in kind from those of more normal times, but certain forms of disorder, especially panic, psychogenic stupor and gross hysteria in men, become commoner and sometimes more severe. People are exposed to unaccustomed dangers ; their privations are both material and emotional ; they have to surrender some of their independence and individuality ; and they are thrown together in groups and therefore prone to share in group-feelings and group-behaviour. It is doubtful whether the losses, fears and psychological infections of war directly lead to an increase in certifiable mental illness, though such exogenous factors as syphilis, alcoholism and malnutrition may do so, under the conditions of thoroughgoing modern war. It is, however, neurotic disorders that are most prominent then.

Course and Prognosis.—The making of a correct diagnosis may in psychiatry indicate the general drift of an illness—towards recovery, chronicity, progression, or relapse—but is of even less use than in the rest of medicine for showing how far this will apply to a particular patient. For this, careful study of the individual history and illness are indispensable. The prognosis can be inferred from the ætiological factors, the mode of development, and the form of the disorder.

Where a known external cause has been at work, its point of attack, its severity and persistence will affect the issue. This applies equally to such "organic causes" as poisons and cerebral diseases and to "mental causes," like economic misery or frustrated love. The physician must consider how long the environmental cause has been acting, what changes it is known to produce—whether in the way of cell-degeneration or habitual gloom, fibrosis or fantasy—and whether it is likely to persist. He must also ask if the patient's previous history has shown that he is specially sensitive to such a trauma. This brings in the intrinsic ætiological factors. How has the patient previously reacted to this sort of interference or to any disturbing circumstances ? Has he fallen more and more into unsatisfactory habits in meeting his daily life and its difficulties ? How has his whole character developed ? Is there good evidence of his being able to cope with partial deviations from mental health ? Has he inherited tendencies to benign or to progressive illness ? Which seem to be the most useful reparative or stabilising features in his personality ? How far are his struggles with the world an outcome of his intrinsic endowment, evident in various guises since his childhood, how far have they been forced upon him by an adverse milieu ? How old is he ? There is more chance, if he is young, of his being adaptable, so that the removal of various stresses

may help him, and his instinctual energies be diverted into less morbid channels; as he grows older, he may gain in stability, but gradually become more disposed to fear and suspicion, bodily preoccupations, and fixed attitudes of mind.

An abrupt onset is favourable, other things being equal. A gradual, especially an insidious, onset may indicate a rooted abnormality that will be hard to shift. The longer an illness has gone on, the more will it have become autonomous, *i.e.* independent of its immediate causes of occurrence, and prone to become a gross or text-book example of some chronic anomaly. A study of the ups and downs in the course of an illness may show favourable influences that can with profit be deliberately brought to bear on it, as well as harmful ones that must be avoided. The more reconciled the patient has become to his illness the less satisfactory the outcome.

As to the form of the illness and its prognostic value, there is much empirical knowledge at our disposal. Thus, a predominantly affective attack will very likely clear up, but may recur; a schizophrenic syndrome is in the long run usually ominous; hypochondria and depersonalisation, especially in young people, tend to last a long time, even years; sexual perversities can seldom be got rid of; hysterical symptoms can easily be changed, but hysterical reactions are persistent; obsessional attacks are either periodic or very chronic; melancholia is often a fatal disease, through suicide or refusal to eat; delirium tremens commonly ends by crisis or lysis after about seven days; untreated general paralysis of the insane goes downhill towards dementia and death, with partial remissions on the way: and so forth. There is a wealth of such special prognostic knowledge, based on clinical observation and statistics.

Obviously prognosis must always take account of treatment. Will treatment be efficacious? Will it be practicable? It is absurd to forecast how general paralysis will turn out if one does not know whether one will be giving artificial fever and tryparsamide, or how hysteria will turn out before one has decided whether psychological and social treatment will be possible. In every mental illness this is one of the essential points to be weighed in prognosis: what will be the conditions, beneficial, neutral and adverse, under which the patient is going to live henceforth; and, in particular, what will be those specially devised conditions of every kind, social, psychological and physical, which can be regarded as likely to have therapeutic effect?

Treatment.—**PROPHYLACTIC.**—Much can be accomplished by social measures; also by individual care, though that is less certain. A striking instance of social influence in preventing mental disorder may be seen in alcoholic psychoses, which have been cut down in this country to a third of what they were before the War of 1914–1918. Morphine and cocaine addiction and lead encephalopathy are now rare, typhoid delirium is exceptional, and typhus unknown. It is the organic mental disorders that have been more accessible to these preventive methods so far, because they have one indispensable cause that can be controlled. The “functional” disorders are partly dependent, it is true, on social factors, such as economic security, lack of employment, imposed moral, cultural and educational standards, competition and ill-judged interference. But the remedy for these is to seek, and their total removal is utopian. There is, nevertheless, much

room for prevention here. A preventable social cause may be well seen in "compensation neurosis" where the administration of a humane statute involving lawyers, insurance companies, doctors, employers and employees often has the inhumane effect of evoking hysterical symptoms, anxiety and a depressive or paranoid invalidism in the injured man.

Individual preventive measures cover both the intrinsic and the extrinsic causes. Eugenic precautions, such as birth-control or voluntary sterilisation (if legalised), may under skilled guidance prevent some mentally unstable persons from being born to parents who, having had mental illness themselves, do not wish to propagate it. If physical factors, *e.g.* diabetes, be prominent in causation, it may be possible to prevent this mental illness, or at any rate to scotch it in its early beginnings, by dealing with the somatic disorder. Thus, there are far fewer cases of syphilitic psychoses now that syphilis is less often contracted and earlier treated. The psychological reactions to a physical disease or blemish may be favourably modified or averted, when foreseen. It is for obvious reasons impossible to counteract mental disorder by regularly protecting the patient from physical or psychic trauma: a life that is guarded against risks and painful experiences is almost certain to issue in mental ill-health, out of its very emptiness. By altering a patient's environment and way of living one may, however, be able to avert an impending illness: only study of the individual patient can show how this end may be achieved.

The difficulties in the way of making the patient's environment easier for him are immensely greater during war, and it may be inadvisable to attempt any such change. Social needs have to come before individual ones in so many instances during war that the measures which would appear most favourable to the patient's mental health are often quite impracticable. Much, however, can be done by careful selection to lessen maladaptation among soldiers and other large groups. The mental hygiene of war is a complicated problem which cannot be divorced from the political and economic issues, as well as the military ones.

How far the treatment of behaviour disorders and neurotic traits in childhood can be trusted to avert outbreaks of definite mental illness in later life is a disputable matter, but it is certain that by taking advantage of his plasticity and responsiveness, a bent can often be given to the energies of the mal-adjusted child, which will result in his being socially better adapted and better able to deal with his problems. The more persistent the beneficial influences one can bring to bear on development at this impressionable age, the more valuable the prophylactic effort.

Measures of mental hygiene that may be recommended to the community as a whole are still of a negative kind: what to avoid rather than what to do. This applies most obviously in the field of sexual practice and belief where needless fears and harmful education are rife, as with regard to the masturbation of adolescence—a normal and comparatively harmless phase of sexual development.

TREATMENT OF THE ACTUAL ILLNESS.—This is almost as varied as ætiology and symptomatology. To use only one method of treatment, however simple or complicated its theory, is to fight illness with one hand behind one's back. There is no valid distinction between palliative and curative therapy: the distinction should be between more efficacious and

less efficacious. The nearest approach to a successful causal therapy is attained with those mental disorders which are closely related in time and form of occurrence to some indispensable cause, *e.g.* a toxic delirium, a reactive depression or anxiety, an interstitial syphilis of the brain. But these are rare conditions if one considers the whole of mental illness. The treatment of general paralysis by fever is not causal, its theory is dubious, its basis quite empirical; yet its success is such that it is the most important therapeutic advance in psychiatry for a hundred years. One cannot despise any measure that promotes the recovery or well-being of the patient: the giving of drugs, the prevention of suicide, occupational therapy, analysis of motives, removal into favourable surroundings, hypnosis, re-education and other means of helping the patient are not to be graded in a hierarchy with an arbitrary scale of values, in which recovery is called spontaneous unless psychotherapy or a novel chemical treatment has been employed.

Sometimes a patient's condition demands energetic intervention; sometimes it demands restrained symptomatic treatment; sometimes social adjustment is called for; sometimes endocrine injections. Whether the accent in treatment shall fall on the physical or the psychological or the social side will often be less important than care that all the available resources are used. It should not be regarded as a matter of course that a diagnosis should connote a method of treatment: *e.g.* that psychoanalysis is the only thorough treatment for obsessions, while for mania continuous narcosis is the "proper" method. Nor, to mention another common error, should it be lightly assumed that a heavily tainted family history or other evidence of a strong constitutional factor indicates that treatment is out of the question, a superfluous struggle against fate.

Treatment may be considered as social, psychological and physical. For some types of illness obviously much more stress will fall on one than on another of these, *e.g.* in hysteria, general paralysis of the insane, epilepsy.

Social and occupational treatment.—The first task in social treatment is to decide where the patient is to be looked after. Is he fit to be at home, should he be in a mental hospital, or in some environment intermediate between these extremes? The decision as to the need of a mental hospital rests in the first instance on the danger the patient presents to others, or the chance of his committing suicide. These two problems of behaviour were at one time almost the only grounds of admission to a mental hospital, but such questions of "certifiability" need no longer preoccupy the psychiatrist, since voluntary treatment has broadened the scope of the mental hospital and modern conditions made it suitable for many patients who would ordinarily be regarded as "neurotic," rather than "mental" "psychotic" or "insane" (*e.g.* early cases of general paralysis masquerading as neurasthenia, or obsessionals who fear their own impulses and want to be protected against themselves). Psychiatric hospitals and clinics dealing only with voluntary cases also bridge the gap between out-patient care and certification.

The social decisions in treatment cover much more than merely the mental hospital issue. If the patient's immediate environment contains many disturbing influences, it will be desirable for him to be away from them temporarily at least, so long as this does not entail worse troubles; summary decisions are here impossible. It may be useless, for example, to get a woman who is

paranoid about her neighbours to move to another district to escape them, unless it is the actual conduct of the neighbours and not the patient's morbid attitude that is provoking her suspicion of them. It requires a close knowledge of the facts as well as wisdom and psychiatric experience to give advice on matters that may wholly alter the course of a patient's life—advice, say, about separating from his wife, giving up his job, or emigrating to the Dominions. Many instances of this might be offered. Neurotic patients are often advised to get married, especially if loneliness and sexual needs trouble them, as though marriage were a panacea; such advice by rule of thumb too often makes their condition worse, ruins the life of the person they marry, and results in offspring that have to be treated at a child guidance clinic. Weary, depressed patients are often harmfully urged to go to dances and lively seaside resorts where they must try to look happy. Hysterical patients do not benefit by being put among people who are hostile and contemptuous, any more than in an atmosphere of mawkish sympathy and compliance.

In the social treatment of patients indispensable help can be given by trained psychiatric social workers. Their assistance is not restricted to the patient's economic problems, though it is most obvious in that field.

Occupational treatment is important for all kinds of mental disorder. Where there is acute overt emotional disturbance, rest is at first desirable, as also for confusional and delirious states. In these conditions opportunity for occupation must be gradually offered to the patient as his disorder subsides; steady, simple work is preferable to the restless unsatisfying fickle activity in which he would often engage, if left to himself. The less acute any mental disturbance, the more necessary is it that occupation should be urged upon the patient, and that it should be disciplined and congenial. This applies equally to gross psychoses and minor affections of the neurotic sort. Allowance must be made for the patient's bent, his symptoms and personality, and especially his more or less conscious reasons for working and not working; hence there will be much diversity in the conditions of his occupation, whether it be therapeutically contrived in a hospital, offered at a Rehabilitation Centre, or sought out as remunerative work in the open market. Mental health cannot be permanently retained unless one does some satisfying work; often it cannot be recovered unless one does. Work is not satisfying, in the long run, if it is done mainly as a diversion, to fill in time.

Psychological treatment.—There is no form of treatment which has not a psychological aspect and result. The term psychological treatment or its synonym "psychotherapy" is, however, conventionally limited to those forms which depend upon direct and personal relationship between the patient and the physician. They have separate names, and are divided into schools and techniques. Stress may be laid upon the prestige of the physician (as in hypnosis), the patient's attachment to him, in all its complicated phases ("transference"), the trained understanding and thoroughness with which he clears up the patient's problems (persuasion, re-education, distributive analysis), or on his qualities of personality—enthusiasm, energy, warmth, candour, wisdom. In so far as psychological treatment is necessarily based on a personal relationship it cannot be made a routine except in its non-essentials: whatever rules the psychiatrist follow or whatever the training he has undergone, he himself will be more important than his method in

benefiting the patient. To that great extent psychotherapy is not a scientific procedure. That is not to say that method and training are of no consequence—far from it—but only that they are devices whereby the influence of one human being upon another's mind and conduct can be turned to the best medical ends, and the dangers inherent in such a relationship minimised.

The more specialised, intricate or esoteric the method, the less suitable is it to be used by any but the most expert. It is not proposed here to detail the many kinds of technique that have been employed. The general rules that must be followed in any psychotherapy are :

1. To regard the removal of symptoms as a good thing, but the maintenance of normal social adaptation as far better. It is bad to get rid of one symptom only to see it replaced by another, but much worse to get rid of all symptoms only to see the patient at the end of treatment a dependent and introspective hypochondriac of the mind, a social invalid.

2. To seek for the psychological cause of the patient's illness only to the extent that the patient's wellbeing demands, which is often far short of what one's own interest and psychological curiosity would demand.

3. To consider carefully whether any shock to the patient, any aggravation one produces in his illness even temporarily, may be a sign of bad treatment. It is on the whole unnecessary to make the patient worse in order to get him better, though many psychotherapists believe the contrary.

4. To be satisfied with the patient's recovery, and not to aim at his promotion to a state of ideal mental health and self-understanding. It is better that treatment should be quick and effective than drawn out to meet theoretical standards.

5. To understand the development of the patient's illness, and to interpret it both to him and to oneself, in terms of real experience and not of hypothetical forces.

6. To treat the patient without allowing one's own emotions to be more concerned in the course and outcome of the treatment than is usual in the treatment of a physical illness.

7. To aim at harmonising the patient's mental life by giving his ill-managed energies fitter material to work at, and release from the burdens laid on them by past experience.

It is impossible to describe in general terms what the psychotherapist does, otherwise than by metaphor or analogy : he promotes the ventilation and desensitisation of emotional disturbances ; he elucidates latent or obvious muddles, disentangles conflicting tendencies, giving them new incentives and a different direction ; and so guides the patient through the maze of his life's experience, as recalled in memory, that he is afterwards better fitted for dealing with current experience, knows himself better and has somewhat purged himself of past harms. All "analytic" methods review the patient's life as he recalls it under special conditions, *e.g.* of free association, hypnosis, biographical scheme, etc. They stop at different points, some aiming at emotional clearance by abreaction, some at a redirection and liberation of the instinctual bases of character, while others remain content with an educational achievement.

Whether psychotherapy, in the above sense, is to be applied to a case will depend on the following factors : the patient should be willing to co-operate in the treatment ; free from such hindering disabilities as, say,

deafness ; able to give the necessary time ; of at any rate average intelligence ; still capable of modification (as he would not be in old age, or with very long-standing and indurated habits of faulty reaction, or with organic cerebral disease) ; and, finally, endowed with a considerable residue of normal mental functions with which one may work. The more profound his aberrations, as in schizophrenia, or the more extreme his emotional disturbance, as in agitated melancholia, the less is he fit for psychological treatment of this individual and specialised kind. Psychological treatment, however, in the literal and larger sense of the words, is essential for every variety or stage of mental illness, and every degree of co-operativeness or intelligence. It is a wide notion, including all that may ease or reassure the patient, bring him to a better relationship to those around him and with himself, and protect him from being distressed by the ignorance, lack of tact, or thoughtlessness of others. It is as much negative as positive. One must avoid arguing with the patient, telling him lies "for his own good" or to avoid unpleasant scenes, cajoling him, making promises that will not be kept, threatening or punishing him, jesting at his expense, losing one's patience with him, assuming he is indifferent to what goes on because he looks indifferent, provoking him by petty supervision or frequent rebukes ; one should not assume that he is quite irresponsible or quite responsible, nor talk theory to him, nor get on a false footing through ready assent to his delusions and his point of view. The physician and the rest of those who are in contact with the patient must do certain positive things : make due allowance for his disorder influencing his conduct, use their understanding of the psychological happenings without saying so, take advantage of every opportunity created by other methods of treatment. When occupation, narcosis, hydrotherapy, massage, a physical illness or other happenings bring him more closely into contact with nurses and physicians there are chances of unobtrusive psychological treatment in the wide sense.

Physical treatment.—"Mechanical restraint" and violence are now foreign to the treatment of insanity ; the patient may be unrestrained and violent, but his treatment may not. It is still necessary, however, to restrain a patient who is bent on harming himself or others, and physical force may be the only way of doing so, or of giving a patient by tube enough food to keep him alive when he abjures the natural way of eating. But force must always be a last resort ; and chemical substitutes for it seem only a little less of an evil. Drugs have their place in the treatment of all kinds of mental disorder, but their use easily turns to abuse. Whether one is giving morphine and hyoscine in an emergency to an acutely excited catatonic, or prescribing aspirin for a mild hysteric, the chief danger must be borne in mind, which is not overdosage, habituation, or suicidal misuse, but the habit of stupefying or satisfying a patient with drugs when other means might be taken, better suited to his condition. Sedative drugs should not be a short cut ; neither should they be eschewed. They should be given when other measures will not serve, as for some obstinate form of insomnia, anxiety, agitation and restlessness, or when their use obviates greater troubles, *e.g.* the pulling of bandages from an operation wound. The symptoms of intoxication must be watched for with more than usual vigilance when bromide is being given, because if unrecognised as such they may lead to certification—for an avoidable drug-made psychosis. Continuous narcosis for nearly a fortnight,

with the patient sleeping through 18 or more of the 24 hours, is sometimes efficacious in abbreviating an acute attack of mental illness; it is a dangerous method except in skilled hands.

There are other drugs to which the above cautions scarcely apply, *e.g.* endocrine preparations, remedies specific and otherwise for the physical basis of "organic psychoses" (*e.g.* arsenical treatment for syphilis of the central nervous system), and aperients. Insulin for promoting hunger, calcium for those with hysterical hyperventilation fits, amphetamine (benzedrine) for anxiety, and a number of substances—from nitrous oxide to amytal—that relieve catatonic stupor, or facilitate psychological inquiry and treatment, have all been found useful on occasion, though their field of application is small.

Two methods of treatment have been widely employed of late years, especially in the treatment of schizophrenia. Insulin has been employed in large doses to produce hypoglycæmic coma repeatedly; and certain convulsant drugs (pentamethylenetetrazol and cyclohexylethyltriazol) given to produce fits. The benefits to be obtained by these methods are not yet clear. Undoubtedly in some cases the duration of a schizophrenic illness can be shortened by the insulin treatment, and recovery brought about in cases regarded as having a bad prognosis. It is, however, in those that would have a favourable outlook by other methods of treatment that the majority of insulin successes are obtained. The technique demands skill and experience, if considerable risks are to be avoided. It consists essentially in daily administration of the appropriate dose of insulin to produce coma. The duration of the treatment is usually not more than two months. Treatment by the artificial production of convulsions was first instituted for schizophrenia, but the results have not proved as satisfactory as was at first supposed, and it is of very limited application. The method has proved effective in terminating obstinate melancholia, chiefly involuntal, and in abbreviating attacks of depression which would otherwise have taken months to clear up. The chief objections to the method are the disagreeable experiences many patients have immediately after the injection, and the likelihood of cerebral impairment, and of fracture of the long bones or dorsal spine during the fit. The last complication is not infrequent, and may occur also when the convulsions are induced, not by a drug, but by electrical stimulation of the brain, a method employed within the last two years.

Exercise or massage and hydrotherapy are beneficial as much for their psychological as for their physiological results; the latter, however, are not negligible, as may be seen in the effect on an excited or an anxious patient of a continuous bath at body temperature. The chief importance of diet lies in the frequent refusal of food by patients depressive, hysterical, stuporose, paranoid, hypochondriacal, or over-active. Feeding by the nasal or oesophageal tube is a necessity in many such instances, after every other method has failed. Rarely, special diet is called for, as in epilepsy, the symptomatic psychoses of diabetes, pernicious anæmia or pellagra, and also for some temporary disabilities of the alimentary tract—*anorexia nervosa*, psychogenic hyperchlorhydria. As a rule, however, such dietetic regime, and indeed all physical treatment of localised psychogenic disturbances of function in a bodily system, is an expedient rather than a settled and adequate mode of treatment. Many patients with a visceral neurosis, a hypochondriacal

preoccupation, a hysterical anomaly, or a somatic delusion are greatly harmed by the prolonged physical investigation and treatment they receive : it confirms the symptom, localises it all the more, and brings fresh ones in its train. Sometimes one has no choice ; a progressive hysterical contracture, a dermatitis artefacta, a sore infected by constant picking, a tooth loosened by obssional knocking at it demand treatment.

The caveat against lightly resorting to physical treatment of psychogenic anomalies is especially applicable to operative surgery. In general there is no more reason for "cleaning up the septic foci" in a person with mental disorder than if he were a mentally healthy person—other things being equal. Removal of teeth, appendix or tonsils, scraping of sinuses, and searchings of the pelvis are operations that in psychological medicine seldom yield the results expected of them.

CLASSIFICATION

The ideal classification would be on a uniform basis, according to the nature of disordered physical and psychological function, or according to innate and external causes. Since we do not know enough to do this, a mixed ætiological, functional and clinical grouping is used, whereby the same illness can belong in several categories. It is obviously provisional. The chief division is between those mental changes accompanying distinctive somatic disorder and those for which no such physical relationship has been demonstrated. The former are called symptomatic, or organic ; the latter constitutional or functional. It is needless to illustrate the point that everything found in the latter may be seen also in the former. The reverse of this is not true, because there are some symptoms—due to the loss or damage of essential tissues, especially in the central nervous system—which can only occur when the material substrate is grossly damaged.

Although the "functional" group is made up of those conditions for which no distinctive somatic disorder can be found responsible, it by no means follows that their causes or basis are therefore purely psychological. Theoretically, such a belief is untenable ; and as a matter of observation certain physical disturbances so regularly accompany these disorders and a physical configuration may be so linked with them that there is small doubt that eventually the somatic disturbance of function in them will be well enough worked out for the terms "organic" and "functional" to lapse, and only the crudity of the physiological changes remain as a point of difference.

As mental disorder thus comes closer to general medicine so must the whole of general medicine reveal its psychiatric side, which is now as little illumined as the physiological side of the psychoses.

The first or toxic-organic group is large, the chief syndromes in it being neurasthenia, confusion, and delirium and dementia. Such phenomena as apraxia, aphasia, agnosia, amnesia and hallucinosis are fairly frequent in this group.

The second group, comprising three-fourths of the recognisable mental illnesses, includes the insanities or psychoses, and those anomalies, outwardly less alien to the normal mind, commonly called "neuroses." The distinction

between neuroses and psychoses is at times convenient, but without substance. To argue whether a dubious case is neurotic or psychotic is like arguing whether a man of medium size is thin or fat : he is both and neither. A genuine decision as to ætiology, prognosis or treatment turns not on whether a case is regarded as neurotic or psychotic, but on more solid findings. Since such words die hard, the best use of them is to term a patient with mental disorder "neurotic" if he has insight into his illness, is co-operative and unlikely to need care in an institution, and to term him "psychotic" if the contrary is the case.

The toxic-organic group is divided into diseases located in the nervous system and those affecting it indirectly, as uræmia or lead poisoning may. Some are toxic, *e.g.* delirium tremens; some degenerative (senile psychoses); some inflammatory, *e.g.* encephalitis lethargica; some plainly hereditary, *e.g.* Huntington's chorea or "primary" mental defect; and some privative, *e.g.* pellagra or myxœdema.

The "functional" conditions are arranged according to whether emotional disturbance is evident and predominant (affective disorder), or whether there is profound derangement of thought, feeling and contact with the real world (schizophrenia), morbid false beliefs have become fixed without intellectual or emotional deterioration (paranoia), repetitive and seemingly irrelevant phenomena hamper mental activity (obsessional), signs of physical or mental ill-health, especially dissociation, readily appear when an unpleasant situation may thereby be escaped from (hysteria).

As will be seen in the special sections, the personality of the patient may also be a criterion of these groupings, with the proviso mentioned earlier that illness does not only occur in those with the appropriate psychopathic anomaly of personality, nor does the latter by any means regularly issue in definite symptoms. Unless, however, psychiatry takes account of the psychopathic personality, even when not accompanied by symptoms of illness, it cannot study delinquency, disorders of behaviour in children, sexual perversions and other not obviously medical anomalies which touch very closely on psychiatric problems in the stricter sense, but are omitted here for reasons of space.

The following is the classification used here :

1. Organic Disorders :

(a) Degenerative and Hereditary Brain Disease.

(Senile dementia, cerebral arterial disease and hypertension, Huntington's chorea.)

(b) Syphilis of Central Nervous System.

(c) Other Cerebral Diseases.

(Lethargic encephalitis, Sydenham's chorea, disseminated sclerosis, cerebral tumour, cerebral trauma, epilepsy, etc.)

(d) Intoxications.

(Alcohol, morphine, cocaine, bromide, etc.)

(e) Infections and Exhaustive Disorders.

(Infectious toxæmias, hæmorrhage, etc.)

- (f) Metabolic, Endocrine and Visceral Disorders.
(Diabetes, pernicious anæmia, pellagra, exophthalmic goitre, myxœdema, tetany, pituitary diseases, sexual epochs, cardiac disease, uræmia, etc.)
- (g) Mental Deficiency.
- 2. Affective Disorder.
 - (a) Excitement.
 - (b) Depression.
 - (c) Anxiety.
- 3. Schizophrenia.
- 4. Paranoia.
- 5. Hysteria.
- 6. Obsessional Disorder.

In the above classification mental deficiency is given a separate heading, though properly it should be systematically distributed among the preceding groups, since it differs from the rest of mental disorder only in the age at which the damage is done. Custom and convenience compel the old distinction to continue.

The above are great clinical groupings, types of morbid reaction, which are the nearest to a valid and useful classification we can get at present. There are subordinate symptom-complexes or syndromes, which are likewise innate and preformed, and likewise evoked by circumstances, but which are not limited to any one of the major groupings—they are the web that runs across the psychiatric pattern. The most important of these are depersonalisation, hypochondria, twilight states, stupor and other disorders of motility, and spasmodic attacks and seizures of different kinds. Between symptoms (classified on a psychopathological basis) and the main groupings which best serve clinical purposes, these symptom-complexes have an intermediate place, comparable say, to that of mononuclear leucocytosis or coma in general medicine.

All the categories of psychiatry stand for mixtures of symptoms due to disturbance of control, capacity or co-ordination and synthesis in mental life. Nor is this true only of mental life, since the same symptoms may arise through physical disturbances of function which may be classified in the same way; this may be plainly seen in such a disease as encephalitis lethargica.

ORGANIC DISORDERS

A. GENERAL DESCRIPTION OF TYPES

The varieties of form and course in organic psychosis are essentially few and simple, in contrast to the causes, which are numerous. In other words, there is no support for the hope that to each physical disease there corresponds a characteristic mental disorder. It is not possible in an organic psychosis by study of the mental picture alone to infer its physical cause; for that the methods of somatic medicine are needed. Many different poisons and lesions

may produce the same effect on the mental state. Differences depend on the degree and duration of the physical damage and its site, which may determine neurological and other symptoms of a typical kind; *e.g.* in G.P.I. or encephalitis lethargica.

They are the least constitutional of all mental affections, yet even in them constitutional factors are far from negligible. To such factors it is due that one man will show a psychosis with physical illness that in another would lead to no such mental upset, and that one patient responds with a manic extravagance to the cerebral disease that makes another patient depressed. Moreover, hereditary factors can be of great importance in these organic affections, as may be seen in amaurotic idiocy or Huntington's chorea.

The few syndromes commonly met with here, though they are not restricted to organic disease, must be described before seeing how particular diseases colour them and determine their course and treatment. In the organic syndromes, a diminution in mental capacity is the central finding. Nearly all of them may occur in cases and types of illness in which no structural damage can be found, as might be expected seeing that the available patterns of structure and function are in all cases much the same. Delirium or confusion may occur typically in non-organic syndromes, though much less frequently than in organic ones.

(1.) NEURASTHENIA.—This term has been over-used and ill-used, like most of the more palatable diagnoses (*cf.* anxiety neurosis), but it need not therefore be discarded now. It denotes a form of irritable, hypersensitive weakness and depression that is not uncommon after infections, exhausting experiences (*e.g.* hunger, lactation, insomnia, worry, hemorrhage), cranial injuries and chronic poisoning (*e.g.* with alcohol or coffee). It is true that a clinical picture indistinguishable from it frequently arises where physical causes are unlikely and emotional causes are obvious: this clinical finding has the same significance as the fact that the anxiety of exophthalmic goitre is like psychogenic anxiety. Just as the anxiety of exophthalmic goitre or constant fear can pass into delirium, so can physiogenic neurasthenia be aggravated until it becomes plain dementia.

The symptoms are partly somatic—active deep reflexes, increased sensory irritability, feelings of pressure on the head and pains in the muscles and elsewhere, giddiness, vasomotor lability, delayed peristalsis and feelings of fullness in the abdomen, diminished libido, slight clumsiness, and tremor of the muscles of the face, tongue and hands. On the more psychological side, there are feelings of languor, and incapacity to concentrate on any mental work, doubts as to the accuracy of memory, loss of interest, slight depersonalisation, irritability and tenseness, lessened control of emotion, and perhaps slight paranoid, obsessional or hypochondriac trends. This general condition is, when physiogenic, less influenced by a change in mood than would be the case with psychogenic neurasthenia, and the patient is better able to control his motor unrest than his features, which are expressive of his agitation. The chief reliance, however, must be put on the history and physical findings for telling whether the neurasthenia is physiogenic or not; psychological causes which seem adequate to explain the illness may be deceptive.

The course of neurasthenia is towards recovery unless the noxa continues to act; where the noxa persists, extreme chronicity can result. Sometimes

an original physical noxa ceases to act, but meanwhile other emotional ones have entered the field, *e.g.* unemployment, domestic fears and frustrations, and so the illness drags on. Treatment depends on assessment of the causes and the possibility of removing them.

(2) DELIRIUM.—Delirium, most familiar in fevers, can also be produced by drugs and other causes of acute cerebral disturbance: severe affective disturbance also may be accompanied by delirium. Its characteristics are general malaise, restlessness, irritability and sensitiveness to external stimuli, headache, anxiety and troubled sleep, or insomnia. Mild forms of this are met with in so transient an affection as cold in the head. Severe forms are marked by illusions and hallucinations of all the special senses, especially vision. Anxiety often becomes extreme, and the patient is terrified of his fantastic visions. Thought becomes as chaotic and fleeting as in dreams, activity is incessant and past experiences of daily life are revived, as in the occupational delirium of alcoholics. Attention is weakened, and orientation in time and space much impaired. There are striking variations in the severity of the condition in the same patient: it becomes worse in the evening or when the patient has hardly any external stimuli to keep him in touch (cf. delirium at night and after a cataract operation). The extent to which consciousness is clouded usually corresponds to the amount of perceptual and affective disturbance. Auditory hallucinations occur with clearer consciousness, visual ones very profusely with a clouded mind. The auditory hallucinations are commonly of an elementary, undifferentiated kind—not voices. Vestibular hallucinations may occur, *e.g.* of floating in the air. Distressing and incoherent ideas pursue each other—ideas of being torn to pieces, burnt, poisoned, buried alive, and so on; also ideas of grandeur.

Closely akin to delirium, and indeed shading into it, is the *confusional state*, in which thought is very incoherent, but the patient is more eager to get in touch with his environment than in typical delirium. If consciousness is not grossly clouded, the patient is perplexed and troubled by the disordered perceptions through which alone he can learn what is going on about him. The picture may be indistinguishable from that seen in some forms of manic excitement and in some catatonic states. Differentiation rests, not on the immediate psychiatric symptoms, but on the history and discoverable causes of the illness. The same is true of *acute hallucinosis* in which orientation and grasp are very little impaired, but auditory hallucinations—especially threatening sounds and voices—abound, and there is a tendency to the formation of delusions on the basis of these and other perceptual disturbances. The name "*twilight state*" is applied to another syndrome in which consciousness is changed chiefly because of some powerful affective influence; anger or fear may so overwhelm psychic life that the patient cannot grasp his surroundings, his thinking is interrupted and slow (except where it falls in tune with the affective disturbance), and his motor behaviour is in keeping with his mood. It is as often of psychogenic as of organic origin—one can hardly, for example, by direct observation tell an epileptic twilight state from a hysterical one. Like delirium and the other conditions just mentioned, it is prone to subside and to be followed by amnesia for what happened during it: where there is some recollection, it may be associated with a conviction that the hallucinations and other morbid phenomena were real external happenings.

(3) DEMENTIA.—Of all gross encephalopathic syndromes this is the gravest and most typical. It corresponds to a diffuse cerebral disease, and is made up of intellectual impairment and lessened control of emotion. Its form depends so much on the stage of the patient's development at which it occurs, that it is customary to consider as dementia only those cases in which the cerebral damage has occurred in later childhood, adolescence, or adult life, and to regard earlier cases, *e.g.* cretins as showing mental deficiency or arrest of development. The distinction is rather artificial, at whatever age it be made. For convenience, only the adult form will be described here. The order in which functions are impaired corresponds to Hughlings Jackson's principle of dissolution: thus, recently acquired memories are soonest lost. There is intellectual weakness—the patient cannot reason, grasp and remember as he could, his attention is less concentrated and sharp, his ideas are fewer, he cannot take in anything complicated or be sure about time and place, he loses himself. His emotions are likewise affected—he weeps over trifles in spite of efforts to control himself, his feelings are shallow and transient, he may be foolishly euphoric, or may burst into anger whenever he cannot get his own way. There are wide variations in the severity of the condition, and its symptoms may be much influenced by the local incidence of the pathological changes in the brain. The extent to which various cerebral functions are impaired may differ widely in the same patient: a man who seems hopelessly demented may be able to play a good game of chess, while another in whom it is hard to demonstrate any intellectual impairment may micturate into his shoes or do something equally stupid and inappropriate; unexpected sexual misdemeanours are not uncommon in demented persons who do not as yet show gross intellectual damage.

Closely connected with dementia are the *amnesic* syndromes, known by the name of Korsakoff. Here the memory disturbance is in the forefront. The incapacity to receive, store and reproduce experience is remedied, as it were, by lying, *i.e.* the patient confabulates to fill up the gaps in his memory. These patients are often ready to adopt suggestions, so that one can lead them to tell absurd tales about their recent movements, *e.g.* that they were yesterday at Vladivostok to see some polar bears. They do not show an intellectual damage or incapacity to deal with ideas that is at all comparable in degree to their memory disorder, but they are always out in their appreciation of time-relationships, especially where the present is concerned. At first blush they often seem to be behaving like mentally healthy people, but one presently discovers that their memory is much impaired, their orientation as to space, time and personal identity correspondingly poor, and their interest and general mood duller than is normal. The disorder of memory is never, as in dementia, a general weakness reaching back even to childhood.

The Korsakoff syndrome is most often seen in alcoholics, in whom it was first described associated with polyneuritis, but it also occurs in a great variety of organic disorders, *e.g.* intoxication with lead, carbon monoxide, and other poisons, uræmia, cranial trauma, cerebral syphilis, and arteriosclerosis—apoplexy may precede it and the amnesic syndrome be thus complicated by aphasia. That it should sometimes follow on delirium is not surprising, since in delirium the same memory disturbance is present,

but covered up by the concomitant excitement, disturbance of consciousness, and hallucinations. Whether a Korsakoff syndrome will clear up depends on the cerebral damage which produces it; the alcoholic form occasionally does so eventually in uncomplicated and treated cases.

Mental deficiency is a special instance of cerebral impairment, as is dementia. It is considered, for the sake of convenience and tradition, in a separate section. (See p. 1846.)

B. DEGENERATIVE AND HEREDITARY BRAIN DISEASE

There is a group of disorders occurring in late middle life and old age, which are clinically and even pathologically near to one another. At the one end of the scale is senile dementia, at the other climacteric anxiety and depression. It includes Pick's presenile dementia, Alzheimer's disease, cerebral arterial disease, and arterial hypertension.

1. SENILE AND PRESENILE DEMENTIA

Ætiology.—Constitutional factors are obviously the most important. A tendency to become dotards may be evident in successive generations of a family; heredity is held responsible for the wide differences in mental health among elderly people. The symptoms of senile psychosis may not be revealed until the patient is exposed to some sudden stress—the death of his wife, the need to move house, the loss of his occupation, some new set of circumstances. Senile psychoses are more common in people with lifelong nervous symptoms.

Pathology.—**PATHOLOGICAL.**—The tissues show the general signs of age, *i.e.* a diffuse atrophy, which makes the convolutions narrower and the weight of the brain less. The nerve cells and fibres are fewer, while the mesodermal and neuroglial tissues ^{are} increased; fatty pigment accumulates. There are also, however, in senile dementia striking histological features in the grey matter, especially in the cortex, namely, thickening of the neurofibrils, which are characteristically twisted and aggregated, and there are remarkable plaques, seldom seen except in this condition. There is no close correspondence between the kind or extent of the tissue changes and the mental state. Plaques and neurofibrils are reported to occur also occasionally in the brains of mentally healthy old people.

PSYCHOLOGICAL.—The previous tendencies of the patients may greatly colour the symptoms. Obscure somatic preoccupations and disturbances in time appreciation lead often to fantastic delusions about eternity and what is happening in their body.

Symptoms.—Memory is poor for recent events; the extent of the damage may increase until only the recollections of childhood and early adult life remain. People and places are falsely identified with those once familiar, and transient pseudo-memories are invented. Events with a strong affective tone, especially if unpleasant, are remembered better. The memory of the remote past is not entirely spared; even matters of personal identity may at last be forgotten. Grasp and judgment, the capacity to follow a train of thought and to eliminate the irrelevant are faulty. Obstinacy and

perseveration go with a rigid adherence to old habits. Prolix and garrulous, the patient does not recognise how little interest there is for others in his repetitive and ill-arranged talk. He may partly cover its emptiness with long and sounding sentences; on the other hand, some patients become monosyllabic, because of their failure to find words to express themselves, and others again will use a word loosely associated with the one they are vainly seeking, or will quite seriously give a punning meaning to a word, and even act accordingly (*e.g.* whistling because "You said I could whistle for my money").

There is a narrow range of interests, in which food, possessions, and bodily well-being are prominent. Grotesque hypochondriacal delusions are common. Patients hoard rubbish and are angry if interfered with in this. On the whole, however, their affective responses are greatly reduced; they meet calamities with composure, partly due to their failure to grasp what has happened. Now and then they show depression and resentment at a slight, and may bear a grudge long after. Their activities are sometimes considerable, on the lines of determined rummaging and collecting; in others a dull inactivity is all. They become dirty and unable to look after themselves. This applies as much to those who are excited and active as to the inert. The former may fight against being fed and washed, and it is not possible to get them to understand what is being done. Delirium and confusional states are prone to occur at night, accompanied by fear and bewilderment. Sleep is bad, and often the patients busy themselves about the place all night long.

Legal difficulties arise through the heightened readiness to accept some suggestions (as in the matter of making a will, or giving away property), the poorer judgment and the lessened capacity to control sexual desire, which is sometimes seen in the early stages. Hoarding may lead to petty thieving. Occasionally the patient sets fire to the house during his nocturnal prowls.

The symptoms need not be obvious. Often the illness has so slowly developed that no one can say when it first passed beyond what is normal in old age. An apparent change of character—a kindly man becoming selfish, a respectable churchwarden assaulting little girls sexually—may usher it in; this is not so much a change in character as a release of primitive trends, hitherto controlled. The psychosis may take various forms—depressive, manic, and paranoid. In the *depressive* variety there is seldom retardation, the affect is rather empty, the patient is irritable, and hysterical symptoms may be commingled with hypochondriacal ones. Ideas of poverty, wickedness and disease are often grotesque in their exaggeration—the patient's urine drowns the whole world, his body is an undying shell of corruption, he is as tiny as a baby—and are monotonously reiterated. The *manic* variety is rarer: pointless activity and a diarrhoea of words, with silly boasting, may be accompanied by a disturbance of memory, giving a total picture of the Korsakoff type: it is sometimes called "presbyophrenia." Many of these patients have always been of hypomanic temperament; their illness may be only slightly progressive and not so severe as to call for hospital care. The *paranoid* variety is especially likely to occur in people who have always been of a suspicious turn of mind. They hide things because they feel surrounded by thieves, and then forget where they have hidden them;

their failing senses, especially of hearing, feed their distrust, and they project their awareness of sexual impotence or waning intellect. Hallucinations and delusions are mingled—gases are pumped into their room, their food is poisoned, people throw bombs at the house by night, greedy heirs are doing them out of their possessions. Some of these patients barricade themselves against their enemies or call in the police. Whereas the depressive and manic forms are commoner in people with corresponding heredity, this paranoid form is genetically often connected with schizophrenia, though the distinction between the three varieties is not a sharp or important one. The name “*involutional paranoia*” has been given to the chronic delusional condition of this type that may develop in single women between the ages of 40 and 52.

Bodily symptoms are those of old age, especially in the central nervous system, where it leads to a slow, careful gait, with short steps and legs wide apart, apraxia and poor co-ordination, tremulous rather whining utterance, small sluggish pupils, and occasionally epileptic seizures. The disorder of movement is conspicuous in the handwriting—pointed, small or erratic in size, and sometimes jerky and tremulous.

The conditions known by the names of Pick and Alzheimer are to be regarded as atypical senile or presenile psychoses.

Pick's dementia consists pathologically of a circumscribed cerebral atrophy, mostly in the frontal or the temporal lobe, or in both; the motor area, however, is seldom affected, nor are Wernicke's zone and the transverse temporal convolutions; other areas of the brain may be involved. Histologically, the ganglion cells are swollen and contain argentophil globules. There is a hereditary determinant. The onset, which is gradual, can be at any age from 40 onwards, but is usually between 50 and 60. Symptoms depend on the localisation of the atrophy. Memory and affect are not impaired till late; they are preserved at a stage in which the patient behaves stupidly—stealing, lying, or otherwise making a fool of himself. Spontaneous attention is poor; at first moody, the patient becomes dull and unresponsive; judgment deteriorates and initiative fails. Stereotypies, echolalia, and repetition of empty phrases, monotonous talking and laughing or singing, and outbursts of bellowing or whining appear in the later stages. There may be aphasia. Diagnosis is difficult during life; it may be assisted by an encephalogram showing the shrinkage of cerebral tissue from atrophy.

In *Alzheimer's disease* the senile plaques and neurofibril changes are very numerous. The onset may be between 40 and 60. Indefinite premonitory symptoms (headache, irritability, forgetfulness) are quickly followed by progressive dementia; aphasia and apraxia are prominent, though less coarse and sudden than in cerebral arterio-sclerosis. In the earlier stages the patients are in fair contact with their environment, and look as though they grasp much more than they actually can. Their deficiencies are shown up in writing and talking. As the disease advances they are less open to affective influences: they sink into themselves and say little. Stereotyped words or syllables and movements take the place of embarrassed remarks and gestures. In the aphasia there is a rather characteristic stringing together of syllables like each other in sound, but meaningless. Muscular rigidity may lead to contractures. The progress of this disease to severe

dementia is faster than in typical senile deterioration and the onset is rather earlier.

Prognosis.—This depends on the previous rate of development of the condition, the general physical health of the patient and any special pathological basis, *e.g.* Pick's atrophy, that may be recognised. Delirious and confusional phases may give a deceptively bad impression, for sometimes, after they clear up, the patient can resume his old routine tolerably well.

Treatment.—This consists in providing for the patient as easy and familiar an environment as possible. Whether institutional treatment is necessary depends not only on the mental impairment but also on the patient's social level and the willingness of his relatives to look after him well enough. Patients often fit surprisingly well into hospital life and routine. Drugs are best avoided, and caution is necessary in letting the patient have the aperients he demands to relieve his—mainly delusional—constipation.

2. CEREBRAL ARTERIAL DISEASE AND HYPERTENSION

The characteristic features here are the focal symptoms. All else is indistinguishable clinically from senile and other cerebral conditions; of course, pathologically many senile brains show arterial degeneration too. The early or mild symptoms of cerebral arteriosclerosis are the same as those of "essential" hypertension; and very like those of many benign melancholias of late middle age.

Pathology.—Atheroma of the cerebral arteries is accompanied by nutritional changes—softening—in the brain tissue, falling into three stages, *viz.* necrosis, degeneration (with masses of granular phagocytes, containing fats and hæmosiderin) and sclerosis (in which cavities and scars of glial—astrocyte—and mesodermal tissue take the place of the necrotic cells). (See also p. 1597.) The cortex on the convexity of the brain may show microscopic areas of perivascular gliosis, but no softening. It is not yet possible to correlate the mental and the cerebral changes in these psychoses, except for the focal lesions.

Symptoms.—Since "essential" hypertension often precedes definite vascular disease and itself produces mental symptoms, a description of these symptoms serves also to describe the earlier stage of cerebral arterial degeneration. Along with headache, giddiness, tinnitus, faintness and insomnia, there may be disturbance of speech and writing—the former becoming slow and at times indistinct—and transient pareses and apraxia. Certain traits of personality may be intensified: the patient becomes irritable, egotistic, moody and easily tired, his conversation lumbers along where once it moved easily: he is depressed or paranoid; but there may be wide variation in the intensity of these changes, which are by no means always found. Brief phases of disturbed consciousness, lasting up to three weeks, may suddenly occur either in a form very like the "absences" of the epileptic, or as twilight states with hallucinations, ecstasy, incoherence, disturbed motility and agitation.

After this stage of neurasthenia and episodic disturbances, the patient with cerebral vascular disease may begin to have trouble in finding words: he perseverates a little, and is at a loss when anything unusual is required of him. His depression and hypochondriacal worries increase, he is distressed

by his own slowness and failures, and may attempt to kill himself. Emotional control falls off so that he weeps and storms when he would rather be calm. Nihilistic ideas may abound—his bowels have not been opened for six months, his trunk is a hollow cavity. Nocturnal delirium is frequent. Aphasia and apraxia are commonest after a focal complication.

The most important feature is the way the patient continues to look normal and sensible when already mildly demented. Sometimes transfer to the strange surroundings of hospital is too much for the hitherto well-preserved outward normality, and the patient goes to pieces, as he also may if he has to give up his usual work or move house.

Diagnosis.—Because a patient has generalised arterial disease, it does not follow that any neurotic symptoms he may show are due to the cerebral vessels being thus affected. Unless there are definite focal symptoms, or evidence of dementia, it is unsafe to hold the cerebral arteries responsible and to give a prognosis based on this. There is no known means of distinguishing many benign "neurasthenic depressions" and involuntional hypochondrias from those due to disease of the cerebral vessels. If there has not been any history of such tendencies until an attack at the age of 60 odd, the probability that it is an organic vascular disease is much higher. The distinction is all the more difficult because so many unstable persons develop arterial disease in later life; especially those prone to anxiety and other affective disorders. Neurological findings (see p. 1599) may be decisive in a doubtful case. The condition of the retinal arteries is not a reliable guide.

Course and Prognosis.—In definite cases of cerebral arterial disease with mental disorder the prognosis is necessarily bad, though the mental symptoms may only progress slowly, and the patient live another ten or twenty years. Much will depend on such sudden accidents as thrombosis or hæmorrhage. An episodic confusional state, perhaps even one produced by drugs, may suggest a needlessly gloomy prognosis. In cases of "essential" hypertension, the course of the mental illness is dependent on the general disturbance, and is often quite favourable. Symptoms that are apparently hysterical, occurring for the first time in middle life, are of bad omen.

Treatment.—Besides the general medical care of such patients, not a little can be achieved by psychiatric methods. In the early stages, where there is much anxiety and depression, too energetic physical investigation and treatment may do harm: reassurance and sedation can do much good. The less said to the patients about their blood pressure and their arteries the better. They should keep at work and in their accustomed surroundings as long as they can, unless an acute phase of the illness or depression intervene. Emotional upsets oftener aggravate their condition than physical ones, so they should be cushioned against such jolts. Their depression may necessitate hospital care, especially because of the risk of suicide, or because they are too irritable and neglectful to be at home any longer. If there be dementia, even of mild degree, the patient will probably remain in a mental hospital once he has gone there. It is, however, not easy to be sure about mild dementia being present; it can be counterfeited by passing disturbances, e.g. emotional ones.

3. HUNTINGTON'S CHOREA

(see p. 1703)

C. SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

Only the mental symptoms will be described here. Hypochondriacal and depressive reactions sometimes follow infection, or the risk of infection: such psychogenic illnesses do not belong under this rubric; occasionally, however, a patient's anxiety lest he be developing neuro-syphilis turns out to be justified. A syphilitic neurasthenia can occur in the early stages of the disease, due to a mild meningitis. The more severe meningo-encephalitis—*cerebral lues*—may be accompanied by disturbance of consciousness, even to the point of delirium or mild dementia: loss of initiative, euphoria or moroseness, poor judgment and impaired memory may persist and the patient be aware of them in greater measure than he is in general paralysis. These conditions are often complicated by the signs of premature arterial degeneration in the brain. The psychoses that accompany tabes are due to syphilitic changes in the brain, often complicated by alcohol, trauma, heart and kidney disease, and other exogenous factors; there are also depressive hypochondriacal reactions to the pains and other disabilities which the patient suffers.

GENERAL PARALYSIS OF THE INSANE.—Dementia is the constant sign of this mental picture; the old descriptions of a "classical" course with an expansive onset are fallacious, but general dementia is almost certain to occur in every case that is not treated early. All the other symptoms are either neurological and focal, or due to the patient's constitutional predisposition and previous experiences.

The dementia may at first be quite undetectable as such, because it appears under the deceptive guise of a neurasthenia, melancholia or mania; only gradually does the intellectual impairment become manifest. In the beginning of general paralysis, which is seldom abrupt (though it may need a careful inquiry to verify the prodromal symptoms), "functional" syndromes can be so "typical" and organic changes so slight that the most expert psychiatrist is misled; only by physical and serological examinations can he avoid a blunder. A faint degradation of personality, a lapse in social refinements may be the first indication of what is wrong. Then memory for the events of yesterday and last week becomes less trustworthy, what seemed at first a trivial absence of mind becomes serious incapacity, and yet the patient remains serene and outwardly indifferent to his lapses. As in senile and arteriosclerotic dementia, he may be all right so long as he is in an accustomed rut, but a holiday or a change reveals his infirmity. His mood and interests as the illness goes on become dull or labile, his rages are fleeting, his activities fussy; if, however, he is in a manic excitement, with little dementia as yet, the affective changes can be violent, and indeed dangerous, just as in a depressive phase the patient may kill himself. Sleepy and slow, careless about social usages, inattentive and ignorant of what he once knew well, the more demented patient cannot escape recognition as having an organic cerebral affection. Elementary problems in arithmetic and questions of general information are more than he can cope with. He gives easy assurances that he can do them, or puts his questioner off with airy explanations (*e.g.* that he has not had his spectacles by him lately); when pressed, he makes bad mistakes or becomes angry. The extent of his failure will, of course, depend not only on his dementia, but on his previous

intelligence and habits (*e.g.* a bank manager retains the capacity to do mental arithmetic when much else has gone). Inability to receive new impressions and to relate them to earlier memories co-operates with impaired judgment to give a gross but patchy and fluctuating amnesia. Because of these disturbances, and especially the bad judgment, patients may commit offences, ruin themselves by grotesque extravagance and brush aside facts that stare them in the face. They will put up with restrictions on their freedom, forgetting their protests soon after making them; silly reasons are sufficient for their compliance, and a tactfully offered cigarette or joke may divert their thought and feeling from some serious matter that angers them. Their delusions are due to the same disorders of memory and judgment, coloured by their general personality; sometimes they are confabulations, rationalisations for their having forgotten or spoilt something. If the patient had in health tendencies to euphoria and expansive behaviour, grandiose delusions and boasting will be to the fore. It is, however, not uncommon to find a fatuous euphoria, though there had not previously been affective swings and hypomania; in such patients one finds abundant proof of gross impairment of judgment, especially shown as defective insight. The most advanced dementia appears as a helpless, vegetative, bedridden state, sometimes accompanied by gross focal symptoms, such as aphasia and agnosia. The physical symptoms (see p. 1638) are much intermingled with the mental ones, as in the patient's clumsy movements and disturbed speech and handwriting: thus, in his writing he leaves out letters, syllables and words, repeats and transposes them, messes the paper with blots and sputters, writes across the lines, puts in meaningless strokes and leaves his mistakes uncorrected; the tremulous script shows interruptions in the usual smooth alternation and tempo of movement, the letters are of very uneven size and ill spaced. Articulatory and aphasic disturbances may affect the sense, intonation, timbre, rhythm and precision of utterance; they must not be evaluated in diagnosis, any more than the writing disorder may, without regard to the patient's previous normal script and speech and the circumstances under which he was writing or talking, since people, habitually untidy in their enunciation or handwriting, can exhibit many of these symptoms when tired or in a hurry.

Besides the above, atypical mental pictures may be seen either ordinarily or as the outcome of treatment with artificial fever. Paranoid states, hallucinosis, a Korsakoff syndrome, epileptiform excitement, hysterical disorders and catatonic symptoms of every kind (except *flexibilitas cerea*) may occur. Hallucinations are uncommon, except during fever or after malarial treatment; in the latter case they are often of paranoid colouring. Not the expansive form, but a simple progressive dementia is by far the commonest clinical picture; depressive, confusional, and hyperkinetic states are almost as frequent as the expansive.

In the "Lissauer" form the slowness of the dementia is remarkable in comparison with the conspicuous focal symptoms, such as the seizures without convulsions or loss of consciousness.

The effects of treatment upon the mental state are of great social moment. In a majority of the cases who do well the personality has the edge taken off it, there may be less initiative and force in mental activity, and emotion may be less controlled, especially in the proneness to anger or to frivolous

levity, yet the patient is able to return to his former work, even though it is responsible and complex; he could scarcely, however, except in the most favourable cases, learn a new job or adapt to new and exacting situations.

In the "*juvenile*" form there may be premonitory symptoms of excitability, grizzling, timidity and backwardness at school. Gradually the symptoms of dementia become plain, and if the onset be early enough, symptoms usually found in severe mental deficiency naturally appear, such as rhythmic or iterative movements, grimaces, repetitive chewing and sucking of an automatic kind, great restlessness and screaming attacks. Simple dementia is the usual form; grandiose ideas are exceptional. If the illness begins before the age of 10 or 11 years speech and writing may be completely lost, or reduced to a senseless smattering.

For *prognosis* and *treatment*, see pp. 1640, 1641, and 1653.

D. OTHER CEREBRAL DISEASES

LETHARGIC ENCEPHALITIS.—The mental disturbance of the acute attack may merge into a hyperkinetic excitement, with choreiform and athetoid movements, insomnia, generalised pains, mild delirium and, occasionally, catatonic symptoms: this seldom lasts more than a few weeks. There may be subsequently a neurasthenic fatigue and irritability with headaches and poor sleep. The distinction between what is neurological and what is psychiatric in the symptoms could scarcely ever be more difficult than in this disease. The motor disturbances, such as oculogyric crises, are not merely responsive to emotional and other psychogenic influences, they are inseparable from concomitant mental happenings (*e.g.* the surging up of anxiety or obsessions), and whole patterns of complicated behaviour, *e.g.* breathing, may be involved. The motor rigidity of the patient's Parkinsonian state may be paralleled by a lack of the normal drive and fluidity of thought or behaviour. Memory, however, and grasp are unaffected. The obsessional symptoms sometimes occur quite apart from oculogyric crises, and may greatly distress the patient. Depressive phases may result in suicide, which is fostered, as it were, by the keen appreciation which many patients have of their ruined careers and their almost imbecile appearance, so different from what they were and, indeed, from what they still know themselves to be. Paranoid, and especially schizophrenic, symptoms may develop in the later stages.

The younger the patient the more likely is it that he will develop disagreeable anomalies of personality, and have attacks of restlessness or even be permanently restless. Many children and adolescents after their acute attack become social problems: they play stupid or cruel tricks, they set everyone they can by the ears, they may steal, behave sexually in an outrageous way or accuse others of sexual offences against them. Their activity is not always purposive, nor always antisocial; they make the same impression as a monkey might who is sometimes mischievous but always on the move. There may be no Parkinsonism in these cases. The prognosis is not good, and they almost always do better when subjected to the regime of an appropriate institution; they do badly at home or in places where

what may be termed normal delinquents and "social problems" are cared for.

SYDENHAM'S CHOREA.—The usual mental changes here are lability of affect and irritability. These are seen as naughtiness, outbursts of anger or crying, resentment at sudden noise or light; in others there is lessened spontaneity, often masked by the choreic movements. In more severe cases, especially in older children, these changes are accentuated; in the fleeting phases of anger or terror there may be slight delusional trends. Still more severe forms, with delirium, hallucinations, delusions of persecution and much excitement, are seen in adults, *e.g.* in chorea gravidarum.

The tics and compulsive utterances (Gilles de la Tourette's disease) which may follow chorea are evidence of the interplay between hereditary, psychic and structural factors. Chorea is more prone to occur in those whose families show nervous disorders, especially schizophrenia. The motor after-effects, especially tics, appear and disappear under emotional influences; they are also conditioned by the original choreic disturbance of neuromuscular function. The obscene ejaculations of la Tourette's disease are dependent on much the same articulatory and respiratory hyperkinesias as are the breathing spasms of encephalitis lethargica, though they are also dependent on psychological tendencies and experiences. They illustrate how psychological influences work through available bodily structures and functions, whether morbid or healthy. The obsessional element in this affection is comparable to that in encephalitis lethargica.

DISSEMINATED SCLEROSIS.—Slight deviations from mental health are frequent, but obvious ones rare in this disease. Affective lability may be conjoined with a slight disorder of judgment, so that a baseless euphoria develops, but this is not universal, and many of the patients are depressed. Acute outbursts of excitement, hallucinosis or delirium occur in a few cases, and dementia in the advanced stages. The most important mental disorder in them is that which appears as hysteria. A hysterical personality has not been present in these patients before the disease began, and the symptoms are in that respect only dubiously hysterical: they do, however, in other respects conform, in that they can be evoked psychologically and removed psychologically; they may centre on, and elaborate, actual anomalies, *e.g.* of movement or sensation, and may still yield to hypnosis or other psychological measures. They can greatly confuse the diagnosis.

SCHILDER'S DISEASE.—In this disease profound dementia gradually develops along with the blindness, deafness, aphasia and agnosia and other focal symptoms. In the juvenile cases there may be at first disturbances of behaviour like those of juvenile encephalitis lethargica.

PARALYSIS AGITANS.—This may be accompanied by hypochondriacal depression. Sometimes this is an expression of the cerebral disease which also causes the Parkinsonism, and in that case the prognosis is bad; sometimes it is a recurrence of depressive attacks which have occurred at times of stress earlier in the patient's life, and then the outlook is fairly favourable. Senile dementia is, of course, not infrequent in these elderly patients.

CEREBRAL TUMOUR.—Apart from any aphasia and apraxia, the mental state here is more closely related to general intracranial tension than to any local disturbance. The size and rate of growth of the tumour are therefore important in this regard. If rapidly growing, there is more disturbance of

consciousness, with impaired memory, disorientation, incoherence and, sometimes, hallucinations and confabulation; this clouding of the mind fluctuates a good deal. In more slowly growing tumours, lucidity is preserved and change of disposition is the prominent feature. The patient's earlier tendencies get freer play, unsuspected ones appear, and a series of foolish investments, for example, or homosexual escapades may for years divert attention from the organic disease. The moria, or fatuous wit and cheerfulness, often attributed to frontal tumours but also found in other cerebral diseases, may give the impression of being a hysterical pseudo-dementia; other apparently psychogenic symptoms may prove misleading. A straightforward depressive attack can occur, or indeed any "functional" syndrome.

Hallucinations may depend on a focal lesion, as in the cases in which they are limited to the hemianopic field, or are solely of taste and smell.

In CEREBRAL ABSCESS the mental symptoms are those of tumour with or without others due to meningitis. In ACUTE MENINGITIS there may be delirium, preceded during the prodromal stage by irritable apathy, and followed by months of moody neurasthenia.

CEREBRAL TRAUMA.—After concussion there is retrograde amnesia; the extent of this and the rapidity with which it diminishes depends on the amount of cerebral damage. In rare instances delirium ensues: it has little that is characteristic, and is more frequent in alcoholic and elderly people; a Korsakoff syndrome may develop. Twilight-states are rather more common; during them acts of violence may be committed, as in epilepsy, and afterwards quite forgotten. Traumatic epilepsy may follow. The later changes in personality are commonly those that may be found lingering after any toxic or other structural impairment of the brain. But sometimes the disturbance of consciousness is more persistent, the intellectual damage greater, the deterioration progressive; in such cases there is usually cerebral arterial disease, an unrecognised alcoholism, cerebral tumour, G.P.I., or some other complicating factor. In predisposed persons the cranial injury may be responsible for a melancholic attack, schizophrenia, or other "functional" syndrome; the prognosis is usually good even if the illness lasts many months.

Hysterical symptoms occur frequently after cerebral trauma. This is partly because of the site of the injury, which favours vague physiogenic symptoms that respond readily to emotional and other psychological influences. Many of these symptoms are, however, produced by psychical rather than physical mechanisms. Not injured cells, but mental attitudes are at the bottom of the tremblings, faintings, weakness, paræsthesiæ and other troubles so often the sequel of a trauma in itself little likely to have such effects. They are not responses to the actual injury, but to the situation created by the injury. Compensation may play a large part in this. It is as unwise to dub all such vague post-traumatic phenomena hysterical as to attribute them entirely to the direct injury. If there is slight amnesia of the typical kind, with difficulty in concentration and headache, it is fairly probable that these are physiogenic residues; if there has been an interval between the actual concussion and the appearance of the indeterminate symptoms, and an adequate psychogenesis (*e.g.* claims for compensation, with repeated medical examinations, and patent uncertainty among the experts) the condition is likely to be hysterical. Much will, of course, depend on the neuro-

logical and other findings, including the demonstration of localised lesions; thus, damage to the frontal lobes may much change the personality, and in other sites be responsible for an apraxia, say, or a visual defect.

EPILEPSY.—Although the motor seizure is the chief symptom of epilepsy and the only decisive one in diagnosis, there are minor or equivalent symptoms, as well as delirium, twilight-states and dementia, to be included among the mental disorders of this illness. The minor symptoms are much rarer in symptomatic epilepsies than in the "idiopathic" form.

Instead of a major fit the patient may become unconscious; or he may pass into a twilight-state in which for a few minutes or longer he wanders about in a dazed way and does inappropriate things, having afterwards complete amnesia for all this; or there may be a sudden interruption of action and speech, during which the patient remains immobile or makes some automatic or aimless movements. Epileptic furor is a delirious state in which acts of violence may be committed: it lasts often for several days, is accompanied by disorientation and hallucinosis, and is much rarer than is popularly or forensically supposed. All the states of disturbed consciousness mentioned above are most often seen as equivalents for a seizure; the twilight-states, however, may precede the motor attack, follow it, or be accompanied by a few violent clonic movements.

Apart from their seizures epileptics are prone to swings of mood—towards anger, shallow sentimentalism or depression—which may pass over into a fugue, during which the patient wanders a long way from home.

The likelihood of dementia later cannot be inferred from the symptoms of the epilepsy, except that it is greater if attacks occur very often. Apparent dementia may be the result of intoxication with bromide, or of the idleness and sterile life in an institution. When there is genuine dementia, it begins as a faint loss of interest and concentration, with increased sensitiveness to supposed slights, then memory falls off somewhat, the trivial and the important are muddled together, and the patient talks with much circumlocution; he is fond of needless system, assumes and parades virtues he has not, *e.g.* an intellectual bias or a devout spirit, and is childishly pleased when anyone praises him. Later, a profound dementia may supervene, but this is not common; it is open to question whether the changes of character just described are necessarily part of a dementing process. It is certain that many epileptics who exhibit some of the most disagreeable features of this sort never become plainly demented, and that many severe epileptics are free not only from dementia but also from these traits. There is ground for regarding this impulsive, pretentious, fawning and snarling way of some epileptics as partly a variable expression of their constitutional predisposition (to which the motor seizures are likewise due), and partly as a reaction to their situation. Consequently it is much less evident, or not evident at all, in those who in spite of their fits live comparatively normal lives.

E. INTOXICATIONS

1. ALCOHOLIC DISORDERS

Alcohol is so permissible and trusted a poison, so easy of access for those who wish to escape from their troubles, that it is resorted to in excess

by maladjusted persons of every type ; consequently its effects may complicate or be complicated by the psychopathic anomaly which favoured the taking of the drug, *e.g.* episodic excitement or depression, anxiety, cerebral arterial disease, syphilis, paranoid states, epilepsy, hysteria. The acute effects of a single dose of alcohol are either the well-known phenomena of intoxication, or an excitement (*mania a potu*) sometimes with clouding of consciousness. The excitement is commoner in people with cerebral trauma, arteriosclerosis, epilepsy, or unstable hysterical personality, and in them may lead to acts of violence ; rarely it may occur in normal persons who have taken alcohol when they were exhausted or upset.

In chronic drunkards, a dementing *demoralisation* can occur. Their narrowing of interest, superficiality of thought, weakness of memory and moral decrepitude are reminiscent of what happens in many epileptics and some early general paralytics. The crudeness and even brutality of their conduct is in ill accord with their maudlin prating about virtues and their pothouse jollity. The mood of these men can be as labile as their abandonment to it is constant : they pass from rage to weeping, and laugh soon after, with no shame for themselves and no thought for the miseries they put on their families. Such degradation is of course far from being the rule : some chronic alcoholics become only cheap editions of themselves, with their former qualities underlined or smudged rather than defaced ; they are perhaps weak and irritable, untrustworthy or lying, but not given to savage fury, nor grossly damaged in judgment and social feeling. Some of them develop delusions, especially of jealousy. They collect, as paranoid people of other kinds do, scraps of alleged evidence which they piece together to prove their suspicions right ; complicated delusions of persecution, however, they rarely develop. Sometimes the delusions of jealousy fade as the patient gets more and more facile, but more often they persist as a chronic insanity and are of the greatest danger to the suspected wife ; murder is not unknown in such cases. The nature of the delusions is to be attributed in part to the lessened sexual potency of chronic drunkards and to the domestic wretchedness and aversion they often create, as well as to the same causes as in "functional" paranoid states, where such delusions are also common, especially in middle life.

The symptoms of *delirium tremens* would appear to differ in nothing but severity from the essential symptoms of any delirium (see p. 1824). Some observers, however, deny this. The anxiety amounts to terror, mixed oddly enough with euphoria ; optic and cutaneous hallucinations are vivid and restlessness can be extreme. There is almost complete sleeplessness, and much disorientation as to time and place, but not as to personal identity. The patient's attention wavers between his hallucinated and his actual surroundings, but can usually be caught and held for a few moments. He is very suggestible, as most chronic drunkards are ; pressing on his eyeballs, for example, will very likely make him see whatever one tells him he sees, and he will read aloud from a blank sheet if one wants him to. Among the visual hallucinations may be miniature ones (*micropsia*), and many illusionary perceptions. The content of the hallucinations changes rapidly, and a false perception in one field (*e.g.* a vestibular one) tends to evoke others (*e.g.* of sight, touch, or hearing). Insight is commonly lacking ; afterwards there is patchy amnesia for what has happened in the

delirium. The death rate, with adequate treatment, has been about one in seven ; and of those who die most of the men are under 40, and most of the women under 45.

In *acute alcoholic hallucinosis* auditory hallucinations of a persecutory kind are prominent and consciousness is not notably clouded. It is rarer than delirium tremens, and is more prone to follow a bout or orgy of drunkenness. The patient is frightened, but not obviously out of his mind ; he is correctly orientated and may be able to go about his business for days. Auditory hallucinations are vivid and insistent, after a premonitory phase in which there are sensitiveness to sounds, and roaring, singing, hissing, etc., in the ears. Tormenting voices, sharply localised but seldom fastened upon bystanders, abuse, threaten or discuss the patient : they may say his wife plays him false, order him to kill himself, describe his every movement, especially at private moments in the bath or lavatory, cast up his more shameful secrets at him, shout his thoughts aloud. There may be many voices, of men, women, and children, all talking together and perhaps rising and falling in the same rhythm as his pulse. They are so real that the patient answers them ; he may be in doubt about the presence of his tormentors and may shout back insults to see if a blow will follow from the owners of these evasive pursuing voices. Hallucinations of sight and other senses are far less prominent than those of hearing ; cutaneous ones, e.g. of being sprayed with a cold liquid, are not uncommon. Delusions are usually inconspicuous : they are as a rule attempts to account for the hallucinations, and they commonly fade out of the picture or pass into a chronic persecutory disorder. Flight or acts of violence may result from the patient's fear or anger. Usually it is a matter of only 2 or 3 weeks before the hallucinosis clears up, if no further alcohol be drunk ; sometimes, however, a delusional state, more rarely a Korsakoff picture, supervenes in predisposed persons. After recovery, there is little or no amnesia for the events of the hallucinosis. Relapse is to be feared if the drinking goes on.

The *Korsakoff* syndrome is not invariably associated with polyneuritis. Nor, as stated on p. 1825, is it limited to alcoholism ; it can follow other severe chemical and mechanical injuries to the brain. In alcoholics it is commoner in middle life, developing either insidiously in the course of chronic alcoholic demoralisation, or after delirium tremens ; women are especially prone to develop this syndrome after the delirium. The symptoms have already been described. The disorientation, superficial appearance of clarity, incapacity for initial perception and subsequent recall (extending often to most of the material of memory) yet with retention of some capacity for learning by repetition, along with confabulation, dullness of emotion and initiative, and grossly impaired judgment make a striking picture. Complete recovery is on the whole uncommon, occurring in less than a quarter of all cases. The mortality rate is higher in women and older people, in those with acute onset and with a red-cell count below 3,000,000, or with a rise in the protein content of the C.S.F. It does not correlate with the severity of the peripheral neuritis.

Chronic Delusional States have been referred to above ; they are sometimes called alcoholic paranoia, but inappropriately so ; jealousy is the commonest and most dangerous feature. Alcoholic epilepsy has been described. It is no more than a symptomatic epilepsy, often atypical ; some-

times in unstable hysterical patients it may be brought about through over-breathing when intoxicated.

Diagnosis.—The diagnosis of alcoholic psychoses must depend much more on a history of drunkenness in any patient than on his clinical psychiatric features, none of which are limited to alcoholic disorder. Since, however, alcohol is far the commonest cause of most of the toxic abnormalities described, it can be safely presumed in some cases in which the certain history of addiction is unobtainable.

Differential diagnosis, so far as ætiology is concerned, will turn on somatic findings, including the results of chemical and biological tests. If the form of the disorder is in question, the chief diagnostic difficulty arises with acute hallucinosis and the chronic delusional varieties. A hallucinosis of similar type can occur in schizophrenia and in affective disorders, but in the latter is recognisable by the ideas of self-reproach expressed; the differentiation from schizophrenia is difficult, since in many of the cases the progress of the disorder is towards a chronic schizophrenic psychosis, and one may suppose that in these patients the intoxication had activated, as it were, the same mechanisms as those involved in schizophrenia, or had complicated a schizophrenic illness. This applies also to the chronic psychosis with delusions of jealousy. There is no value in differentiating carefully the clinical varieties of alcoholic psychoses, since they overlap.

Treatment.—Prophylaxis is the main thing. The incidence of alcoholic psychoses in London has fallen to one-third of what it was before the War of 1914–1918; and this may be attributed almost entirely to social influences, of which the increased cost of alcoholic drinks is the most effective. Individual prophylaxis is scarcely to be considered, save as a by-product of psychiatric treatment, since a great proportion of unstable persons are potential drunkards, and in any case we cannot yet tell which alcoholics will become mentally ill through their drinking. Social prophylaxis is so immeasurably better in forestalling alcoholism and the psychoses and degradation that sometimes spring from alcoholism, that deliberate individual prevention is here negligible.

When alcoholism is itself to be treated, independently of its ill-effects upon mental health, the problem is that of any drug addiction. Absolute removal of the drug is essential, yet this cannot be done unless the patient, the treatment, or the environment is exceptional. A suitably exceptional environment can be provided by getting the patient into a hospital or home where he cannot obtain the alcohol he desires, but the other requirements are more difficult to meet. The exceptional patient who after years of excess can put aside alcohol while it is within his reach is as rare as the treatment that can bring him to this state; and when such a change does occur, a great emotional upheaval, *e.g.* bereavement, religious conversion, fear of death, has usually led to it. For the most part, treatment of alcoholism without restrictions upon access to the drug is a failure; the restrictions must at first be imposed from without, not left to the patient's self-control and judgment. He should remain in the hospital or home for a year at least. Psychotherapy is an essential feature of the treatment in those cases in which inner struggles and neurotic disabilities have been the basis for the addiction; it must, however, be conjoined with social and other measures (see p. 1815). Special chemical methods have little or no value.

The grosser mental disorders due to alcohol need hospital treatment. Delirium tremens should be treated as far as possible without hypnotics, which have little effect upon the excitement and sleeplessness unless employed in dangerous doses ; if any, paraldehyde or hyoscine should be used. Circulatory failure and accidental self-injury are most to be guarded against. The continuous bath at body temperature is sometimes beneficial ; otherwise the patient should be in bed with a minimum of necessary restraint, the company of an experienced nurse, and ensurance of adequate diet—mainly fluids and glucose and large amounts of vitamin B₁. No alcohol should be given. Occasionally lumbar puncture is helpful.

Especial care must be taken against the early discharge from hospital of alcoholics with delusions of jealousy. If they have been certified, they may add a deep resentment on this score to their other grounds of morbid hatred, and there is grave danger that they may, if they resume drinking, attack their wives murderously.

2. MORPHINISM

Only the effects of this belong among the organic disorders ; its causes, and the incapacity of the addict to escape from it, are due to social and intrinsic factors, not to any physical damage. Weak, unstable, unhappy people, *e.g.* many homosexuals, are most likely to become addicts ; it is rare to meet an addict who has not shown pronounced psychopathic traits before his addiction began ; and few of those who profess to have been seduced into the habit by more or less injudicious administration of morphine for some pain they had, are in that telling the whole truth. Yet it is a wise caution that withholds morphine from all chronic disease that is not hopelessly progressive, and hesitates to prescribe it at all for those whose personality or opportunities make the risk of addiction greater.

Symptoms.—These are not at first noteworthy, unless the patient be seen during the next 2 or 3 hours after he has taken his drug. The symptoms of withdrawal, sometimes severe, are more likely to occur in those whose tolerance has been raised by the habit ; they consist of yawning, sneezing, overflow of tears and saliva, fullness in the head, then restless movements, malaise, twitching in the face, tremors, palpitation, indigestion, vomiting, diarrhoea, strangury, sleeplessness, and circulatory upset which may go on to collapse.

It is difficult to judge how far the drug itself is responsible for the demoralisation that is met with in chronic morphine addicts ; probably as important in causing it are the psychopathic personality of the addict, and the underhand life he must lead. Laziness and lying are frequent, and the patient may resort to subterfuges, or even crimes, to get his drug. Dementia does not occur ; delirium is rare. The physical effects of chronic morphinism are dryness of the skin, hair, and nails, constipation and anorexia, partial impotence, and poor resistance to infection.

Prognosis.—This is poor as regards recovery from the addiction. The more normal the patient's personality, the better the outlook. After apparent cure, however, relapse is frequent, and the outlook is then correspondingly worse unless the patient can be stopped from getting the drug. Many morphine addicts also take alcohol, cocaine, and such other drugs as they can get. Suicide with morphine is not uncommon, for obvious reasons. Death

is sometimes the result of cutaneous infections, especially when the patient is grossly undernourished.

Treatment.—This must be in an appropriate institution; general hospitals seldom have the necessary facilities. Treatment at home is bound to be a failure. It should be impossible for the patient, however skilled in stratagems, to get hold of morphine. He should, if possible, contract to stay for at least 2 months. The withdrawal of the drug should be abrupt and total, except in very debilitated patients; "tapering-off" prolongs the distressing period of withdrawal symptoms and gives opportunity for the patient to develop psychopathic reactions and dodges. If the patient's condition demand a gradual withdrawal, this need seldom extend over more than a fortnight. When an abrupt end has been put to the taking of morphine, the rigours of the first 4 or 5 days (after which the worst is over) can be alleviated by sedatives in fairly large doses, copious fluids, warm baths, massage and fresh air; gastric lavage and alkalies help, and for circulatory symptoms caffeine may be given, with small doses of morphine also in very severe cases. After this phase is past, sleeplessness may still be intractable: in giving sedatives or hypnotics for this, barbiturates and paraldehyde, with occasional doses of hyoscine, are the safest, but should be used sparingly, with frequent changes and, it need hardly be said, complete refusal to let the patient know what he is having. Psychological treatment is of great importance, but there is no specific technique applicable to this addiction. To be successful, the psychological treatment requires the co-operation of the patient's family as well as of the patient himself, and it will be wise for him to keep in touch with his physician for years. The great difficulty of getting the drug in this country, because of the vigilance of the Home Office, is an immensely favourable factor after active medical treatment has ceased. It is wise for the patients to eschew alcohol and, of course, all hypnotic drugs.

3. OTHER INTOXICATIONS

Cocainism is very rare in England. The causes and symptoms are similar to those of other addictions, *e.g.* alcohol and morphine. Deliria, hallucinations, Korsakoff syndrome, or demoralisation can occur: in the former conditions microptic and cutaneous hallucinations, *e.g.* of bugs under the skin, are prominent. A paranoid schizophrenic state sometimes comes on, usually clearing up after the drug has been stopped. In treatment what was said of morphinism mostly applies here, though withdrawal symptoms are far less severe.

Bromide intoxication is common, and often unrecognised. It is probably now in England the most frequent cause, after alcohol, of mental disorder due to a drug. All the organic syndromes can occur, usually as complications of a pre-existing disorder for which the bromide has been prescribed. A delirium and a paranoid confusional state or lachrymose amnesic syndrome are the usual forms. In the more long-standing and severe forms cachexia, circulatory failure, and even death may occur. Acne and other physical signs of intoxication or idiosyncrasy may not be evident. Diagnosis rests on the history and the amount of bromide found in the blood, more than 50 mgms. per 100 c.c. being indicative of a considerable intake or retention of

bromide. Treatment consists in complete withdrawal of the drug, promotion of its excretion by giving sodium chloride and fluids in large quantities, and general physical and psychiatric measures.

Barbituric acid derivatives, such as barbitone (veronal) and phenobarbitone (luminal), can in rare cases lead to apparent euphoric dementia, likely to be mistaken for general paralysis because of the ataxia, tremor, articulatory disorder, and other neurological signs. Recovery is the rule when the drug is stopped. Picrotoxin may be needed for acute poisoning. For the addiction itself, essentially the same problems and methods of treatment are in question as with other drug addiction. This applies also to *ether*, *chloral*, and *paraldehyde*.

Mercury and *lead* poisoning may lead to mental disorder (see pp. 376 and 366), *manganese* to a Parkinsonian syndrome with compulsive symptoms (reminiscent of encephalitis lethargica) and a mild paranoid or euphoric dementia; and *benzene* or *carbon disulphide* may cause delirium.

Acute carbon monoxide poisoning in rare instances leaves behind severe mental disorder of the amnesic-aphasic kind, which may not become apparent until several weeks after the recovery of consciousness. More commonly, it results in a clinical picture almost indistinguishable from hysteria; this may take months to clear up, and is in no wise benefited by psychotherapy. Chronic poisoning by small quantities of carbon monoxide causes neurasthenia.

F. INFECTIONS AND EXHAUSTIVE DISORDERS

1. INFECTIOUS TOXÆMIAS

Delirium and a Korsakoff syndrome are the more acute, and *naurasthenia* the milder, signs of mental disorder due to an infectious fever. In many of the cases, however, in which mental disorder is attributed to "sepsis", or other infection, either the mental changes are unconnected with the infectious process or there has not been an infectious process, as is often found when one inquires into an alleged attack of "influenza" and finds it was nothing of the kind. There are three possibilities: the mental changes are mainly due to the infection; they are independent of the infection; they are partly due to the infection and partly to other, usually constitutional, causes. The depression that occurs in and after many infections is usually of the third category mentioned; delirium instances the first possibility; and the second is often exemplified when some non-organic syndrome is put down to "latent sepsis" in the bowel or the tooth socket or some rather inaccessible cavity. It is not that infectious toxæmia is always innocent of doing this sort of psychiatric harm, but that it is far too often charged with the offence when it is blameless.

Wherever a delirium or other mental disturbance of one infection differs from that of another, *e.g.* the delirium of typhoid from that of pneumonia, the difference lies only in the severity and duration of the physical effects of the intoxication and in the peculiarities of the affected person; no mental symptoms specific to any one infection can be demonstrated. Among the individual peculiarities just mentioned must be included a constitutional

predisposition or readiness to respond with symptomatic psychoses to mainly physical ills.

There are a few infections that hardly ever cause mental disturbance, *e.g.* tetanus and diphtheria; others do so by their local cerebral incidence, *e.g.* malaria or encephalitis lethargica. *Tuberculosis*, from its chronicity and its occasional incidence on the central nervous system; has a special position. Its treatment, moreover, especially in the pulmonary form, necessitates an abnormal, unsatisfying life for a time, and this with the toxæmia seems to be responsible for euphoric or anxious restlessness in which erotic tendencies and irritability are often prominent. *Spes phthisica* is partly attributable to toxic euphoria, in part it is a form of over-compensation for fear.

2. EXHAUSTION AND INANITION

These, especially if conjoined with some shattering experience—an earthquake, an invasion, a bereavement—bring about severe mental disturbance, *e.g.* a twilight-state or a delirium. *Hemorrhage* and cachexia may be responsible for “light-headedness,” as in advanced carcinoma, or after a severe operation.

G. METABOLIC, ENDOCRINE, AND VISCERAL DISEASE

1. METABOLIC DISORDERS

Various metabolic disorders can similarly, *i.e.* non-specifically, affect mental health. *Diabetes*, for example, which is especially frequent in families with a predisposition to affective psychosis, may be accompanied by transient phases of depression, anxiety or excitement which correspond to changes in the blood-sugar level, or a ketosis may be ushered in by mild delirium. A diabetic pseudoparesis, with peripheral neuritis, may cause slight difficulty in diagnosis. In children, insufficient carbohydrates may be responsible for anxiety, naughtiness, and other disturbances of behaviour. Anomalous psychic states may be produced in the rare condition of hyperinsulinism, and be mistaken for hysteria or an anxiety state of the psychogenic sort. *Gout* may occur in people predisposed to affective disorder; often a depressive phase precedes an attack. Alkalosis and anoxæmia may each be the cause of mental disturbance of the organic type. In *pernicious anæmia* there may be symptoms, *e.g.* an acute confusional state, referable to the structural changes in the central nervous system, but more often depression occurs without “organic” features; mania can also occur, and in some cases a chronic paranoid condition. The more “organic” the picture, the poorer the prognosis for a return to mental health. Of deficiency diseases *pellagra* is the one most commonly productive of mental disorder. It must be remembered that a long-standing anorexia, of psychogenic origin, or occurring in the course of a chronic melancholia, may itself lead to a pellagroid condition, so that the symptoms of mental disorder will then be those of the original illness plus those due to the deficiency. The clinical picture is sometimes very like that of hysteria; or the usual organic

syndromes may be produced, especially florid confusion with perhaps hallucinations of fire.

In the metabolic disorders just mentioned the physical phenomena are relatively coarse and obvious. It is in some cases proven and in others highly probable that less obvious metabolic disturbances are either among the primary symptoms of "functional" mental illness, or are its pathological basis. The acid-base equilibrium and the electrolytes of the blood, the metabolism of carbohydrate, fat, and protein, and the chemical regulation of the vegetative activities are all, in such forms of mental illness as schizophrenia and mania, subject to changes which have not as yet been used in the pathology or treatment of these conditions, because the findings are not sufficiently constant or specific; it is also likely that our methods of investigation are not delicate enough.

2. ENDOCRINE DISORDERS

These play a more prominent rôle in the investigations than in the clinical practice of psychiatry. Many endocrine preparations have, it is true, been administered to schizophrenic, sexually perverted, and melancholic patients, either empirically or in accordance with a premature and ill-devised theory, but the good results of all this are negligible. Estrin treatment of menopausal neuro-vegetative symptoms is a rational procedure. The blind use of the endocrine glands in the theory and practice of psychiatry has had its day.

Exophthalmic goitre is more prone to occur in anxious, nervous people, especially after some sudden shock. The usual concomitants—restlessness, tension, irritability, difficulty of concentration, and liability to sudden changes of mood—may be complicated by a definite mania or depression and, if the disease be severe or advanced, delirium and confusion may supervene. Though such organic syndromes mean as a rule a bad prognosis, they sometimes clear up dramatically after operation. The interaction of constitutional and psychogenic factors with the actual thyrogenic intoxication^a makes some treatment of the anxiety by psychological as well as other methods^s desirable in many cases of exophthalmic goitre, either as a preliminary or supplement to partial thyroidectomy.

In adult *myxœdema* the slowing of mental activity may sometimes be accompanied by a chronic paranoid psychosis, or there may be a phase of excitement with hallucinations; the variety of syndromes that can occur is referable to pre-existing constitutional tendencies and to the varying severity and rapidity of development of the thyroid deficiency. An apparently "functional" syndrome may precede the overt myxœdema.

Juvenile and congenital myxœdema is described elsewhere (see p. 495).

Tetany may be signalled by epileptiform seizures, or there may be a proneness to psychogenic fits; thus the patient may spontaneously overbreathe until a sufficient disturbance of calcium, etc., results in an induced convulsion. Hysterics sometimes use hyperventilation in this way to induce a passing tetany. In severe tetany a resistive lethargy or an excited incoherent confusion may occur.

Pituitary diseases are more often accompanied by mental symptoms that are a comprehensible reaction to the physical symptoms than by organic syndromes; the latter when they occur may be due to increased intracranial

tension. In acromegaly, depression, reserve, touchiness, and irritability are not surprising, though some acromegalics remain cheerful as long as their disabilities are moderate, and sometimes there is a blindness to the disease, a lack of insight, even when it is advanced. In dystrophia adiposo-genitalis a rather childish placidity may be met. In adiposis dolorosa depression may be severe, or hysterical symptoms may develop. Simmonds's disease may be accompanied by depression, severe anorexia, reaction to the psychosexual disturbance, and, in the later phases, by organic syndromes due to the cachexia. Similarly, disorders of pituitary function have been found in some cases of "anorexia nervosa." In Cushing's basophil syndrome depression and other mental disturbances can occur: we have seen a severe paraphrenia develop during the course of the illness.

Addison's disease is accompanied by a neurasthenia of which for a time the physical basis may be quite overlooked (as may also occur in myasthenia gravis); in the later stages delirium has been known to occur.

Sexual epochs may in women be associated with mental disorder of the organic type, e.g. some psychoses of pregnancy and the puerperium. During pregnancy plain psychosis is rare, but hysterical symptoms, depression, and anxiety are fairly common, especially if the mother is reluctant to have another baby; a gross psychosis may, however, break out during the latter months of pregnancy. The organic mental syndromes may develop along with polyneuritis, eclampsia, or chorea gravidarum. Termination of the pregnancy is called for on account of the mental condition when there are symptoms of organic psychosis which are likely to get worse, a history of suicidal attempts or infanticide in connection with previous pregnancies and a depression again in this one, or if on other grounds there is a clear risk of suicide or other untoward result of the mental illness, should pregnancy continue. The decision is often a very difficult one, requiring an expert knowledge of psychiatry for the careful appraisal of aetiology and prognosis essential in every case. The question must turn mainly on the therapeutic value of terminating the pregnancy, so far as the mother's mental state is concerned, as well as upon the stage of pregnancy reached.

In the puerperium "functional" psychoses often develop in predisposed women; if there be septicæmia as well, a confusional state or a delirium, followed by a period of neurasthenia, may occur. In many cases the delirious puerperal psychosis clears up in a week or two; the more endogenous varieties have sometimes a less satisfactory outcome than their form and onset suggest. Infanticide may occur in a puerperal psychosis, especially if the mother has, while pregnant, felt resentful at having a baby or been troubled by murderous preoccupations, e.g. obsessions. Psychoses of lactation are rare, and seldom of the organic type. Menstruation is apt to be associated with depression, irritability, and languor in many women, especially during the few days before the period begins; there are no menstrual psychoses, but the liability to suicide and to psychopathic reactions is somewhat higher at this time. Puberty and the climacteric are periods of stress during which schizophrenic and affective disorders may occur. The effects of castration are dependent on the age at which the gonads are removed: intellectual development is unaffected, but the emotional and conative activities of those castrated in adult life may be impaired. Neurasthenic symptoms are frequent, and in women anxiety symptoms may appear.

3. VISCERAL DISEASE

This may be directly responsible for mental disorder of the organic type. Thus *cardiac* disorders predispose to an anxiety, which at night may take the form of mild delirium, with restlessness, terror, disorientation, and auditory and sometimes visual hallucinations. With improvement in the circulation, the mental symptoms disappear, or remain only as a moody unrest. Reference has already been made to arterial hypertension (see p. 1829). The connection between *alimentary* disorders and neurasthenic states is well attested, and is striking in children. Jaundice may be accompanied by severe depression, but seldom leads to delirium, save in the case of acute yellow atrophy. *Uremia* may disturb consciousness greatly, in the form of any of the organic syndromes, from a twilight-state to a euphoric dementia; a Korsakoff condition can occur, but is infrequent.

II. MENTAL DEFICIENCY

As already stated, there is nothing in principle to separate these from other forms of cerebral impairment save that they occur at an earlier stage of life. As with mental disorder, they shade into normality so that no man can say where stupidity ends and feeble-mindedness begins. Again, as with mental disorder, the same clinical picture may be due to a variety of causes ranging from heredity to trauma. They are, moreover, delimited rather by social than by other criteria, and they are not definitely associated with any constant pathological findings. In that they are capable of only limited improvement when well established, and that the intellectual functions are more obviously damaged than any others, their similarity to dementia is easily seen. They are not by any means cases of purely intellectual defect; they represent, it is true, one extreme on the scale which has people of great intellectual ability at its other end; but they are also examples of a general impairment of mind, affecting the emotional and conative functions, and often associated with a more general impairment of the whole organism, which may be seen in its physical structure. Since the milder forms are indistinguishable (except on an arbitrary reckoning) from what may be termed normal stupidity, it is difficult to use rigorously the official definition of mental defect, as a condition of arrested or incomplete development of mind existing before the age of 18 years, whether arising from inherent causes or induced by disease or injury; but the description is serviceable. It should be recognised that, just as "psychosis" differs from "neurosis" only in a rough social sense, turning on the need for special care; and "neurosis" from "normality" only in respect of the limitations the former imposes on one's daily life as a social organism, so does the distinction between normality and feeble-mindedness, and between gross or certifiable deficiency and the lesser forms, turn on the social adaptation of the person in question. To complete the points of similarity there is recognised a "moral defectiveness," which has its parallel in some kinds of "psychopathic personality." The effects of encephalitis lethargica, parenchymatous

syphilis, and thyroid deficiency upon the mental state and development at different ages, or the varying results of amaurotic familial idiocy in the infantile and the delayed juvenile form, illustrate how important is the stage of growth or maturity at which damage is done.

Ætiology.—The common division is into primary and secondary, though an alternative distinction has been proposed between those who represent the lower extreme of normal variation (the "subcultural" group) and those in whom a gross structural pathology is discoverable. The primary or hereditary group is a large one, making up approximately three-quarters of all cases of mental defect. This is an estimate arrived at by independent workers, but likely to be changed as we acquire better methods of determination and subtler views of the interplay between environment and heredity. The grosser the deficiency the less important the hereditary factor, except in some rare well-defined anomalies such as amaurotic idiocy. Familial concentration of a given form of defect is specific for each clinical type. Dominant inheritance is more evident in the families of simpletons than of idiots. An incompletely dominant single factor or a combination of several genetic factors may be responsible. The mode of transmission of amaurotic familial idiocy and of phenylketonuria is recessive; that of epiloia dominant.

The environmental causes are prenatal (*e.g.* mongoloid idiocy), congenital, or infantile (*e.g.* birth injury to the brain, meningo-encephalitis, hydrocephalus, cerebral syphilis). Various poisons and deficiencies may be responsible, as in the well-known instance of cretinism, as well as certain malformations of the cerebral tissue, *e.g.* microgyria and porencephaly, and of the cranium, *e.g.* oxycephaly. Sensory defects, as in a deaf-mute, may greatly impair mental development. It is possible that some cases of schizophrenia beginning in the first few years of life are indistinguishable from mental defect and are diagnosed as such; in more general terms, it may be said that the mechanisms commonly implicated in the adult illness schizophrenia may be those chiefly affected from the beginning in some cases diagnosed as mental defect.

Pathology.—In many cases there are no significant findings; this is particularly the case with high grade defect. It is probably impossible from the histological appearances to infer the extent of hereditary or exogenous causation. Developmental anomalies, such as general hypoplasia and macrogyria may, however, be mingled with evidences of a past lesion, as in porencephaly or hemiatrophy, or with signs of a disease actually present, as in amaurotic idiocy, cerebral lucs, and tuberosc sclerosis.

Symptoms.—The customary classification is into idiots (who are too defective to be able to guard themselves against common physical dangers like falling into the fire), and imbeciles and feeble-minded persons (who need to be looked after because of their incapacity to manage their affairs or to profit by instruction). Imbeciles cannot earn their living; the feeble-minded cannot get on in an ordinary school, but may learn a good deal in a special school and be able to earn a living. The criterion is in each case mainly a social one; the same is true of "moral defect," *i.e.* mental defect coupled with strong vicious or criminal propensities. Although these terms are defined in an Act of Parliament, they are vague and of administrative rather than medical use. An attempt has been made to render

them more precise by psychometric means: the customary tests for mental age are applied, and if the subject's intelligence-quotient ($\frac{\text{mental age}}{\text{actual age}} \times \frac{100}{1}$) be less than 20, he is called an idiot; if it be between 20 and 50, an imbecile; if between 50 and 70 feeble-minded. It must, however, be recognised that though mental defect is mainly a matter of intellectual capacity, it is not solely this, and that intelligence tests, however valuable and trustworthy, cannot give a complete indication of the degree of mental defect. Even the intellectual defect may be uneven, showing much more in some tasks than in others, and it would be an error to suppose that a mentally defective person with a mental age of, say, $9\frac{1}{2}$ years is mentally in the same state as a normal child aged $9\frac{1}{2}$ years.

The *physical* symptoms are chiefly due to lesions of the central nervous system: birth trauma may have led to paralysis, spasticity, athetosis; or there may be evidence of an inflammatory condition of the brain and its membranes, as from syphilis. The whole clinical picture may be greatly coloured by the motor disturbance, *e.g.* continual rocking and twisting movements, grimaces, and abnormal posture. The special senses may be affected, as the result of an independent anomaly, *e.g.* coloboma, misshapen ears; or from a common cause, *e.g.* interstitial keratitis, the retinal changes of amaurotic idiocy. It is dubious whether the "stigmata of degeneration," such as a "Gothic" palate or a Darwinian tubercle, occur any more frequently among defectives than in the rest of the population: at all events, there are none that can be used diagnostically, except in the case of mongoloid idiocy. There are, however, some correlations between somatic anomalies and mental defect. Thus, there are more physical defects among these people than in the average population, and this becomes more evident as one looks lower in the scale of mental defect, in which skeletal and cardiovascular anomalies may fairly often be found, sometimes, but not always, due to thyroid or pituitary disorders. The mongoloid variety is described below.

The *mental* symptoms are lack of intelligence and of the normal exercise and control of primitive tendencies. This may be extreme, as in idiots, who cannot be taught to feed themselves and keep clean or who can only just recognise their companions and make their elementary needs known—they are, indeed, much less intelligent than an animal. Imbeciles are usually incapable of learning and remembering any but very simple matters. They may, however, be able to do automatically what they cannot understand or put to independent purpose: thus, "idiots savants" are especially clever at doing mental arithmetic, recalling dates and other such operations. What imbeciles manage to learn they cannot utilise in any but the most familiar circumstances. Abstract concepts are too hard for them, and their judgment is as poor as their grasp or awareness of what is relevant in any situation. Though in many ways suggestible and accessible to flattery, they may be obstinate and egotistical, and readily fall into antisocial courses, *e.g.* prostitution, vagrancy, crime. Crude sexual offences or murder may be committed as lightly as some minor deception. The personality of imbeciles varies widely: some are docile and kindly, others rough or deceitful and vindictive. It depends much on their upbringing. It has been found that in satisfactory conditions only about 8 per cent. of defectives show antisocial or

troublesome behaviour. But though the deviations of personality may not lead to delinquency, it is common to find in mentally deficient persons defects of temperament and character, as well as of intelligence, which are reflected in social inefficiency. This is most important in the feeble-minded, who have intelligence enough to learn an occupation; whether they can earn their living by it will depend on their character and the way they have been brought up.

Many persons who are high-grade defectives, when measured by formal tests, are not taken to be such because of their social adaptability, their fluency and capacity for keeping their head above water as long as economic and other stresses are light. There are instances of people classed as mentally defective during childhood, because of their backwardness in school and their low score in tests, who later in life amass money by their own efforts, or even hold a responsible position. A majority of high-grade defectives, however, live dependent and often troublesome lives; at most they do simple repetitive work. Many of them are unstable creatures, whose psychopathic personality may be sufficiently antisocial for the term "moral deficiency" to apply to them. Hysterical trends may show themselves in crude phenomena, *e.g.* convulsions, counterfeit insanity or fantastic lying; and religious and artistic pretensions may take in gullible followers and even lead to the founding of ephemeral movements.

Defectives are prone to disturbances of mood, sometimes arising out of awareness of their inferiority and its social consequences. Sudden outbursts of excitement may show similarity to manic or catatonic hyperkinetic states; they may be accompanied by a paranoid hallucinosis, mainly auditory, which clears up with startling rapidity in a day or two. In respect of these psychotic episodes, defectives are like epileptics and juvenile encephalitics, in whom a cerebral impairment has likewise occurred before the attainment of maturity. Some of the morbid phenomena, especially in idiots, are very similar to the disorders of motility seen in schizophrenics, because, it may be assumed, the same bodily mechanisms are implicated.

The *mongoloid* type of idiocy is characterised by striking physical features. Probably the outcome of intra-uterine conditions, it is most frequent in last-born children in a large family, or in children born of elderly mothers; parental syphilis may occasionally be the cause. In many cases the brain-stem and cerebellum have been disproportionately small, and other signs of maldevelopment have been reported. It is likely that hereditary factors of a recessive nature also play a part. The condition is usually present from birth; physical growth is slow, and has stopped by the time the child is fifteen. Defective growth of the skull, leading especially to abnormalities of the base and the orbit, are responsible for the peculiarities of cranial shape. The pituitary gland has been reported as showing an increase in eosinophil cells and deficiency of basophil cells. The appearance of these usually happy idiots and imbeciles is rather suggestive of a Mongol or of a fetus. The skull is small and round, and the junction of occiput and back of neck flat; an epicanthic fold across each inner canthus, narrow tilted eye-slits and lids without lashes, red cheeks, fissured and often protruding tongue, stubby depressed nose with nostrils looking forward, irregular late-appearing teeth, coarse hair on the scalp, small facial bones and occasional neurological anomalies, such as nystagmus, make the head of every mongoloid a disagreeable but ready

index to his disorder. That the disorder is a general one the rest of his body testifies; his limbs are lax and over-mobile at the joints; he has broad, clumsy feet and hands, with short fingers and a crease running straight across the palm, protuberant belly and low stature; and perhaps a congenital cardiac lesion. The similarity in a few respects to juvenile myxœdema, and the occasional concurrence of the two conditions sometimes make differential diagnosis difficult; not all of the signs here mentioned need be present in any one case. On the mental side, there is a liveliness and amiability not often seen with so much intellectual defect: the patients like music and little jokes of a primitive sort; they will imitate gestures, but seldom learn to speak properly with their rough harsh voices.

The forms of deficiency due to *thyroid insufficiency* and *cerebro-macular degeneration* are referred to elsewhere (pp. 1844 and 1624). *Epiloia* is the name given to the rare condition in which tuberosc sclerosis of the brain, adenoma sebaceum and tumours of the kidney and heart may be associated; epilepsy is common, and there are gross mental disturbances. *Gargoylism* is a rare chondrodystrophy, with hepato-splenomegaly and mental deficiency. *Phenylketonuria* may be recognised by the characteristic metabolic disturbance. It is a hereditary disorder, due to a single autosomal recessive gene.

Diagnosis.—Recognition of gross mental deficiency calls for no skill. The degree and kind of impairment, however, and the somatic variety or cause have to be worked out in every case. The latter problem—a minor one, except in the case of juvenile myxœdema and syphilis—is to be settled by careful physical examination and inquiry into the history. The former is a matter of assessing intelligence and social aptitude.

The assessment of intelligence is nowadays a matter of giving the patient tests which have been standardised on average samples of the population. What is average or normal at a given age is therefore known, and the defective child's performance can be compared with this. The most popular and serviceable tests are modifications of those put forward by Binet and Simon in 1908. As these may give a rating that depends unduly on the child's educational opportunities and facility in language, and may not indicate special abilities, *e.g.* in mechanical matters, many other tests have been worked out which supplement or, in certain cases, replace the Binet scale. A child under the age of 5 cannot be satisfactorily dealt with by the Binet tests, which moreover have only limited value for measuring the intelligence of adults. It is difficult to agree about what in a normal child must be regarded as the limiting age at which he becomes of adult intelligence; it is generally taken as 14 or 16 years. In all tests the emotional state of the subject is a factor that influences his performance. The emotional reactions to being tested must be taken, along with responses to more familiar situations, *e.g.* at home or at school, as evidence of the soundness or instability of the child's personality; by such criteria must be judged the social development of the patient, his fitness for living in the community or being put under lasting surveillance and control.

Treatment.—**PROPHYLACTIC.**—Eugenic measures are desirable for the rare hereditary conditions, like amaurotic idiocy, and in the case of those imbeciles and feeble-minded in whom genetic rather than environmental causes have been responsible for their maldevelopment and who are capable

and desirous of procreating—idiots do not procreate. Voluntary sterilisation for eugenic reasons has been recommended by a Departmental Committee, but has not yet been explicitly sanctioned by law. Birth control, therefore, is the eugenic measure to be advised in cases in which defectives seem likely to transmit their defect; unfortunately few such defectives can be relied on to observe contraceptive precautions effectively. Segregation may indirectly serve the same end. Well-managed parturition and treatment of parental syphilis are the only other practicable ways of forestalling defect.

Educational and social.—Much improvement may be attained by the training of defectives: it is work for experts. Where there are special disabilities, *e.g.* of the senses, or of such capacities as reading and writing, attention to these may lift the child out of the class of mental defectives altogether. Whether the child lives at home or in a colony or institution will depend not only on the degree of his intellectual and social deficiency, but also on the adequacy of his home circumstances. There are many kinds of provision for the care of the 300,000 defectives in England and Wales, ranging from special schools and statutory supervision to mental hospitals; 40,000 defectives are in institutions and nearly 35,000 are under statutory supervision. Well-run colonies serve to socialize many defectives hitherto vicious or violent, who can then go out and live more or less usefully in the community. Some, however, prove intractable, especially those who have epileptic fits.

Physical.—The bodily disturbances, *e.g.* contractures and paresis, call for orthopædic treatment, which sometimes indirectly benefits the mental state. The treatment of the forms due to thyroid deficiency or syphilis is described elsewhere.

AFFECTIVE DISORDER

This is of three types:

1. Manic excitement and hypomania.
2. Melancholia and mild or neurasthenic depression.
3. Agitated depression and anxiety state.

There is in each case a major and a minor form. Each is related to a more or less characteristic personality, and for each the cause of occurrence may be chiefly environmental or chiefly hereditary. Combinations are frequent (mixed forms), or there may be successive appearance of the different types, often with an interval between the attacks. A benign outcome or periodic course is the rule for the major forms, but not for the minor, which often tend to become chronic. This is partly because the environment can have more influence, whether for good or bad, on the course of the minor than of the major, more explosive and sweeping, forms. It would be a very convenient thing if endogenous cases could be sharply differentiated from psychogenic ones, as in the Kraepelinian scheme, but it cannot be done.

Ætiology.—**INTRINSIC.**—Hereditv is the most constant single cause. Research has been mainly into the major manic-depressive cases. The genetic factor is weakly dominant. It may be that more than one gene is concerned, but this is hard to tell, because the predisposition to an affective disorder may be latent in persons who have not been subjected to the stresses

that would make it manifest, and consequently the usual Mendelian figures are not obtained. The present state of knowledge is illustrated by studies on manic-depressive twins, among whom 69 per cent. of those monozygotic (*i.e.* with identical heredity) were alike affected with the disorder, while the corresponding figure was only 16 per cent. for the dizygotic pairs (*i.e.* with dissimilar heredity). In the 31 per cent. of monozygotic twin pairs who were not alike in respect of mental illness, the difference must have lain in the environment, thus showing the relative importance of external factors in causing the inherited tendency to become manifest. Although not manifest as illness, the inherited tendency may express itself in bodily and mental constitution.

The bodily habit that is found in a majority (not the overwhelming majority) of those with affective psychoses is called "pyknic." It is best seen in men after the age of 30. It is characterised by large visceral cavities (head, thorax, belly), a tendency to fat on the trunk, slender shoulder girdle and extremities, stocky build, a broad face on a short massive neck, thick receding hair and, later, baldness, venules on the cheeks, and a disposition to arthritis, gout, diabetes and especially arterio-sclerosis. As this John Bull build is so common in mentally healthy people, it cannot be regarded as a precursor of mental illness, but only as an indication that some of the constitutional and genetic causes, or biological requirements, for affective psychoses are present.

The same is true of the mental constitution or personality. Here there are several groups, shading off on the one side, by way of cyclothymia and other intermediate forms of mild disorder, into definite affective psychosis, and on the other into normal and stable personality. There are those with a pervading gloominess, pessimism and feeling of insufficiency that spoils their lives; others who are for ever anxious, keyed-up, wondering whether something has gone wrong or will go wrong, and whether it is their fault—careworn worrying creatures; while a third group is made up of the lively, enterprising, confident, sociable people, whose euphoria is patent. Irritability may be found in any of these groups, especially the second and the last. Contrasted or different features are often found mixed in the same patient. The most striking characteristic of the personality of manic-depressive patients is their ready responsiveness and lability of mood; they fluctuate with their surroundings, and in many instances pass suddenly and with small occasion from one mood into another far removed from it.

The signs of affective illness may appear in childhood, though major outbreaks of mania, depression or agitation are rare before puberty. When these occur, the phases are usually brief and the environmental influences strong. Milder forms are often regarded as normal, since night-terrors and other fears, mischievous gaiety and sulky gloom are all familiar enough in children; it is the degree, occasion and persistence of the affect which must decide whether it is morbid.

The psychological crises of puberty are only occasionally affective—chiefly self-reproachful depression or agitation—but during adolescence the illness becomes more frequent; it seldom, however, calls for mental hospital care. Each menstrual period may be accompanied by depression or restlessness, usually coming on about two days before the period. In the

third decade of life the number of cases steadily rises, and there is another peak in frequency between the ages of 45 and 55. The latter, "involutional," cases show the influence of age strikingly, so much so that they are often considered as separate disorders.

There is little to choose between the curves of age incidence for morbid depression and morbid anxiety of whatever degree; for mania the frequency is highest before the age of 30, as also for affective illnesses with a strong confusional flavour. Pregnancy is frequently accompanied by depression and agitation; psychological factors are mainly responsible. After childbirth, though there be no septicæmia, affective illness can occur, running a typical and often lengthy course.

The female climacteric is a time when anxiety usually mounts, and is accepted as an ineluctable effect of "the change." It may become definite illness, persisting even for two or three years. It is doubtful whether there is a specific connection between the endocrine causes of the menopause and so-called climacteric insanity; the melancholia then coming on is like the melancholia of five or ten years later, or the melancholia of middle aged and elderly men in whom the endocrine changes are not the same. The influence of sex as a whole is obscure. Women have this illness more than men, though the manic form is relatively more frequent in men. The reactivity is often greater and the syndrome less clear-cut in women.

There are geographical differences, sometimes thought to be racial, in the incidence, but the little that is known points to environmental rather than intrinsic causes for this. It has been suggested that affective psychoses are commonly linked up with high intellectual gifts; another says they have affinity with mental defect. The former statement has much better support than the latter, but both probably are fallacies depending on the material selected for study.

EXTRINSIC.—Physical.—Chronic toxæmia and acute infections, especially influenza and pneumonia, can be responsible for the illness. Various drugs help to heighten the anxiety to a morbid degree, *e.g.* alcohol in certain circumstances, insulin, or hyoscine. Cerebral trauma may provoke an attack. The list of physical factors could be much added to, but it must be borne in mind that wherever a distinctive, rather than incidental, physical cause can be found, the condition passes over into the category of organic psychoses. The most difficult cases in practice are those in which there is a question of cerebral arterio-sclerosis or exophthalmic goitre; the affective disorders indisputably due to these two diseases may be quite indistinguishable from others for which there is no such organic basis. The problem here is clinical rather than fundamental; since vascular, cerebral, endocrine and autonomic functions are particularly concerned in the mechanism of emotional change, certain disturbances of the physical apparatus will necessarily be accompanied by many of the psychological phenomena of these emotional changes. The depression of paralysis agitans and the anxiety of coronary disease are of the same order. The notion that coitus interruptus and other sexual practices produce anxiety is unfounded, but they may contribute to it by psychological means.

Psychical.—A recent misfortune may be the cause: of such, there is a great variety, ranging from commonplace to tragic. Any calamity to which human beings are liable may provoke an affective breakdown. Sometimes

it is induced by the insanity of a close relative. However trivial it seems to outsiders, the event that has precipitated an affective attack has been felt as a catastrophe by the patient; there are no records of great and sudden happiness causing an affective psychosis. The nearest approach to a specific connection between the precipitating happening and the type of affective illness is seen in the anxiety disorders which follow a terrifying experience such as exposure to shell fire and bombardment from the air; morbid depression following bereavement, financial setbacks or degradation is an understandable response, it is true, but to ascribe the type of response directly to the nature of the experience is specious, since on another occasion it may be with hilarious mania that the calamity is met.

Moreover, the experiences of a lifetime will have determined what calamities are most felt; they need not be calamities in other people's eyes at all. Experiences, spread over years, are the common extrinsic cause of the more chronic neurotic forms of affective illness: this applies least to chronic hypomania. In these chronic conditions the patient's own behaviour has so much to do with what happens to him, as it were, from outside that to separate extrinsic from intrinsic is very hard.

Pathology.—The *physiological* changes are characteristic only of emotional disturbance, not of morbid emotional disturbance; and therefore they are not of diagnostic value. They consist in lability of blood-pressure and pulse-rate, abnormal motility of plain muscle, especially in the alimentary tract, carbohydrate disturbances, variations in either direction of the rates of salivary and other secretions, and decreased psychogalvanic activity. The changes are variable from patient to patient and are not always discoverable. More significant are changes in basal metabolism, weight, sleep and menstruation; loss of weight is the rule during the illness. Irregularity of menses and then amenorrhœa often occur. Hypercholesterinæmia, rise in the blood iodine content, changes in the K/Ca ratio, and diminished cellular respiration have been alleged, not as yet conclusively.

The *psychological* changes, in spite of great external differences, have the following in common: the morbid phenomena are in accordance with the prevailing mood, though not wholly derivable from it; thought is less purposively directed to impersonal ends than it would normally be, but more purposively to personal ones; there is a small number of topics of pre-occupation in each patient, but his ways of arranging and embellishing them can be many; the whole body (or parts of it) often receives much of the patient's attention, because of more or, it may be, less feeling in it (hypochondria, depersonalisation); misconstructions abound, with consequent ideas of self-reference and persecution as well as misidentification; and there is a feeling of inner tension, unrest and excitement, however apathetic or care-free the patient's demeanour.

The seemingly greater quickness and capacity of manic patients has not been confirmed by psychomotor, intellectual and association tests; hypomanic patients sometimes, however, do better than in their normal state. This can be compared to the effects of increasing doses of alcohol. Patients with affective disorder are more irritable and excitable than is normal. Time appreciation may be grossly disturbed: personal time seems to pass very differently from clock time; time may seem to stand still; no future is conceivable. Perplexity may be conspicuous, and explanations of this

in terms of Gestalt psychology, conditioned reflexes, and toxæmia have been proffered.

The effects of experience in bringing about this illness cannot be explained in terms of a logical and coherent system, unless one accepts the premises of that system and infers what cannot be observed. Consequently, as there are several such psychological systems, there are several explanations. They state the conjectured ways in which instinctual energy or libido may become misdirected because of environmental conditioning, frustration and loss. It is possible to eschew such conjectures and at the same time to see the conditioning, the threats, frustration and loss that have understandably brought the patient to an excess of sadness, excitement or fear.

Symptoms.—**SYMPTOMS OF MANIA.**—There is excitability of mood and movement. The *mood* is mostly one of jollity, rather infectious, but likely to become boring or overbearing; occasionally it turns to anger and resentment. It is labile; tears will flow readily on some trivial occasion, to pass into laughter in a twinkling.

Thinking is apparently rapid. There is flight of ideas, with successive words and phrases loosely connected only by similarities of sound or chance associations. Consequently, the patient wanders from the point; whether he can come back to it depends on the severity of his condition. Jokes, self-praise, flighty comment on his surroundings, and facile optimism make up the tenor of his exuberant conversation. Nevertheless, the number of topics he touches on in the course of the day is often more limited than if he were in normal health: he reverts to a few matters over and over. He may criticise himself, with cynical bitterness or humour, as he criticises others; he may talk a lot about bodily disturbances, e.g. his varicose veins or his sore throat. His mood and expression are consonant with what he says. He is distractable, herein seeming at the mercy of his sensations and of every small detail, whether it be inside himself or, as is more common, connected with things about him. Judgment is impaired.

Delusions are less common than *distortions* and misstatements. People are wilfully called out of their names, events misrepresented, bodily sensations exaggerated, and accusations of ill-treatment or persecution irresponsibly preferred and sometimes long persisted in. The more confused and excited the patient, the more likely to be deluded and even hallucinated. Most of the seeming hallucinations are *façons de parler* or illusions; sometimes the patient is, as it were, pretending or acting the part of a hallucinated person.

Activity is exaggerated, and in severe cases incessant. Its object may change from moment to moment, but sometimes the main end is kept pertinaciously in view. The patient, if tactlessly thwarted, gets angry, sulky or violent. He feels very strong, and seems untiring. He has many schemes, of an optimistic cast, and, in the course of putting them into action, may be extravagant, inconsiderate or interfering. Sexual excesses or drunkenness may occur and bring much harm, especially when the patient is a young woman. Troubles with the police arise through silly pranks or self-confident exploits.

Sleep is brief but deep. In the early and mild stages the patient looks exceptionally well, but after weeks or months of over-activity and little sleep he looks exhausted, with sordes on his lips, hoarse voice, drawn skin and perhaps less total activity but many unfinished little movements. Food is

welcomed in the mild stages ; when the activity is great, the patient does not give himself time to eat, but plays with his food or is continually diverted to something else. Sexual desire is at first heightened but potency less.

The symptoms vary widely in degree. Mild hypomania may be an enviable time of well-directed expansive energy, unencumbered by some habitual restraints ; gross mania may be a delirious, hallucinatory condition, with incoherent talk and little free activity.

SYMPTOMS OF DEPRESSION.—In the early stages or milder forms, the patient finds concentration and recollection difficult, he has less interest and pleasure in life, he feels that this world is unreal and himself changed, he dreads effort or responsibility.

The *mood* is one of grief and misery, looking in every direction for material to feed on. The past supplies peccadilloes or graver lapses ; what is wretched in the present is dwelt on inordinately ; the future is foreseen as hopeless ruin. Anxiety is mixed with it, often in extreme degree. Weeping is less common in the extreme forms. The patient's expression usually conforms to his affect.

Thinking is more difficult. This "retardation" in thinking shows itself as incapacity to deal quickly and purposively with impersonal topics, while brooding on personal matters goes on, with a press of inner activity, a ceaseless roundabout of painful thought. The making of decisions is dodged. Conversation may become meagre, even monosyllabic, though some patients are ever ready to tell their troubles. The content of their thought is sombre—the product of ruthlessly unfair examination of their frailties and misfortunes. Some criticise themselves remorsefully or with cynical detachment ; some bewail their losses ; others abandon themselves to resigned and world-shunning despair. There are many varieties of misery, and melancholia knows them all—as many varieties as can be made from the experiences, character and imagination of a human being. Consequently they reflect the moral, economic or hygienic standards of what is good and bad that are imposed on us by modern society and our particular education.

Delusions occur in proportion to the depth of affect ; they are the extreme form of the doubts or preoccupations just mentioned. Patients often fluctuate between uncertainty and conviction about their troubles even during the same day or the same conversation. Insight may be good and judgment sound, when the affect is not overwhelming. The delusions are the product of the depression, which is primary ; they are not its occasion, though often adduced as that. Most of them concern the future as well as the past ; anxiety is prominent. Wickedness to be visited with damnation ; secular crime to be punished in this world ; loss of property that will mean starvation and beggary for one's family ; mortal or corrupting diseases—these are the common substance of delusions and are often commingled. For example, some patients blame themselves for having caught venereal disease which will expose them to the loss of their job and of their hope of salvation, exclude them from decent society, and do loathsome damage to their bodies ; no evidence, no argument shakes the erroneous belief. The delusions may be grandiose in that the patient affirms himself the chief of sinners, no one has ever been as wretched or wicked as he, he alone has done the unpardonable sin ; or they may be of a minimising sort—nobody cares about him, he is of no account, let him go into a corner to hide, people despise him. This last

belief is often understandably associated with ideas of reference or persecution—people make contemptuous gestures or remarks as he passes, they set detectives to watch him, they tell each other how bad he is. He accepts this almost always as his desert, though occasionally there may be overt resentment. Apart from this resentment, his beliefs derive understandably from his affective state. There are, however, features that betoken underecurrents at variance with the professed attitude or delusions. Thus many depressed patients, professing humility, are importunate in their demands on those around them.

Such hallucinations as occur are in keeping with the patient's affect and are of much the same nature as the delusions, though expressed more in perceptual terms. People are making derisive remarks, his body gives off foul smells, food has a different and disagreeable taste—it is often the mode of expression rather than of subjective experience that decides whether these are hallucinations or delusions. This is notably the case with bodily preoccupations, when, for example, patients report their food to be stagnating in their belly, their skin dull or fetid, their eyes impaired, their head empty. Much of this depends on depersonalisation, in which the body as a whole feels bereft of life and feeling, and emotional deprivation or emptiness is translated into bodily experience. In mild forms of depression there is no question of delusion or hallucination, and often no recognisable content to the gloom; the patient cannot say why he is sad. In the more chronic forms a settled and partly justified conviction about ill-health, present troubles, and the dark future prevails; the ideas may be obsessional and partly divorced from the prevailing affect.

Activity is limited, thus contributing to the "retardation." The more severe the depression the less does the patient do, unless the concomitant anxiety makes him restless. It is possible, however, for a patient to be depressed without "retardation." In typical cases facial expression is rather fixed and movements delayed, as though done against resistance; more or less complex activities, dressing, say, or writing a letter, take unduly long. The most extreme form is stupor or lack of all spontaneous activity; it is seldom absolute. Patients rarely become wholly indifferent to cleanliness in defecation and micturition.

Suicide is the greatest danger in depression. Whereas manic patients thoughtlessly do themselves harm or get into a fight but do not try to get hurt, depressive patients are often bent upon doing away with themselves. The risk is not proportionate to the degree of depression; many very retarded and melancholy patients make no attempt, while in depersonalised mild cases a fatal outcome is not uncommonly brought about thus. There is consequently much risk during the phase of improvement—often more risk than during the preceding severe "retardation." Deliberate self-mutilation is rare.

Sleep is bad—hard to come by, light and unrefreshing. The *appetite* is bad too: food may be constantly refused for this reason. Commonly also the patient eats too little because of feelings of fullness and other discomfort in the abdomen, or because of delusions about his bowels or his food. Mild constipation is common, but is often given much exaggerated importance by the patient. The *weight* diminishes, chiefly, but not by any means wholly, because of insufficient intake of food. Daily fluctuation in the general condition, with improvement towards evening is common. The skin may be dry

and sallow, and in some severe cases pigmented, as it is in pellagra. Menstruation may lessen or cease ; sexual desire is much less.

Here, too, there are wide variations, between the mild "neurasthenic" and the grossly deluded melancholic who craves death. There is every gradation between the two extremes, and a single case may during its course exhibit them all.

SYMPTOMS OF ANXIETY.—The *mood* ranges from uneasiness to panic-stricken terror. It may be an abiding or a recurrent state. Though chiefly turned to the future, as fear must always be, it rests on past experience, often painful and largely repressed, and it reverts to the past to account for the troubles in store. Herein, as with rationalisation and some other psychological devices, there is evident a strong desire to make things understandable in a causal nexus—a tendency to be found not only in patients but also in those who observe them. The patient's expression varies with the strength of his fear.

Thinking is troubled, the disorder showing itself in speech somewhere between frightened dumbness and the voluble talk that seems designed to cover up embarrassment and disquiet. The patient can seldom follow a train of thought for long without a limited number of preoccupations forcing themselves in. How far this interferes with daily life or set tasks depends on the amount of anxiety, as does also the impairment of judgment and insight. The content of thought is as manifold as in depression, every normal matter of human concern enters into it. Fears centring strictly on a few special topics, *e.g.* the fear of being run over in the street, may be to the fore ; the fear of insanity is particularly common.

Delusions are frequent in the grosser forms, which are most strikingly though not exclusively seen in patients of late middle life. They may say that their bowels are stopped up and their bodies about to rot ; their enemies are waiting to tear them to pieces ; their families will be tortured ; their names abhorred for ever. Hell, they are certain, awaits their souls though their bodies cannot die ; time stands still and no redemption is possible. There are many delusions less extreme than these mainly hypochondriacal and nihilistic ones ; *e.g.* beliefs that employment will be unobtainable, or that the patient will be victimised for having had such an illness. Hallucinations can occur : at the height of fear every sound and sight and smell may be misinterpreted as meaning some pain to come ; but most of this is illusional colouring of actual percepts. Depersonalisation is common with all degrees of anxiety.

Activity is much disturbed. There may be sudden attacks of panic in which the patient rushes blindly out into the open, or aimless wandering, ceaseless agitation, with movements especially at the small joints—wringing of the hands, rubbing the face, picking at sores, pulling out hair. Starting many tasks and finishing none is as characteristic of anxiety as of mania. Anxious people are distractible : their eyes follow a trivial movement—a fly walking on the window-pane—though they only comment on it when some interpretation that chimes with their mood can be fitted ; their ears are sharp for hints of alarm. During an attack of anxiety with strong somatic repercussions (*e.g.* vaso-vagal attack) activity may be completely interrupted—so-called collapse—while the patient, terror-stricken, expects his death ; alternatively he may run for air or help. Very agitated patients may

lie or sit in semi-stupor, with starting eyes and parted lips, incapable of speech unless under some strong stimulus.

Suicide is uncommon in those with episodic, highly somatic attacks of fear, and in those with chronic mild hypochondriacal anxiety, but not infrequent in the grosser forms and in those mingled with depression.

Sleep is bad: in the mild forms the patient may be afraid to fall asleep because of his horrifying dreams and the terror into which he suddenly awakes.

Sudden highly somatic episodes of anxiety, vaso-vagal attacks, are common: the patient feels his heart palpitating, his bowels turning over within him, he sweats, his limbs tremble, his mouth is dry, he feels he will fall or collapse or die; he turns pale, his pulse-rate changes, usually becoming more rapid, his blood-pressure rises, he may want to open his bowels or pass his urine. When anxiety is long-standing and severe, such attacks are rare. It is possible for parts of this general affective disturbance to be isolated, and to occur with little conscious anxiety, as in muco-membranous colitis, effort-syndrome, aerophagy, neurotic indigestion, enuresis, impotence, ejaculatio præcox, psychogenic asthma, hyperidrosis. The factors determining such special emphasis on one or other system are partly physical (some organic defect or innate functional anomaly) and partly psychological; in depression a comparable problem would be weeping or constipation. In anxiety thyroid enlargement can occur; weight falls off; menstruation is irregular or ceases; the deep reflexes are very active.

Diagnosis.—Typical cases are easy to recognise. The common errors of diagnosis lie in: (1) Missing organic disease (*e.g.* general paralysis, cerebral arterio-sclerosis); or the converse (*e.g.* mistaking the more expansive manic patient for a general paralytic). (2) Forgetting how mixed the symptoms of mania, melancholia, and anxiety may be, so giving rise to atypical pictures that may be mistaken for schizophrenia, if too superficial an examination or too static and rigid a diagnostic criterion be used. (3) Forgetting the influences of age, general personality, and milieu on the content of a patient's mind, *e.g.* his having lived among spiritualists may lead to deceptively fantastic statements. (4) Expecting to be able to diagnose solely on presenting symptoms, without regard to previous history and constitution; the reverse is also to be avoided. (5) Expecting diagnosis always to lie between distinct entities which could not possibly be mixed together in the same person, as though hysteria were incompatible with affective psychosis, or both of these with schizophrenia; in fact, they often are mingled. This is not to make light of diagnosis, which gives the psychiatrist much knowledge that he cannot gain from study of the individual case before him.

Nothing in the mental state of a patient with affective disorder enables one to exclude an organic basis such as general paralysis or cerebral arterio-sclerosis. This decision must turn on the physical findings. The problem becomes simpler when signs of dementia supervene. (See p. 1825.)

From schizophrenia, diagnosis depends on a picture of the whole illness, on the presence of characteristic thought-disorder, incongruity of affect and bizarreness of behaviour, as well as on the previous personality and constitution, rather than on any positive features of affective psychosis; the remoteness and unconvincing manner of the schizophrenic, so hard to describe but

almost conclusive when recognised, may help. Later, when complaints have become empty and repetitive to the point of stereotypy, and catatonic symptoms mix with the anxiety, diagnosis is easier. As between schizophrenic and manic excitement, the setting in which the excitement occurs is almost more important than the *prima facie* symptoms. In young people schizophrenic features may often be found without their being of much significance; in the elderly what seem to be catatonic features may rest on an organic cerebral basis. The more easily one can get in touch with the patient, enter into his mood and understand what he says and does, the more is it an affective, not a schizophrenic disorder. The range of benign affective phenomena is wider than a textbook description can convey.

There is no need to try to diagnose affective psychosis from psychogenic depression, cyclothymia, anxiety neurosis, neurasthenia, or involutional melancholia; these are only subdivisions of it, in which the age, reactivity, severity, or chronicity of the condition is being stressed. Periodicity is sometimes made the hallmark of affective psychosis; this historically interesting point of view is hard to apply, because so many patients have only one definite attack in their lifetime, and because periodicity can be striking in other conditions, such as obsessional disorder and schizophrenia.

From obsessional disorder the diagnosis may be difficult when there is localised anxiety or depression with sharp content and good insight; so closely alike are the conditions, that eminent authorities would include obsessional disorder also in the manic-depressive group, thus disposing of the diagnostic problem. It is best, however, to keep them distinct, and to discover in a particular case whether the characteristic subjective rejection of the obsession occurred at its first appearance; often the anxious or depressive patient at the beginning has welcomed the thought which accords with his affect, though later he struggles against it and may disclaim it. Genuine obsessions, however, are common in affective psychoses.

Course and Prognosis.—The varieties of outcome and sequence are many. They depend on the balance between particular intrinsic and extrinsic causal factors in each case, and on the extrinsic factors which are brought to bear on it in the form of treatment. The more typical the illness, the surer the recovery in favourable circumstances.

A history of definite affective psychosis in a parent or grandparent points to recovery from the attack, but it is unsafe to infer the course of the illness from hereditary data alone. A well-adapted personality and a pyknic build, a history of similar illness followed by complete recovery, a fairly sharp and fairly recent onset, and precipitation by external troubles which will not be likely to continue are all of them points to the good. Advancing years make the prognosis poorer, but a first attack of involutional melancholia, if there be no vascular disease, eventually clears up in two-thirds of the cases. Bodily symptoms are often the best indication of coming recovery. Improved appetite and regularity of the bowels, cessation of anxiety attacks, clearing of the complexion, increase of weight and return of menstruation may be noted, even before any increase of activity and long before any admission of feeling better can be got from the patient.

A first attack of excitement or anxiety will seldom be the only one; of depression it may. Periodic depression and anxiety is less likely to cease in middle life than periodic excitement. The occurrence of hallucinations or

delusions is in itself of little consequence prognostically. A transition from anxiety to depression or mania, and from mania to depression, or *vice versa*, is commonly gradual. Only in predominantly reactive attacks can one surmise how long the illness will last, or when another attack is to be feared. After recovery complete insight into what happened during the illness may not be attained, especially by resentful manic patients, melancholics who are sensitive and suspicious, and agitated patients who feared personal harm.

Generalised somatic disturbances, *e.g.* loss of weight, especially if acute and brief, are of good prognostic import, other things being equal. The more the somatic preoccupations or symptoms are diffused over a period of time and localised to one system, the poorer the prognosis; this, however, does not apply so much to children as to adults. Hypochondria and depersonalisation suggest a long illness, as do nihilistic delusions (*e.g.* denying that one's bowels are opened at all), and, to a far less extent, admixture of hysterical or schizophrenic features. The more the psychogenic causes have been obviously operative for a long period, the greater the tendency to chronicity. In the more chronic forms or after a series of attacks, there may be impaired initiative and judgment, irresoluteness, dullness, and social deterioration—none of them conspicuous. Puerperal and pregnancy psychoses have a good outlook. The milder forms of anxiety and depression, if not already chronic, respond well to treatment, especially to psychotherapy.

Death may occur from suicide, insufficient food, and intercurrent disease, especially pneumonia. Sometimes it is inexplicable on such grounds; a wasting disease, reminiscent of pellagra, carries them off.

Treatment.—**PROPHYLACTIC.**—Genetic prophylaxis is occasionally possible, as when two persons with definite affective disorders marry each other and are advised not to have any children. Rules of thumb do not apply in this matter; it is wrong to tell a patient he should marry or not marry, procreate or not, unless one has been able to weigh the dubieties of our genetic knowledge, the pedigree of the patient and all his transmissible qualities with an informed and cautious judgment.

Individual prophylaxis is not practicable until after symptoms have appeared which bring the patient to the doctor; social prophylaxis, mental hygiene, and child guidance have not yet been proved to have permanent value in staving off or mitigating affective illness except in the matter of depressive suicide. In so far as one finds environmental factors (*e.g.* heavy responsibility, unemployment, or sexual frustrations) important in inducing an attack, advice on these matters may be helpful; it may be practicable by psychological and social treatment during the healthy interval to do much good in this way. But some cases, in which intrinsic factors seem all powerful, are proof against such measures, and in any case it is not easy to persuade the patient when he is well again to put himself for a long time in the doctor's hands.

TREATMENT OF THE ACTUAL ILLNESS.—It is convenient to consider separately the acute major forms, and the minor more chronic ones.

For the former, treatment is directed to safeguarding life, relieving distress, and providing the best conditions for the emotional disturbance to subside; the situation is like that in tuberculosis or typhoid fever. Exhortations to "pull yourself together" are as out of place as advice to take a

voyage or an argument about the delusions. If the attack is sufficiently severe to unfit the patient for ordinary duties, treatment at home is probably inadvisable. Although in such attacks all argument is futile and active psychotherapy harmful, yet the loss of relation between current experience and emotion is never absolute; there is virtue in separating the patient from real trouble and distressing associations, reassuring him, giving him firm, kind management. The essential combination of these, and especially the last, is rarely obtainable at home. The patients, however boisterous or suicidal, usually recognise their need of treatment and are willing to enter hospital voluntarily. They should not transact any business if it can be helped; their judgment may be too much disturbed, they lay up trouble for themselves. Continuous narcosis sometimes seems to curtail an attack; ephedrin and acetylcholine have also been reported as doing so. The former treatment, *i.e.* narcosis, demands experience and care, no treatment cuts short an attack regularly or dramatically enough to justify any set conclusion about its efficiency in this regard.

Prolonged baths—for 8 or 10 hours daily at a constant temperature of 96° to 98° F.—have much value in allaying restlessness, whether of the manic or the anxious kind, especially the former. They have the further merit of diminishing angry contact with other people, permitting fairly free movement and lessening dirtiness, besides promoting sleep.

Drugs are indispensable. The fear of habit formation should not prevent hypnotics being given when there is persistent insomnia. Barbiturates, phenobarbitone or paraldehyde often suffice: it is well to ring the changes, to prescribe the barbiturate in divided doses, and in each case to diminish the dose without the patient's knowledge. For severe anxiety bromides, opium (*e.g.* as papaveretum) and hyoscine may be helpful; the risks of the two latter are obvious. As to bromides, the risk is intoxication, which makes the patient worse; estimates of the bromide content in the blood and clinical scrutiny should prevent this. Continuous narcosis is valuable, and in some cases convulsant therapy cuts short an attack. Both these methods must be used with caution and reserve, because of the risks, which may be disproportionate to the advantages. Food must be given in adequate quantity and kind. Artificial feeding, preferably by nasal tube, may be necessary because otherwise the patient would die of starvation. The presence of acetone in the urine and a falling weight curve are strong indications that nutrition must be attended to promptly. A good nurse may sometimes, by unusual patience and sense, get over an obstinate refusal to take enough food and drink, but often nothing prevails against it. Apart from hydrotherapy, rest in bed, fresh air, attention to the bowels, and other measures of general hygiene are desirable.

Suicide is of the first importance. Prevention of it can be better ensured by close knowledge of the patient and his day-to-day condition than by mechanical precautions, but if he is bent upon it, these may be unavoidable. It is possible to make them unobtrusive without nullifying them. Certain it is that excessive use of bolts and bars can defeat its own ends, and excessive supervision aggravate a patient's misery, his fears, or his resentment. Two good rules are: (1) to discredit the maxim that those who talk of suicide never commit it, and (2) to remember that most suicides are surprises. Convalescence from melancholia is a risky time.

Occupational therapy is good, as soon as the patient can be got to co-operate ; but it is not rational treatment to pester a melancholic, to encourage the fretful restlessness of the agitated, or to give the manic patient more things to muddle himself with and destroy. Still, it is often surprising to find how soon, under tactful guidance, these patients will enter into ordered activity of a more or less simple sort, and how helpful it can be to them. During the stage of improvement the same is true of recreations and social activities. Patients should not leave hospital till recovery is assured, unless it is obvious that the hospital surroundings and the absence from home and work are an actual cause of their persistent anxiety or dejection.

To revert to the *milder* forms, which tend more to become chronic. Here manipulation of the conditions in which the patient lives at home and at work may be conjoined with psychological treatment, both depending on an appraisal of the causes of his illness. There is nothing distinctive (though much that takes account of the individual patient's needs) in the psychotherapy and social treatment called for (see pp. 1815-1818) ; danger signals must be recognised as they occur. Zeal must give way to the real needs and resources of the patient, which are often not appropriate to a drastic or very lengthy treatment. Simple measures of inquiry, explanation and reassurance, together with small environmental changes, may have much effect. A fixed regime imposed in detail by the doctor is helpful ; this becomes more and more necessary as the affect dwindles in long-standing cases. Hypomania does not usually respond to causal treatment of any kind ; it seems to run a largely autonomous course. Anxiety may yield very satisfactorily to patient psychotherapy.

SCHIZOPHRENIA

Definition.—The forms of illness under this name are so diverse that many efforts have been made to distribute them, so far in vain. What is common to them all is a detachment from the world without, and a breaking up of normal psychological connections within. The personality is not integrated as in normal people ; thinking, emotion, and conduct are discrepant and morbid, yet there is no impairment of formal intelligence such as is found, for example, in organic dementia. The obsolescent name “*dementia præcox*” is not a synonym for schizophrenia, but a reminder of its recent history. At the end of the last century a large number of patients in mental hospitals were found to have begun their illness before they were 30, and to have passed ultimately into a deteriorated state that looked like dementia ; their illness was closely studied, delimited, and called “*dementia præcox*.” When the same clinical picture, however, came to be found in cases that had not such an outcome or onset, the latter criteria were waived in favour of a descriptive analysis of the actual symptoms, and along with this larger conception came the new word “*schizophrenia*,” which betokened a more psychological approach, and a more elastic and generous notion of what might be included. Theories of causation, psychopathology, and clinical boundaries are implicit in any view of what “*schizophrenia*” really is ; consequently, it is still possible for two experts to disagree about what should properly be included under this name, yet over the diagnosis and prognosis of any particular patient they will attain a measure of agreement and

certainly surprising to those who know the condition only from reading or limited experience.

Ætiology.—**INTRINSIC.**—The intrinsic factors are very important. Studies of the incidence in twins and in the members of a family demonstrate a hereditary factor in a majority of cases. If one of a monozygotic pair of twins be schizophrenic, the other is also in 70 per cent. of cases. The frequency of the illness among various relatives of patients indicates that it is not transmitted as a simple dominant, nor indeed as a simple recessive; probably more than one recessive gene is responsible. It has been suggested that people of definitely schizoid personality are heterozygotes for the genes concerned, actual schizophrenia requiring that the genes be present in homozygotic form; this is as yet conjectural, though some investigators find about as many schizoid psychopaths as schizophrenics among the brothers and sisters of schizophrenic patients.

The constitutional features that betoken an innate predisposition to this illness are more of the psychological than the physical kind. The bodily attributes have been said to be an "asthenic" (weedy and lank), "athletic," or "dysplastic" build; but, since these are found in much the same proportion among healthy people as among schizophrenics, there is little to be said for them here. It is, however, certain that "pyknic" build (see p. 1844) is uncommon among schizophrenics. More significant, however, are the features of personality, commonly called "schizoid"; they are to be found in a large number of cases, though not by any means in all. The patient is reported to have shown slight peculiarities from his earliest years; he has been quiet, shy, and solitary, a "model child," given more to day-dreaming or abstract speculation than to ordinary interests and activity; sometimes he has been unduly submissive and sentimentally affectionate, or touchy, suspicious, obstinate, and resentful of advice and control. A single "typical" schizoid personality is a myth. It is, moreover, to be stressed that a "frozen" description of the schizoid varieties of personality does not do justice to the true state of affairs: characteristic deviations from the conventional norm of behaviour can always be understood better if the patient's way of dealing with his circumstances is viewed historically as a biography of individual tendencies and experiences, rather than described as a bundle of traits. By paying heed to the development of faulty as well as healthy habits of response, the psychiatrist can often see the march of events that led up to the patient's illness, and escape too artificial a sundering of inherent tendencies from the external happenings by which these tendencies have been evoked and given shape and substance.

EXTRINSIC.—The illness sometimes breaks out after childbirth or an acute infection. None of the efforts made to inculcate some specific infection have succeeded, nor does intoxication in general seem to play any considerable part in the causation of schizophrenia. The same is true of cerebral trauma. There are, however, many instances of a chronic schizophrenia supervening on an intoxication, and of schizophrenic symptoms, especially of the catatonic sort, appearing in the course of an organic disorder, such as G.P.I. or encephalitis lethargica. In these, the same structural and functional systems must be supposed to have suffered impairment as in the "endogenous" forms of schizophrenia, and it has been particularly urged that in the chronic paranoid conditions that may follow an acute alcoholic psychosis, it is really

a matter of schizophrenia that happens to be associated with alcoholism, if not partly activated or released by it. It is further to be remembered that at least one intoxication, namely, with mescaline, produces a mental disturbance that is in some respects similar to schizophrenia, and that any chronic hallucinosis comes in time to look very like a long-established schizophrenia, because the possibilities for abnormality of any human mind are few, the deprivation symptoms almost uniform, and our methods of clinical examination imperfect. Endocrine disorders, especially of the gonads, have been held responsible, but satisfactory evidence is lacking, except in a few cases which can scarcely be representative.

Recent mental stress may sometimes be the starting-point of an attack, but in a considerable proportion of these cases the reported overwork, disappointment in love, or other painful experience, is found to have been a product of the already existing illness, or the last of a long series of disturbing events. No recent experience is ever sufficient to account for the illness without regard to intrinsic causes. Nor is any remote experience either. No matter how searchingly the patient's life be resurrected and analysed, it is scarcely ever possible to discover that anything happened to him which would have led to his adopting a schizophrenic way of shunning daily life unless he had been somehow disposed to it from the beginning; although, of course, much may have happened to him that has strengthened and fostered the disposition.

Among contributory factors, age and sex are noteworthy. An onset after the age of 40 is uncommon. In three-quarters of the cases that later exhibit the characteristic chronic syndrome, the illness begins between 15 and 25. The condition may become overt in children before puberty. Men are more often affected than women—in the proportion of 113 to 100, according to the largest available statistic, the matter is dubious, however, because of the different standards of diagnosis used.

Pathology.—**PHYSICAL.**—Histological changes in the brain are not characteristic; it is doubtful if they are even frequent. A cellular loss in the third and fifth layers of the cortex, with lipoid accumulation, has been found, but it occurs in many other conditions. Many claims about cerebral pathology, and the chemical and physiological changes in schizophrenia have now been discredited, so that all findings in this difficult field have come to be matters of suspicion. Variations in the same individual may be wide. Recent investigations have purported to show:—a disturbance at the acid-base equilibrium towards the acid side, with a diminished excitability of the respiratory centre to carbon dioxide; lowered rate of oxygen consumption; polyuria; diminished gastro-intestinal motility; poor response to epinephrin; abnormal heat-regulation; decrease or sluggishness of total blood-volume; and slowing of the arm-to-carotid circulation time. These findings have not so far been controverted; they represent disorders of metabolism and regulation which may be partly a concomitant of the characteristic mental disorder, and partly an effect of it, *i.e.* they may be essential physical disturbances in the illness, or may be secondary to the abnormal, often inert life the patients have led since they became ill. There is no ground for supposing them causal.

Some inferences have been drawn from the similarity of catatonia to the extrapyramidal syndrome that can be produced in animals by bulbo-

caprine; the argument from analogy cannot be pushed further than to say that certain functional systems are available in the brain, which are sometimes involved in schizophrenia, as they also may be in poisoning or in encephalitis lethargica, G.P.I. and other diseases.

Very significant are the well-attested metabolic findings in the rather rare cases of cyclical catatonia. In these the nitrogen balance varies periodically; with alternating phases of retention and over-excretion, corresponding to the mental change from excitement to stupor or *vice versa*. By means of thyroxin a thorough emptying of the patient's nitrogen store can be brought about and subsequent nitrogen retention prevented, thus leading to clinical improvement. The correlation between metabolic happenings and clinical condition in these patients is now established.

PSYCHOLOGICAL.—The large and inconclusive literature on the psychopathology of schizophrenia is of five main kinds, namely:

- (1) Minute description of the phenomena observed, and abstraction from them of general principles of disordered function.
- (2) Experimental study, chiefly quantitative (e.g. psycho-galvanic).
- (3) Studies of artificial hallucinatory psychoses (e.g. mescaline intoxication) and parallel experiences.
- (4) Comparative study of animals, children, poets, primitive people, etc.
- (5) Intuitive or speculative interpretation.

It will be obvious that these methods overlap and that they differ widely in acceptability and usefulness. The findings of almost all can sound plausible, when stated in general terms; discrepant or abstruse, when stated in detail. Their exposition touches on the most intricate problems of normal and morbid psychology, and therefore is highly technical and unsuitable here. A working hypothesis for clinical purposes is that in schizophrenia there are inherent faulty habits of reaction, whose severity and persistence depends largely on education and other external circumstances. These faulty reactions are characterised by a deficiency in the function of synthesis, so that there is an inco-ordination, "intrapyschic ataxia," as it were, a splitting up of the mental life, which justifies the name "Schizophrenia." Thereby the whole psychic life of the patient, cognitive, emotional, and conative, is changed in a way that is alien to normal understanding. We can observe the change but cannot enter into it or describe it adequately in terms of our own experience, as we mostly can depression, manic excitement, hysteria, or obsessions. It shows itself also as a turning away from the contacts and realities of daily life, a preference for what the mind can supply from its own stores, however morbidly, rather than for the current experience that the outer world affords.

Symptoms.—Schizophrenia may be regarded for clinical purposes as a form of maladaptation in which there are certain characteristic defects of inner harmony and consistency in behaviour, thought, and emotion. These are rarely seen in childhood, but from puberty onwards they may appear in varied combinations (often in persons who for years have been introspective and unsocial). There is discrepancy between mood and utterance, disturbance of conduct (briefly summed up as catatonic or hebephrenic), self-absorption and incapacity for sustained thinking along normal lines. A guarded or artificial demeanour may conceal these essential features, whereas they may be conspicuous in a florid or "deteriorated" case. Hallucinations

and delusions may fill out the picture ; affective or other morbid types of reaction may complicate it.

The onset is not always abrupt. There is often a long history of preliminary symptoms in which it is arbitrary to decide where personality has merged into illness. Complaints of headache, weakness, anxiety attacks, loss of appetite, and dysmenorrhœa may have accompanied slight oddities of behaviour, such as rudeness or apparent absence of mind and indecision. The patient may have felt an alarming change in himself, in his capacity to think and feel normally, and been notably depressed and anxious. Ideas of persecution or of exaltation may occasionally escape him, or he may have become stilted in his talk and shown other affectations and mannerisms. The more gradual the onset—and in many cases it has spread over many years—the more unlikely is it that it will have been recognised as morbid.

The commonest or basic symptoms are: (1) Disorder of thinking. (2) Emotional incongruity. (3) Hallucinations. (4) Disturbed impulses or conduct. From these can be derived most of the other symptoms, such as delusions, feelings of influence, autism, catatonic phenomena, anomalies of speech, negativism, and the rest.

The *disorder of thinking* is a characteristic and central feature. The patient cannot command the whole range of an act of consecutive thought ; he misses the point, fastens on details and brings in irrelevant associations which are correct in themselves, but which divert him from the main end of his original process of thought ; consequently his thinking is incoherent, rambling and jumbled. He brings together the most far-fetched topics, so that the connections are sometimes so superficial as to be empty of meaning, and at another time profoundly influenced by symbolism and highly individual values. The usual logical sequences are ignored : cause and effect are interchanged ; temporal, spatial, verbal, and accidental relationships are unduly turned from abstract to concrete, treated as grounds of identity, played with or flouted. Things linked only by analogy and chance association are taken to be the same. The condensation of several conceptions in one, or transference of a set of attributes to some inappropriate object, may become a matter of course, so that only the closest knowledge of the patient and his surroundings will enable the psychiatrist to follow his meaning. It is not necessary, however, that such extreme incoherence be evident in the patient's talk ; he may not show any at all when speaking, or may suddenly obtrude a startling lapse from normal ways of thought which he then ignores, justifies or explains away. Inconsistent thoughts can be present together in a way impossible for normal people ; and the same object or notion can appear to him in several interchangeable guises, each of which would normally exclude the others. The patient himself is often aware of his disordered thinking, and may describe it : he feels his thoughts are suddenly taken out of his mind, other thoughts, foreign to him, are put into his head, his mind is not his own, his thinking is suddenly interrupted, some external power controls it.

The thought-disorder is illustrated by the following characteristic remarks of patients : " There were bats and bees coming through the window ; of course that was because my brother-in-law kept teasing me. He said I had bees in my bonnet." " If I should return during my absence, keep me here until I come back." " I have a lot of forced thoughts. My thoughts

are all drawn-out words, they ought to be pin-pricks. There is an unnatural stoppage in my thoughts, too. . . . I have heard voices say 'He is conscious of his life.' . . . To get my feeling back to normal I feel like changing motor-cars into battleships, to be superior to them."

This disorder may only be demonstrable when the patient gets on the topic of his delusions; in other matters he may seem quite sensible. It is not essentially different from what normal people experience during states of altered consciousness, *e.g.* in dreams, or when falling asleep; the schizophrenic, however, has it with clear consciousness, so that a listener often feels that the patient is making fun of him in giving such transparently absurd answers with an air of knowing exactly what he is about. Some chronic well-preserved schizophrenics make their living as comedians, the audience much enjoying the allusive, half-comprehended nonsense, with its background of innuendo and symbolism. Autism, *i.e.* immersion in his own fantasies and preoccupations, may account for much of the oddity and detachment the patient shows; it accounts also in part for his "negativism," in that he resents any stimulus that interferes with his day-dreams.

Delusions arise mainly out of the thought-disorder. They are often bizarre; they may occur to the patient with a suddenness of conviction that puts them beyond all argument; and they are egocentric in that they commonly bring indifferent happenings or people into a special relationship with the patient—*e.g.* he suddenly knows that when his cousin yesterday said he had been reading about Napoleon's divorce of Josephine, it was a subtle way of telling the patient that his wife was committing adultery with this cousin, whose name is Joseph. The delusional ideas may not be firm conviction, but fleeting notions, readily given up, and based upon some casual instance of the thought-disorder; sometimes they are schizophrenic ways of saying something commonplace—*e.g.* the patient declares his wife has poisoned him, but when he is further questioned says airily that he means she gives him ill-cooked food which is bad for his digestion.

Fixed delusions are, however, common, and are usually of a paranoid complexion; they may develop out of more or less ephemeral ideas of reference. They are often intermixed with hallucinations. The patient gets into a state of mind in which he feels there is meaning in everything, something is going on behind the scenes, he is perplexed by all this, and mystified, it has to do with him in some uncanny way. Presently, he begins to "see through it all," sometimes he gives it some religious or cosmic significance, especially if he has much anxiety as well—the Last Judgment is at hand, he is to be responsible for the regeneration of the whole world. The delusions are not always enacted on so grand a stage; there may be homely fancies about neighbours who whisper and sneer, or about some vulgar bogey like the Jesuits or the Jews or the C.I.D. Often, the patient complains that people work on his mind, hypnotise him, influence him for his own good, set about to drive him mad or ruin him. Delusions of grandeur may be linked up with these paranoid ones (*e.g.* he is being persecuted because he is the Messiah), and may be likewise pedestrian or lofty, according to the patient's previous education and interests, the severity of his disorder, the copiousness of his fancy, and the amount of normal mental function still in evidence. Here, as elsewhere in psychiatry, the symptoms are a mixed outcome of impaired or perverted function on the one hand, and of normal

function on the other, the latter either reacting to and modifying the disorder, or obtaining freer play through it. If, for example, a patient feels his thoughts being controlled by some external influence, and he has queer tinglings in his body, his conviction that he is being hypnotised, and that some one is playing an electrical instrument on to him, must be regarded as a normal attempt to find the cause of an almost inexplicable happening. The delusions are sometimes about past events, which are falsified retrospectively, *e.g.* the patient relates details of his having been a changeling or a predestined hero. Delusions about bodily transformation or disease are frequent, and may be complicated and bizarre.

Patients often do not act in accordance with their delusional beliefs, especially when these are fleeting or chronic; they may, for example, be friendly towards a nurse whom they believe to be persecuting them cruelly. But this is, on the whole, unusual in the early or acute stages of the illness: a patient will then act on his beliefs violently or in terror; he may go to the police or be driven to suicide.

Constantly the matter of a patient's delusions will be found to be intimately dependent on his experiences, his emotional attachments and sufferings, his struggles and frustrations; it is impossible, however, by any such analysis and derivation of his delusions to account for the fact of their occurrence, *i.e.* for the patient's choice of this way of dealing with the experiences in question. The same is true of the general thought-disorder: *e.g.* interruption or "blocking" of the train of thought may take place only when some emotionally weighted topic, some complex, is touched on. This accounts for the place where "blocking" occurs, but not for the "blocking" itself; that, like the other fundamental disturbances of function in schizophrenia, eludes a wholly psychological explanation.

Intellectual defect does not occur. There is usually no clouding of consciousness. Intellectual laziness or evasion is often conspicuous; the patient may repeat questions in a musing way, or profess ignorance. Orientation and memory are not, as a rule, diffusely impaired, though hallucinations, delusions, and lack of interest may interfere with them, and consciousness may be disturbed in stupor or excitement. Many a patient who has long borne the appearance of gross dementia will suddenly show that his intelligence is still a sharp instrument: drugs, *e.g.* sodium amytal or insulin, and intercurrent disease or shock can thus dramatically reveal how little ground there is for calling this illness a dementia. Schizophrenics often do the unexpected. Amnesias, and deliria, when they occur in schizophrenia, may be hysterical; obsessional and hysterical symptoms, like anxiety and depression, are compatible with schizophrenia, and are often an intimate component of the illness.

The speech and writing of the schizophrenic betray the extent of his thought-disorder. Stiffness, pedantry, fantastic euphuisms, words of his own coining, queer symbols and grammar, stereotyped repetition, and infantile twists like speaking of himself always in the third person may be conspicuous features of the patient's use of language. There may, of course, on the other hand, be little or nothing outwardly amiss in his conversation and writings. In florid or chronic cases the patient may talk in an unnatural voice, or without any modulation. Writing may be set forth as though it were painting, and the converse: in subject and matter the patient's insanity

may be patent, but his treatment of his matter, however odd, is seldom odder than some forms of modern art, and it cannot, therefore, be called typical of the illness. These anomalies of symbolical representation are as open to psychological explanation as are the delusions mentioned above; the neologisms, for example, can be analysed up to a point; and these phenomena have enriched our knowledge of the psychopathology of schizophrenia.

The *emotional incongruity* is the chief, but not the only, sign of disturbed affect. Often the patient himself notices in the beginning of his illness that he is less moved by habitual affection, or even feels hatred towards a parent he has loved. The strongest and rarest of human passions are not infrequent in this illness: ecstasy, mystic communion, despair, horror, agony of death, limitless abandon, apotheosis, salvation, are approximate names for these exceptional states that are probably indescribable in the current language of normal people. Apart from these, and much the commonest of the affective changes, is apparent emotional shallowness: the patient receives moving news without any sign of being touched by it, or his response is perfunctory; he smiles or looks bored when talking of a recent tragedy in his own family. This shallowness and incongruity of affect is, however, not to be taken at face value. What the patient says, and what he means with his words, may be very different; so may what we say be very different from the meaning the patient attaches to it. It is unsafe to assume that the patient's words have reference to what is mainly going on in his mind at the moment, or that his outward expression is a trustworthy index of his emotional state. Violent emotional outbursts—of anxiety, rage, love, misery—can certainly occur in a patient who has lately seemed empty of all affect. The schizophrenic patient is undoubtedly different from normal people in his emotions, but not in so negative a way as his seeming apathy and lack of affective rapport would suggest. His attitude towards the same person may change quickly, in accordance with conflicting or opposite tendencies in himself; this ambivalence is often understandable in the light of his earlier history. Sometimes the illness leads to a blunting of ordinary reserve, a lack of reticence, or a levelling down of the gravest matters, so that frivolous or cynical indifference and imperturbability are signs of the patient's morbid condition.

Hallucinations are not so frequent as superficial examination of patients might suggest; many of the patient's assertions about queer sights and sounds are not the expression of vivid perceptions but of passing fantasies, imagined more plastically than is normal; this is particularly true of many of the so-called visual hallucinations, or of cases where the unreal perceptions occur in several senses together. Hallucinations are nevertheless extremely common and persistent in schizophrenia: auditory ones occur most often, diffuse somatic ones not infrequently, those of smell, taste and sight more rarely.

The "voices" are sometimes so closely linked with the thought-disorder that it is difficult to tell whether the patient is relating what he has heard or what he has thought. He may show the intermediate stages between the two, declaring that people repeat his thoughts or that everything that passes through his mind is spoken aloud inside his head; his actions are described publicly, he cannot go to the lavatory without shameless comments. What the voices say may be abuse or encouragement, trivial repetition or threats and commands; this content can usually be accounted for by the psychiatrist,

when he knows the patient and his history well. The voices may come from strange places, *e.g.* from inside the patient's own chest or abdomen, and are then often accompanied by curious somatic hallucinations, indicative of morbid attitudes, both physiogenic and psychogenic, towards parts of the body. The latter often occur independently. Queer sexual feelings, or distortions and impossible growth of various organs, may be reported. They are usually bound up, as any schizophrenic symptom is likely to be, with delusional and emotional components, which are partly derived from the patient's experiences and psychological development. The visual disturbance, like the gustatory, is more often illusional than hallucinatory, *e.g.* people's faces look fiendish or artificial or transfigured.

The *actions and bearing* of the patient are often characteristic. Abruptness or lack of grace in movement may be seen early; it can be indistinguishable from the fidgety self-conscious hobbledelay stage of adolescence. The patient may pull faces at himself in the mirror, or may be unaware of his grimaces. Asymmetrical movements of expression, twitchings, mannerisms, queer rituals and tic-like gestures are to be met with. The meaning of the patient's movements can usually be worked out, but after they have been present for long their sharpness is rubbed off, as it were, and the empty stereotyped movement at last gives little clue to what was once a significant emblem of experience and feeling. The movements often seem to become automatic, like the "verbigeration" of empty phrases in the patient's speech. Negativism, talking and acting beside the point, and bizarre escapades may be seen at any stage of the illness.

There may be a suspension of movement, or the reverse: akinesia or hyperkinesia. Both may occur in the same patient, who may lie for weeks or months in a catatonic stupor, from which he suddenly emerges into swift action. He may carry out some impulsive action and then promptly return to bed and stupor; or he may become wildly excited and imperil his life by his blind and raving activity. During catatonic stupor, patients may adopt strange postures, *e.g.* holding their head off the pillow all day, pursing their lips. They may be indifferent to cleanliness about faces and urine, or actively dirty in this regard. Waxy flexibility is rare, but many patients are automatically obedient so that they keep up an imposed posture.

The variety of schizophrenic anomalies of conduct is too great to be described here. They must not be assessed absolutely, but always in relation to the setting in which they occur. Then they have meaning in the individual case, and are not merely so many examples of "ambivalence," or "mutism," or "negativism." It is, however, true in this matter also that understanding the content of an anomaly does not make its occurrence likewise understandable. Much of the schizophrenic's conduct is so close to certain disorders of movement in organic disease of the central nervous system, that somatic mechanisms may be assumed to have suffered damage in this condition. There are three main things to be done with any schizophrenic symptom: (1) to search out its psychological origins, and its meaning for the patient in his present situation; (2) to link it up with the other functional disorders that he shows; and (3) to consider its background of physical structure and function. It is not always practicable to attempt all three, nor is it as yet possible to do them well, but none can be ignored without detriment to a full analysis.

Often the most significant yet intangible effect of the illness is upon the patient's *personality*. After florid symptoms have died away, or when there are no definite symptoms at all, a change in the patient's ways is remarked by his intimates. Not only is he outwardly different—more “peculiar,” less understandable and predictable, rather shut-in upon himself, remote, with queer values and impulses—but in many cases he is also aware of this change, and may complain of an inner perversion of himself, a loss of that unity which we take for granted when we say “I,” or “me.” Insight in schizophrenia, in this respect and more generally too, may be penetrating and just, as many self-descriptions attest. There may also be varying degrees of impairment up to gross lack of insight.

None of the *bodily symptoms* are characteristic of this illness, though many occur. Besides the somatic complaints and preoccupations already mentioned, patients, especially if young, show vegetative anomalies. Thus, vasomotor disturbance may take the form of cold bluish extremities, exanthems or oedema. Seborrhoea is common. Abnormal growth of hair occasionally occurs in women. Loss of weight in the acute stages, and fatness in the chronic condition, interruption or irregularity of menstruation, and fluctuations of temperature may also be observed, especially in catatonic cases; of the schizophrenic states, stupor is the richest in demonstrable bodily changes. Fleeting neurological signs, *e.g.* pupillary anomalies, may be found. In states of acute excitement attacks of unconsciousness may occur, but epileptic seizures are very rare.

VARIETIES.—There are three main forms—catatonic (with acute outbursts); hebephrenic and simple (early onset, chronic course); paranoid (fairly late onset, delusional). They are not exclusive categories, and it is usually profitless to try and apportion a doubtful case to one or the other. They do not correlate closely enough with outcome to be of much use clinically.

In *hebephrenia*, the least common variety, delusions and hallucinations are inconsiderable, but abnormal conduct is to the fore: the patient may be silly and mischievous, abruptly eccentric or inert and without initiative. The illness may progress without acute episodes (*dementia simplex*), or be interrupted by phases of excitement or obvious insanity, which subside, leaving the patient worse than before. In *catatonia*, the most favourable variety, the symptoms are plain even to the layman: akinetic or hyperkinetic states may appear and subside quickly, sometimes for good or for several years. There are usually, also, characteristic disorders of thought and emotion, which may clear up when the stupor or the excitement does. In the *paranoid* form, generally rather late and insidious in its development, but less damaging to the personality than the hebephrenic, partial systematisation of the delusions is common in the earlier stages, but may be later swallowed up in the general thought disorder and deterioration (*dementia paranoides*). The more bizarre the delusions, the more likely is affective emptiness to replace gradually the initial resentment and distress, but sometimes the patient passes into a chronic paranoid state, obviously schizophrenic to the psychiatrist, but compatible with ordinary life outside an hospital. Hallucinations and luxuriant delusions may, however, be conspicuous in the paranoid form (*paraphrenia* and *dementia phantastica*).

Diagnosis.—The chronic and advanced cases—“typical *dementia præcox*”—that abound in mental hospitals, are easy to diagnose, but

early or inconspicuous cases often extremely difficult. The chief positive points to look for are : characteristic thought-disorder, a qualitative change of affect, and other evidence of " intrapsychic ataxia," as well as feelings of being under external influence. Catatonic symptoms are of limited diagnostic value, because of their frequency in organic and symptomatic psychoses. More important than any single feature is the impression of the case as a whole, the development away from normal interest and response to the real world, and the establishment, instead, of " autistic " self-satisfactions so that the patient's personality is twisted awry, as it were, and withdrawn from easy contacts.

From organic syndromes—syphilis of the central nervous system, alcoholic psychoses, disease of the cerebral vessels, encephalitis lethargica, etc.—the differentiation turns on the physical findings, more than on the mental state : a schizophrenic syndrome may appear in an organic condition, because the brain, as Kraepelin said, is like an organ whose stops give out the same sound, whoever works them. Often it is not a matter of deciding whether the syndrome is organic or schizophrenic, but whether, being schizophrenic, it has a discoverable somatic basis or not. Alcoholic delusional states are an instance of the complicated relationship that may be found (see p. 1838). If, after consciousness has become clear again, the other phenomena of toxic confusional psychosis persist, then schizophrenia is the more probable diagnosis.

Diagnosis of schizophrenia from an affective syndrome is difficult, because both are often combined in the same patient. Some of the significant points have already been referred to (see p. 1859). Catatonic excitement differs from mania in that the speech and acts of the latter are intelligible as expressing a general affect and are conformable to the situation in some measure ; the onset and cessation are not so abrupt as in catatonic excitement ; and there are usually characteristic features which make the distinction easy. Melancholia becomes suspect when delusions are repeated without the appropriate affect, and there is a readiness to project responsibility for the illness, to complain of external influence. The inertia of the depressive is not so complete as that of catatonic stupor, nor so likely to be abruptly broken through. States of severe agitation are not always easy to distinguish from schizophrenic excitement, but a more frequent problem is that of deciding whether some bodily fear or conviction of disease is schizophrenic or not. Whether in regard to a preoccupation or a delusion, the chief point to consider is the appropriateness of the affect to the alleged hypochondriacal notion ; the more bizarre the bodily change described, the more likely to be schizophrenic. Depersonalisation is sometimes at the bottom of these somatic complaints ; what is significant is not the depersonalisation, but the way it is elaborated and regarded by the patient.

Hysteria can offer great difficulties, largely because hysterical mechanisms are so often operative in schizophrenia. Plain motor or sensory disturbances commonly give less trouble than hysterical dissociation, stupor, and pseudo-dementia. The previous history, the relationship of the outburst to a particular set of happenings, the behaviour in the intervals, the demands upon the attention or response of bystanders must be taken into account. The mistakes and oddities of the hysterical pseudo-dement may be theatrical, in accordance with his ignorant notion of what insanity is like ; the deliriously dissociated

hysteric does not identify correctly the people around him, as the schizophrenic usually does, even when in a dream-like state; the hysteric who is acting some imagined scene does so without discrepancies or gross interpolations, whereas the schizophrenic is seldom so consecutive and persistent. The degree to which the patient is being influenced by his immediate surroundings is, however, the chief guide, apart from definite schizophrenic features.

Obsessional states offer difficulty when the patient is in doubt as to whether his alien thought or impulse comes from within his own mind or is imposed upon him. If he shows indifference as to the occurrence and content of the compulsive ideas, it is suggestive of schizophrenia; but careful examination of the development of the symptom, and the patient's attitude towards it, permits a clear diagnosis in most cases. Complicated rituals, odd obsessions and chronicity make an obsessional illness look very like schizophrenia; as does intoxication of an obsessional patient by bromides. Obsessions may develop into schizophrenic symptoms (see p. 1888).

Prognosis.—Schizophrenia is always a serious condition. Though some recover, the tendency of this morbid change is to do permanent damage to psychic life. In the individual case, however, pessimism is not justified. It is certainly never possible in the early stages of the illness to be certain that recovery is out of the question.

Heredity is a poor guide to the prognosis, except in the rare cases in which an identical twin of the patient has for some years had a schizophrenic illness, or in which one parent is schizophrenic, and the other has schizophrenic relatives; even then it is difficult to prognosticate with assurance regarding the present attack. If one parent has had an affective illness the prospects of recovery are brighter, but this can better be assessed from the patient's own bodily and mental constitution. If he is of pyknic build, the outlook is much better. Similarly, the patient who has for years tended more and more to withdraw from his surroundings, to be careless of social requirements, to lie late and live alone, given up to day-dreaming and eccentricity—such a one should he become overtly schizophrenic, has a poorer chance of doing well than the active, suspicious and impulsive man, or the self-conscious, introspective worrier who similarly falls ill. A narrow and rigid previous personality makes deterioration more likely than if there had been wide interests and possibilities of adaptation.

The more abrupt and stormy the onset, the better the outlook. This is one of the most reliable guides. When the onset has followed upon a recent painful experience, and the content of the patient's talk and his behaviour refer to this, or when a physical damage appears to have provoked the symptoms (e.g. influenza or head-injury), the outlook is rather better than when the provoking factors are obscure; but this is by no means always the case. If the attack occurs during puberty or adolescence, prognosis must be cautious, because of the difficulty of distinguishing between the transient upsets of this period of adjustment, and the progressive schizophrenia that may then show itself plainly. The earlier history is of great help.

The nature of the symptoms is not a safe guide. Very severe departures from normality may clear up, yet an outwardly mild condition be of grave omen. Symptoms such as stereotypies of movement and speech, which indicate that the illness has been going on a long time, and that there is a

general narrowing and fixity, are grave; as are also hebephrenia, and a long-drawn-out stupor, with negativism, impulsive violence and vasomotor changes. The more manic or depressive features, the better. Previous attacks, with an interval of normality between them, are prognostically favourable. If the patient first falls ill after 30, he will scarcely go downhill in the tragic way young people sometimes do. He may develop fixed delusions, which are often rigid and encapsulated, so to speak, and therefore he may be able to return to ordinary life, with reservations; or it may be that his morbid beliefs absorb all his mental powers, and compel institutional life. The more the psychiatrist can discover healthy modes of response in the illness itself, as well as in the previous personality, the happier the outlook. Many patients, after an attack, do not return to work, but have narrower interests, and less spontaneity than before; they are more easily tired, and may be hypochondriac, or show other symptoms thought to be "neurotic." Such patients have sometimes made a poorer recovery than others who return to work and can meet most social demands, though careful inquiry reveals definitely schizophrenic sequelæ in their thinking and emotions.

The simplest rule is that an abrupt onset of the illness, an adequate cause for its occurrence, and a well-adapted non-schizoid personality are the criteria of good prognosis. Sensible early treatment may avert disaster.

Treatment.—**PROPHYLACTIC.**—This, whether eugenic or individual, is limited and uncertain. Even if effective, it can reach only a minority at present, and its effectiveness is a matter of faith. Probably child guidance and other measures of mental hygiene do good in averting potential schizophrenia, but no one can be sure of this. Such treatment aims at diverting the child into social activities and keeping him out of situations in which he will be mortified or otherwise troubled emotionally. However wordily or abstrusely the prophylactic treatment be described, it is essentially a matter of trying to make an unusual child into an average one, or making his surroundings unusual to suit him.

TREATMENT OF THE ACTUAL ILLNESS.—There is no one treatment of the disorder that has manifest superiority over any other. Painstaking attempts at readjustment of the patient's outlook and behaviour by means of psychotherapy (not psychoanalysis), occupation, games, etc., are the most systematic and rational way of making a permanent change for the better. The co-operation of the patient is here necessary, and also the help of a social worker desirable, who may do much to modify and arrange the patient's circumstances in the interests of his mental health, *e.g.* getting him suitable occupation, and schooling his relatives in a sensible attitude towards him. Such treatment is not practicable for those acutely ill, but for the mild, the convalescent, or the imperfectly recovered case it is of great value. By means of it many patients can be discharged from hospital before they have settled into apathy, or become unresponsive to the claims of the external world; it is better not to keep a schizophrenic patient in hospital waiting for complete recovery, but to get him back into ordinary life as soon as possible, provided conditions there are not too adverse for him, or he too abnormal to cope with them.

Treatment in a psychiatric clinic or mental hospital is usually necessary at some stage of the disorder, and must be decided chiefly by the severity

and social risks at the time. For the large number who become permanently in need of institutional care, much of the deterioration formerly customary may be averted by the energetic use of occupational therapy and recreation which make the patient's life less sterile.

There are few conceivable ways of altering a human being that have not been tried in this illness. Many of them have been those believed to be efficacious in other illnesses; some have been intended to shock the patient somehow. Of the former may be mentioned endocrine preparations (in large doses), transplantation of gonads, removal of supposed septic foci, induction of fever by malaria, etc., injection of human serum, manganese salts, production of aseptic meningitis and continuous narcosis. Of the latter, *i.e.* shock-methods, many of the procedures of a bygone time are examples; the whirling chair, precipitation from a height, immersion in ice-cold water, and so forth. The most recent methods which entail a profound and alarming disturbance are those which use insulin or a convulsant (see p. 1819). Though the direct emotional effects of such treatment are not negligible, metabolic changes are no doubt chiefly responsible for the clinical effects seen. Insulin has more value than the convulsant method. It should be employed only in hospitals fully equipped and staffed for the purpose. The treatment is most effective in cases which would have a good prognosis for the attack if treated by other methods. It is not a panacea for schizophrenia, but may shorten an attack in suitably chosen cases.

The details of treatment, whether in hospital or at home, must be individual; even in such matters as the allaying of excitement no uniform procedure, *e.g.* continuous baths, or narcosis, can be a routine measure. When excitement is extreme, disturbances in water metabolism and loss of salts may be combated by giving 5 per cent. saline intravenously, 300 c.c. every other day, alternating with forced fluids. During stupor, general measures for ensuring adequate food (in some cases feeding by tube), cleanliness and evacuation of urine and faeces must always receive attention. It has been found that various chemical agents, such as carbon dioxide inhaled in a 30 per cent. mixture with oxygen, and sodium amytal, will temporarily interrupt a catatonic stupor; this finding accords with the chemical metabolic changes reported in the condition, but its therapeutic value is slight.

PARANOIA AND ALLIED STATES

The words "paranoia" and "paranoid" are used loosely by many. Kraepelin gave paranoia its modern meaning, describing it as the endogenous, insidious development of a permanent and unshakable delusional system, with complete preservation of clarity and order in thought, will and action. If the illness cleared up, if it showed symptoms of an organic, affective or schizophrenic syndrome, or if it was provoked by external happenings, it could not be paranoia. Thus delimited, the condition is exceptionally rare; so rare, indeed, that there is no use in having such a category. Moreover, cases that Kraepelin himself called paranoia have since become obviously schizophrenic. There is now no profit in thinking of paranoia, or paranoid states either, as syndromes in their own right, so to speak, and of the same order as schizophrenia or affective disorders. They are on the

same subsidiary level as stupor, hypochondriasis, anxiety and depersonalisation. When met with, they must be distributed according to the accompanying symptoms and the general trend of the illness; and their prognosis and treatment must be assessed accordingly.

Besides the paranoid beliefs and attitude referred to in previous sections, there are a number of instances of this unhealthy relationship between the patient and his surroundings, which are mild in their outward form, easily understandable in the light of the patient's history, and fairly responsive to treatment. Sensitive and shy people are often troubled by doubts and shame as to their physical or moral worth; and, by projection, attribute to others the dislike or contempt they do not acknowledge in themselves. This occurs in youths who masturbate, and suppose others to remark it, and in old maids who believe men to be pursuing them; but there are many varieties of shame and desire, besides the sexual, which lead to such ideas of reference or persecution. The development of paranoid reactions of this sort is usually plain. So is that of the querulous, resentful type of reaction, *e.g.* in the man who believes himself done out of his rights and who becomes a persistent litigant or writer of memorials. Before judging such a man psychopathic, the extent of the injustice he has suffered must be compared with the degree of his resentment and his relevant conduct. Commonly the injustice is found to be fanciful or trifling, and the man's sense of grievance immoderate, so that he comes to believe there is a veritable conspiracy to wrong him, and devotes most of his time to useless appeals or threats. He may persuade his wife or his children of the justice of his complaints, inducing delusions in them, *i.e.* *folie à deux*, etc. Many such patients, however, never become deluded: they are contentious about their wrongs, and waste years, perhaps, in proclaiming them or seeking redress, but they are well aware how other people regard them, and what has actually happened. Many claimants of compensation, "grouzers," "old soldiers" and unstable adherents of more or less cranky movements, are to be placed here. There is no sharp dividing-line between these psychopathic people, and the more or less normal, often socially precious, leaven who detest injustice and are willing to do much to defeat it. Some deaf people become paranoid, misinterpreting what they cannot hear plainly, and construing it into a jeer or an insult.

HYSTERIA

In hysteria, symptoms of illness are represented by the patient for the sake of some advantage, without his being fully conscious of this motive. The form of representation will vary widely according to the circumstances that have provoked the illness, the patient's experience of what the symptoms are that he is trying to represent, and his somatic resources. These factors, presently to be discussed, bear on the hysterical symptoms that simulate physical disease. But it is impossible to restrict hysteria to this physical form. The illness that is represented by the hysteric may be a mental one; moreover, it is not possible to consider hysteria without regard to the mechanisms of its occurrence which manifest themselves in the personality and are mainly psychological. Hysteria is the most psychogenic of all illnesses. Its recognition is therefore a double problem: (1) exclusion of

what may be called "genuine" illness, *i.e.* of a recognised morbid pattern; and (2) discovery of an adequate motivation. To ignore either of these requirements is to court error, since hysteria may occur along with physical or mental disorder, elaborating upon it and mimicking it, and, on the other hand, some physical diseases give rise to symptoms indistinguishable in their form and apparent psychological mechanism from those of hysteria.

Ætiology.—A hereditary factor is probable in many cases. Thus, a group of hysterics who were pathological liars were compared with the average population in respect of the proportion of their brothers and sisters who were in mental hospitals: it was five times as many; and of the parents of the group, a sixth were psychopathic. From these and similar figures it is not possible to tell the mode of transmission or the nature of what is transmitted, but only to infer a hereditary factor. The occurrence of hysterical mechanisms in children, and their frequency in healthy adults, especially after calamities or in unendurable conditions, such as may occur in war, suggest, however, that hysteria is potentially present in most people and that environment is more important here than heredity. The combination of heredity and environment may result, long before actual illness occurs, in a *hysterical personality*. This is not found in all patients who show hysterical symptoms, but nearly all people of hysterical personality show hysterical symptoms. Many of the features of this personality are socially obnoxious, but other features are not, and it is wrong to use "hysterical" as a depreciatory epithet for a set of qualities that one dislikes. These people are unduly responsive to the situation they are in, especially if by their excessive response they can fulfil wishes of which they are hardly aware, or evade what is painful in the situation, instead of meeting it and disposing of it adequately. Unsatisfied with their own capacities, they seek to cut a better figure than their endowment warrants, and are constantly posing and pretending. This, like all their behaviour and aims here described, is not done with full consciousness, but with a more or less sincere ignorance or ambiguity of purpose; it is not a question of deliberate deceit, of studied histrionics or malingering. In thus responding to situations and turning the response to some inadequate end, the hysterical person is characterised by a lack of inner stability and of constant standards of behaviour, and also by a lability of affect and an exuberant fancy. The fantasies normal in childhood are here seen in physically mature adults, who, like children, can temporarily live their fantasies, absorbed in this unreal compound of past experiences and longings, yet not so wholly divorced from their real surroundings as might appear. In an attenuated form, this is evident when they almost unwittingly manufacture some situation, according to their needs—literally "making a scene"—and enter into it emotionally with a rapidity and fervour impossible for more stable people. Egotism and untruthfulness (*pseudologia phantastica*) may be pushed to the point of delinquency. There may be a longing for prestige, sympathy, love, or some other emotional relationship, which leads the hysteric to behave in a way strikingly out of keeping with his demeanour on other occasions; the inappropriateness of his behaviour even at the time may be obvious to a detached onlooker, but is not always so. Many of these people can use illness or well-acted fantasies of illness to satisfy their hardly conscious needs; they may also gain their ends by forgetting what it would be painful

to remember. Here again the onlooker may find it hard to tell how genuine or complete is this forgetfulness, but the question is of little moment compared with discovery of the motive for the hypomnesia. The much-stressed suggestibility of hysterics is a notable aspect of their responsiveness to situations and of their especial responsiveness to a person with whom they develop an emotional relationship, often unrecognised by themselves as such. The emotional attitude of a hysteric towards other people is often influenced by sexual factors. Hysterical personality is believed to be much commoner in women than in men, and may be associated with psychosexual immaturity. Coquetry and frigidity are not uncommonly allied in hysterics; there may be much flirting and sexual excitation, but not actual coitus; it is, however, juster to say that the sexual lives of hysterics show instability and inadequacy than to specify any particular aberration.

Hysterical personality can be recognised before puberty; in younger children, however, it must be extreme to be recognised, because of the great frequency of hysterical mechanisms then (*e.g.* behaving as though fantasies were real, counterfeiting illness, somnambulism). Some of the grossest instances of hysterical behaviour have been recorded in girls not yet adolescent, cf. the Salem witches. Much of the work of Child Guidance Clinics is taken up with the treatment of hysterical tendencies, not perhaps taking the form of definite symptoms but plainly evident in the child's personality.

The precipitating factor for the onset of hysterical symptoms is usually a situation, emotionally charged, out of which the patient's symptoms will bring him more or less overt, but unacknowledged, gain. This gain need not be material and obvious, and may run directly counter to such accepted values as health and ability to work. One of the plainest instances of a partial unsubstantial gain is that created by an accident, and the resulting claim for compensation; hysterical symptoms flourish in such a soil, and are usually influenced for the worse by repeated medical examinations. Hysteria occurs among soldiers under active service conditions, and can readily be fostered in them by injudicious measures. It is not infrequently a sequel of the acute panic or stupor which may in some men be the effect of intensive bombardment and exhaustion during active warfare.

Pathology.—This is almost wholly a matter of psychopathology. It is true that disseminated sclerosis and many other organic diseases of the brain may be accompanied by hysterical symptoms, but the association is not a constant one. The psychological changes can usually be traced further back than the happening that provoked the illness; often they are the continuation of normal tendencies of childhood that have been fostered and extended by ill-judged upbringing. The hysterical symptoms that appear as motor or sensory phenomena show the patient's readiness for the translation of experience into bodily symbols; this is a special instance of the universal tendency for somatic representation of experience, converting it into action. It is the facility and exaggeration, not the existence, of this "conversion" mechanism that is characteristic of hysteria. What is thus translated or "converted" into physical terms has been something painful and unacceptable; the partial exclusion of it from consciousness, "repression" of it, is therefore understandable; in its physical, symbolic form it is tolerable and may even be prized. Identification with other people is responsible for the frequent imitation of symptoms and for the epidemics of

hysteria. Suggestibility, with its characteristically quick formation of habits of somatic response, is another way of describing the phenomenon. Clearly the mechanism need not be limited to the production of physical symptoms, though bodily structure and local weaknesses may conduce to this. There can be hysterical phenomena, such as the dissociation seen in fugues and so-called splitting of personality, which are instances of the exclusion of recent and remote painful experience from clear consciousness. The wishes and fears that deviously attain outward expression as hysterical symptoms do not derive solely from the recent past, though much of their strength may come from it. It must be admitted that there are some hysterics in whom this psychopathology cannot be demonstrated, and that such cases are among the most intractable.

Symptoms.—These may be divided into: (1) sensory; (2) motor including fits; and (3) quasi-psychotic.

The symptoms can be like those of any conceivable affection of which the patient has a notion. The cruder his notion, the less will his symptoms be like those of the simulated condition, but after he has been demonstrated to a class or repeatedly examined he may better his notion, and consequently his symptoms come closer to those of organic disease. Or, if he has had opportunity of seeing insanity, his pseudo-insanity may smack less of the stage than it otherwise usually does. The range of hysterical symptoms is so great that to describe them all in detail would take inordinate space, and there is no need to do so.

The *sensory* or, more properly, the *perceptual* symptoms include clonus and globus hystericus, blindness, deafness, and anæsthesia. The two former are so common in all sorts of mental disorder, especially those accompanied by anxiety, that they are of little specific importance in hysteria; inquiry as to their presence will often in these suggestible, rather hypochondriacal patients lead to their occurrence. The difficulty in swallowing reported by hysterical women may be associated with a strong disinclination to eat—*anorexia nervosa*; it should not be confused with depressive *anorexia* or that of pituitary *cachexia*. Any cutaneous disturbance of sensation that the patient has a notion of can be presented, *e.g.*, anæsthesia, either mono- or bi-lateral, or of stocking and glove distribution, and analgesia of any part. The anæsthesia seldom corresponds to any nerve trunk, nerve root, or spinal segment, unless the patient has had special opportunities of knowing. With an anæsthetic hand objects may be identified, and any test which the patient does not recognise as referring to this disability he will perform satisfactorily. Such tests are not a means of “catching the patient out” as though he were a malingerer, but of ascertaining whether the symptoms express only his notion of some illness. The tests for a malingerer, it is true, amount to the same thing, though one assumes the malingerer to be clearly conscious of his purpose; consequently any distinction between hysteria and malingering must depend on the observer's impression as to the patient's honesty and self-knowledge; certainly it cannot be decided by tests. The tests for blindness (*e.g.* using a stereoscope with a supposedly blind eye), deafness (*e.g.* effect on pulse, respiration and psychogalvanic reflex of exciting remarks addressed to the patient), and for other forms of perceptual defect all depend on the physician's greater knowledge of what should or should not accompany the symptoms of which the patient complains; they are not intended to dis-

cover hysterical "stigmata" or characteristic anomalies. The ovarian and other hyperæsthetic spots, the pharyngeal anæsthesia and the concentric limitation of the field of vision formerly used diagnostically, were all products of suggestion or, as in the last instance, phenomena that may occur in normal fatigue, in hypochondria and in certain cerebral lesions.

The *motor* symptoms are paralyses, pareses, spasms, contractures, and tremors. Hysterical paralysis or paresis never affects individual muscles, but always movements. By various devices it can be shown that the patient can still use the affected muscles, as long as he does not know that the movement in question requires their use. The paralyses affect chiefly the left side of the body, are common in the legs (preventing proper walking or standing), and often occur in limbs or other structures that have earlier been the seat of an organic disability, *e.g.* trauma or paresis. If the paralysis be flaccid, no loss of tone or of reflex response is found, and the patient, through his ill-informed notions of what should happen, behaves otherwise than a patient with organic paralysis would—*e.g.* if asked to rise from the supine to the sitting posture without using his hands, he keeps his paralysed leg flat on the bed. If the paretic part be kept stiff, the antagonists will be found to come into action first when the patient is asked to perform the movement he says he cannot; and if the movement has to be made against resistance, sudden removal of the resistance reveals how much of the apparently tremendous effort was going into associated irrelevant or antagonistic movements. Passive movement to overcome the spasticity or subsequent contractures cause the patient to be more upset than could be accounted for by any pain he may complain of. The varieties of abnormal gait are numerous; many of them fantastically elaborate and, from the look of them, exhausting. Not only the musculature of the limbs may be affected but of the trunk (leading to curvatures and odd postures) and indeed any voluntary muscles, *e.g.* of the tongue, larynx, pharynx or eye. In hysterical aphonia the voice may sink to a whisper, or there may, more rarely, be complete mutism; the voice can, however, be used normally for coughing and similar purposes. The aphonia often comes on after some local inflammation that has caused hoarseness, or after a fright. Stammering, usually of the exaggerated kind, may also occur. Spasm of the external ocular muscles, leading to a convergent squint, may accompany a spasm of accommodation. Ptosis and blepharospasm sometimes occur. Many of the tics and spasms that used to be thought hysterical are now recognised to be often physiogenic, *e.g.* residual symptoms of encephalitis lethargica and chorea; spasmodic torticollis, for instance, is far less often psychogenic than used to be thought. When a spasm or paresis has long been maintained, trophic disturbance may follow: blueness and cedema, shiny skin, fibrosis of periarticular structures, and similar effects of rigidity and disuse. Tremor is most often seen in patients with a spastic paralysis, but may occur independently, as in many of the war cases. It is variable in degree and rhythm, and usually disappears when the patient's attention is turned from it; this does not apply, however, to some very long-standing cases.

Hysterical *fits* commonly occur in patients with obviously hysterical personality. They may be little more than a fainting-attack or an outburst of temper, significantly like the tantrums of an ill-behaved child. Often, however, they are more differentiated than this, and diagnosis from an epilep-

tic fit may be difficult. Sometimes the fit grows out of a tremor induced by fright or anxiety, as in many war-hysterics, or it may express some emotional state, such as great pain, anger or erotic excitement. Occasionally the patient shows plainly by her expression and movements that the fit is erotic; it may be a typical orgasm. The "classical" four-phase fits which Charcot described were artefacts of the clinic; they do not happen now.

Sometimes the patient's fit becomes very like an epileptic one after he has spent some time at a neurological clinic, or he may be an epileptic who also has hysterical fits. Some hysterics, by overbreathing, induce an epileptiform convulsion, which can be abruptly terminated if an injection of calcium chloride or gluconate be given. They may pass from one such fit into another, so that the condition suggests a status epilepticus. The unconsciousness that often appears to accompany a hysterical fit is seldom as complete as it looks; neither is the subsequent amnesia. There may, however, be a delirium, corresponding to the emotional upheaval. Patients very rarely hurt themselves seriously in the fit, however violent, or have a fit when alone or asleep. The length of the attack and its degree often depend on the audience; the more the bystanders try to restrain the movements, the wilder do the kicking, struggling, biting, shouting, panting, spitting, etc., become and the longer they go on. There is neither the pallor nor the cyanosis, the regular sequence nor the subsequent headache and sleepiness of epilepsy; urine is not passed nor the bowels opened; reflexes, including the corneal response, are unaffected, and the end of the fit may be abrupt.

The *quasi-psychotic* symptoms are stupor, twilight-state, pseudo-dementia, and fugue. In the stupor, seen typically in harassed weary soldiers under bombardment, the patient lies motionless, taking food like a twelve-months baby, non-resistive, sometimes incontinent of urine or faeces, and without any predominant emotional tone. It is of brief duration if the exciting circumstances cease to prevail. In less acute forms there may be a sullen resistive akinesia, or a condition lasting even for years, with an occasional break; this is a rare form. The confusional or delirious states may accompany a fit or represent an important emotional experience, *e.g.* some sexual episode. They are often histrionic, and represent wishes of a religious or grandiose sort; or the patient may behave as though he were an animal or a child. Sometimes they occur during the night, and in a somnambulist state the patient repeats some past happening, or may do complicated work. This is closely akin to the hysterical fugue, in which there is not so much a clouding as a narrowing of consciousness, a "dissociation." In the fugue the patient says he has forgotten some or all of his life before a certain date, and later he may profess to remember nothing of what has happened during the fugue. There is, in short, a double set of memories, which may alternate, and since the patient's own identity is commonly included in the repressed and temporarily forgotten material, he may be said to have two personalities, and sometimes three or four. Actually there are no cases in which it is strictly correct to speak thus of multiple personalities; it is only a matter of different aspects or fragments of the one personality. In the fugue the patient may live out some fantasy or—as more commonly happens—simply says that he does not know who he is or where he lives. Nearly always a hysterical fugue with gross amnesia turns out to have been a means of evading some predicament, and it is well to keep in mind in such cases that the patient may have

broken the law or otherwise exposed himself to disgrace and punishment. The amnesia is seldom as complete as the patient states. Fugues may occur as a hysterical mechanism in an organic psychosis ; we have seen a man with arrested general paralysis who had been prominently and in detail reported as a case of multiple personality which responded to psychotherapy.

" Pseudo-dementia " covers the large group who behave as though insane. It may occur, as in the so-called Ganser syndrome, in prisoners awaiting trial. Whatever the circumstances, its motive is escape from a disagreeable situation. It is likely, however, that it is mainly those with a predisposition towards severe mental illness, especially schizophrenia, and the high-grade defectives who have recourse to this kind of hysterical behaviour. It sometimes comes on after brain injury. The patients' behaviour corresponds necessarily to their notion of insanity, which is usually far enough removed from anything the psychiatrist knows as such. Occasionally, however, it is very near the buffoon-like conduct of some schizophrenics. The patients say that they do not know their own age, affect not to understand simple remarks, give absurd answers which nevertheless indicate that they know the right answer (*e.g.* by inverting the correct order of the figures in a sum). When asked about some simple matter, they look as though they were making terrific efforts to remember (herein behaving differently from the schizophrenic). The most characteristic thing is the disparity between the patient's alleged deficiencies and his general alertness : he says he does not know anything about his own past, he cannot read or spell or do the simplest arithmetic, and yet he may be behaving quite naturally and adapting himself to the situation in a way which would be inconceivable if he had actually so advanced a dementia.

Some hysterics go to great lengths in their representation of ideas of illness. They will allow themselves to be put among grossly insane people, or submit to repeated operations, such as amputation. Self-inflicted injuries, *e.g.* keeping wounds and sores open, are not uncommon (*cf.* *dermatitis artefacta*). In some such cases masochistic tendencies can be recognised, but by no means in all. Suicidal attempts are not infrequent. They often have as their purpose revenge, the satisfaction of some spite, and the patient may leave behind a lying, fantasy-coloured letter, indicting someone. Frequently the suicidal attempt is in the nature of a theatrical demonstration, done in such circumstances as make it unlikely to be fatal ; and if the patient kill herself, it is more through bad management than intention.

Diagnosis.—It will be plain from what has been said that diagnosis must be both negative and positive—negative, by excluding any organic cause for the symptoms ; positive, by finding motives and relating the symptoms to them. Neither method is alone sufficient, because of the occasional concurrence of structural disease with psychogenic symptoms. As to the former, *i.e.* the negative method, it is unnecessary to enter here into all the differentiating points. Many of them have been mentioned in the foregoing description of symptoms, and all turn on the disparity between what experience tells us would occur if these symptoms were of organic origin, and what the patient knows about such matters. Consequently a doctor who has hysterical symptoms is extraordinarily difficult to diagnose, in this negative sense. The method of arriving at a diagnosis by suddenly taking the patient unawares, and seeing if his symptoms persist, is to be deprecated ; it antagonises him. Likewise undesirable is the procedure of see-

ing whether one can suggest new symptoms to the patient, *e.g.* an *anæsthesia* ; it can be both misleading and harmful. Neither is the hysterical nature of a symptom to be judged solely by whether it can be removed by suggestion ; for some organic symptoms are temporarily got rid of thereby, and many hysterical symptoms are not. An intimate knowledge of the range of symptoms of physical disease is much more useful to the physician than an equipment with special tests and lists of differences between "functional" and "organic." It is not only a problem of neurology but of the whole of medicine, since the hypochondriacal tendencies of many hysterics lead them to complain of visceral symptoms ; usually, in doubtful cases, the symptoms are those which might well occur in the earlier stages of some physical disease. It is, however, in neurology that the most difficult cases of all arise, *e.g.* in disseminated sclerosis, carbon monoxide poisoning, cerebral vascular disease or encephalitis lethargica ; here there is more likelihood of the organic disease being overlooked than of its being wrongly diagnosed. The patient's previous personality, any provocative situation or emotional disturbance, the previous occurrence of organic signs, *e.g.* transient diplopia, and the age of the patient must be considered. Hysterical symptoms appearing for the first time in middle or later life in someone whose personality has been stable, are probably not solely psychogenic. If the symptoms diminish when little or no attention is paid to them, they are more likely to be hysterical.

Course and Prognosis.—This depends mainly on the patient's personality and social setting, and on the treatment employed. A long history of hysterical traits prior to the illness, a continuance of circumstances favourable to the symptoms, and inadequate or excessive treatment are all unfavourable. This is, however, an illness that sometimes confounds prediction, patients recovering when many adverse factors have been operative and the symptoms have been present for years. In children the prognosis is fairly good if treatment can be undertaken promptly ; it is best if the hysteria is monosymptomatic and has come on after a fright. In all cases in which the situation which provoked the illness persists, the outlook is bad ; for example, in the compensation cases, for which no medical treatment is of any avail—for obvious reasons—until the litigation is settled once for all. Similarly, during war, psychotherapeutic successes are often dazzling while the hysterical soldier is under treatment in hospital, but the symptoms come again when he must return to duty. There are many varieties of outcome, chronic invalidism being the commonest. A few patients later become schizophrenic, and a few become involutional melancholics. The prognosis in respect of the patient's hysterical personality is more important than that of his hysterical illness ; it is, however, no more to be assessed by rules than the general future of any human being's life and personality. Patients do not necessarily tend to become anti-social ; delinquency is certainly a likelihood in some hysterical people, but bravery and self-devotion may be conspicuous in others.

Treatment.—Too much treatment is worse than too little. Injudicious physical or psychological treatment of hysterics often makes their symptoms worse and their illness intractable. Recondite methods should be eschewed by all but experts. Common sense is as important as psychological understanding ; and social usefulness more to be aimed at than removal of symptoms or attainment of self-knowledge. In short, it is not the hysterical illness or

the mechanism of repression and conversion that calls for remedy, but the patient's inadequate way of dealing with difficult situations. Consequently, the whole treatment must aim at the patient's return to ordinary conditions of life as soon as possible, and at a re-education of his ways of meeting difficulties. To this end it is profitable to go over with the patient the situations, emotional disturbances and motives that led up to the illness, and to do this without implying moral judgment or social indifference—certainly without teaching the patient one's psychological theories. It is a matter of general psychotherapy (see p. 1816); and it may entail a far-reaching analysis of the patient's past life, her emotional development and her instinctual tendencies. It is questionable, however, whether anyone without special psychiatric experience is wise to enter lightly upon this way of benefiting the patient. For, on the one hand, he may be misled into a wilderness of fantasy masquerading as once-repressed, now-recalled psychic trauma; and, on the other, he may be at a loss how to deal with the attachment and dependence upon him which the patient will come to show, and which may in fact be the chief influence in bringing about her precarious recovery. A great deal may be achieved—perhaps as much as by more thoroughgoing methods—if the physician, himself mature and with impartial insight into the psychological motivation of the symptoms, leaves aside in his dealings with the patient any very detailed inquiry into the causes of the illness and the purposes it served; and, instead, directs her towards a better social adaptation, by advising her to avoid when possible the situations that, as he sees, favour the production of symptoms, getting her into a disciplined way of living, and stepping in with explanation, support and advice whenever fresh difficulties arise. His success in getting rid of individual symptoms at the beginning may be an important factor in establishing the necessary relationship with the patient. Such a line of treatment is not heroic, it is scarcely even rational, in the sense of being causal, but it avoids some of the commonest blunders and may be strikingly successful. For this, or indeed any treatment, admission to hospital is not essential; but it will help when there are adverse factors in the patient's situation and, of course, will be essential if there be such symptoms as self-injury, suicidal attempts, pseudo-dementia, or gross paralysis. The danger of the patient's picking up new symptoms in hospital should also be weighed. Isolation is usually inadvisable.

Many of the symptoms of hysteria will not wait upon general treatment, but demand energetic intervention. Anorexia, for instance, cannot be allowed to go on to an avoidable inanition, nor a paralysis to the stage of contracture; a mute patient, or one who is deaf or blind or ignorant of his own identity, offers such practical obstacles to almost any kind of treatment that the symptoms must be tackled and disposed of early. For this purpose suggestive measures are valuable and appropriate physical treatment may be called for, e.g. supervision during feeding, or even tube-feeding in anorexia nervosa, physiotherapy for paralysis, voice-exercises. Suggestive measures need not take the form of hypnosis; suggestion in the normal waking state has many advantages over hypnosis, though those expert in the latter are sometimes very successful in their treatment. Suggestion, like almost every form of treatment of hysteria, has pitfalls, and its triumphs, like those of every other method, sometimes prove vain, but in the hands of a physician who is at once confident and cautious, this method may result in a satisfactory

recovery. If, in using suggestion, such physical devices as faradisation can be avoided, it is better to do so. As a means of demonstrating that the illness is not due to local disease, however, such methods sometimes take their place in a detailed plan of treatment. Motor and sensory symptoms can usually be got rid of in one or two sittings if the physician is patient, determined and confident in the use of persuasion and suggestion.

The choice of occupation, the settlement of any social cause of illness (e.g. claims for compensation), and the obtaining of a healthy attitude—neither complaisant, much-enduring nor harsh—on the part of the patient's relatives and friends, are all important factors in treatment. The hysterical reactions to injury call for special mention because of their frequency. Though often of transparent motivation, they are not by any means to be regarded as outright malingering; for the patient's feeling of illness may be sincere, his symptoms distressing, his anxiety typical, and his irritability and insomnia symptoms that he would gladly get rid of. But they are none the less psychogenic. It is often assumed that so far as an illness is psychogenic, it must be treated only by psychotherapy. This is false theory. There are few mental disorders in which psychotherapy alone produces such small benefit as in the hysterical conditions due to the compensation or pension situation that may follow an injury. Putting an end to the situation early and the resumption of ordinary activity as soon as any physical injury has been repaired are the most potent measures in the earlier stages. Even if the symptoms have been present a long time, the ending of disputes about claims and the return to ordered routine and regular occupation achieve more than do frequent medical interviews. Psychotherapy is then an adjunct, not an essential feature, of the treatment. Marriage should never be recommended as treatment for hysteria; the superstition about this has resulted in lamentable troubles, especially for the person the hysteric marries. This is not to say that every hysteric is to be dissuaded from marrying; there are more things than treatment to be considered then. Married hysterics, however, should not be recommended to have a child. Contrary to popular notions, pregnancy and puerperium more often aggravate than benefit hysteria. Moreover, hysterical women are not usually satisfactory parents, and commonly induce psychopathy in their children.

ANXIETY STATE

As already stated, the emotional syndrome so called is part of the group of affective disorder, in which depression and manic excitement are also included. It is there described. It would be indefensible to put into a special category all the forms of mental illness in which anxiety is conspicuous, for it can be severe in the most diverse conditions, ranging from delirium tremens to schizophrenia. The outwardly mild form, tending to chronicity and often largely psychogenic, responds well in the less advanced stages to psychotherapy; it is therefore important that its recognition should not be delayed because of a doubt as to physical disease. Yet often the correct diagnosis is overlooked while the patient is being investigated or treated for some local disorder. This arises partly because of the quasi-physical signs of fear which he may show—dizziness, tremor, nausea and

vomiting, indigestion, diarrhoea, shortness of breath, palpitation, a sense of oppression in the chest, rapid pulse, flushing, sweating, frequent passage of urine, etc. It is still more due to the patient's anxiety turning on his health, especially his physical health, and leading him to ask for more and more medical opinions, X-rays, laboratory investigations, etc., the favourable results of which, however, do not allay his worry. Over-cautious advice as to regime, based on a possibility that there may be some early physical disease, can be harmful to the patient's mental health in that it restricts his normal life, and may constantly recall and reinforce his anxiety. The converse error of mistaking some early symptoms of physical illness for hypochondriacal anxiety is equally to be avoided. Physical investigation of doubtful cases is, in short, indispensable, and should be prompt as well as thorough. When it fails to confirm the presence of a physical disorder the patient should not be treated as though he will still be in danger of the physical illness unless he takes special precautions in diet, exercise, etc. This is well illustrated by such a condition as effort-syndrome, where care taken to avoid any damage to the heart intensifies the illness. The patient should be fully investigated on the psychological side and treated accordingly; this does not mean that he should be treated only by psychotherapy. The discovery of a possible psychological cause for the symptoms does not prove that there is not also a physical cause for them, but it makes it less likely. The converse is also true. For ætiology, diagnosis, prognosis and treatment see section on Affective Disorders.

OBSESSIONAL DISORDER

Definition.—In this condition the characteristic feature is that, along with some mental happening, there is an experience of subjective compulsion and of resistance to it. Commonly the mental happening (which may be a fear, an impulse, or a preoccupation) is recognised, on quiet reflexion, as senseless; nevertheless it persists.

Ætiology.—**INTRINSIC.**—The hereditary factor is strong. A third of the parents of obsessional patients, and a fifth of their brothers and sisters, have themselves shown pronounced obsessional traits; the proportion is in each case higher if all forms of mental abnormality be included, since both schizophrenia and affective illnesses occur with more than average frequency in families of obsessionals. The abnormal personality of the parents is probably also potent as an environmental cause. Very many obsessional patients have for years before they became ill shown a rather characteristic mental constitution: they are excessively cleanly, orderly and conscientious, sticklers for precision; they have inconclusive ways of thinking and acting; they are given to needless repetition. Those who have shown such traits since childhood are often morose, obstinate, irritable people; others are vacillating, uncertain of themselves, and submissive. "Obsessional" traits occur, however, in many people who never become mentally ill, and in many who become mentally ill otherwise than with an obsessional disorder. Consequently these traits cannot be rigidly held to be the forerunners or non-morbid counterpart of obsessional illness.

EXTRINSIC.—The influence of strict, morose, cruel, overconscientious, or obsessional parents has just been mentioned. It is difficult to weigh its

importance ; certainly in some cases it plays no part. There is nothing specific in the situations which supply the content of an obsession : they might equally well have preceded hysterical symptoms, for example, in a person so predisposed. Nevertheless, the fright or pain which once accompanied a particular experience, or a long series of experiences, must not be overlooked in working out the multiple causes of some obsessions psychologically related to this experience.

Encephalitis lethargica and a few other cerebral diseases may produce typical obsessional symptoms in persons previously free from demonstrable tendencies in this direction.

Pathology.—Apart from the difficult instances in which lesions of the brain are accompanied by obsessions, this is at present wholly a matter of psychopathology. Some elements of an obsession are universal human attributes : all little children tend to ritualise and repeat ; all human beings are at times uncertain of the rightness or sense of what they have done ; they try to avert trouble by symbolical acts and other magical devices, whose effectiveness they may question (*e.g.* superstition) ; many normal people, moreover, have mild obsessions that do not bother them (*e.g.* scruples). The manifest struggle going on in the obsessional patient may be restated in terms of hypothetical instinctual tendencies. Such attempted explanation cannot be verified ; and it is more useful to pay heed to the repression, displacement and substitution which lead to symbolic representation of emotionally significant earlier experiences, and to the protective mechanisms by which the patient tries to ward off the painful and overwhelming obsession, with the result that he develops complicated rituals and similar devices which may be mistaken for the essential symptom. The transition from obsessional to schizophrenic is easy to understand psychopathologically, since in both some contents of consciousness are separated from the main stream.

Symptoms.—Obsessions are conveniently classified as : 1. ideas or images ; 2. impulses ; 3. phobias ; and 4. rumination. These overlap constantly.

Among obsessional *ideas and images* are tunes, phrases, mental pictures of a disagreeable sort (*e.g.* of a mutilated corpse), and obscene associations (*e.g.* every cranny reminds the patient of a vulva). Obsessional *impulses* are often of a suicidal or aggressive character : the patient may feel an urge to kick people in the street, to push his friend over a cliff, or to throw himself under a passing train. In many other cases, however, they are less alarming ; *e.g.* impulses to swear loudly in church, or to laugh at a funeral ; or more of an intellectual sort, such as an impulse to count and manipulate numbers senselessly or to avoid typing any word with a given number of letters or beginning with a particular consonant. *Phobias* are closely bound up with the other varieties of obsession : thus, the patient who has an impulse to plunge a knife into his friend's or his own neck has an understandable phobia of knives ; the patient who is troubled by obscene thoughts whenever he looks at a naked statue develops a phobia of museums. Not all phobias can be so accounted for ; they may rest on some forgotten alarm, and take a queer form, such as a phobia of lavatories or of one-legged men. It is loose usage to give the name "phobia" to every case in which an individual develops fear that is excessive or inexplicable ; the essential features of an obsession, already mentioned, should also be present. Fears of dirt or infection are very common phobias : they are symbols of moral, usually sexual, taint, and they

lead to much washing, etc.; thus, a patient who has blamed himself for masturbation may be constantly washing his hands, or following a complicated ritual of touching nothing with his bare hands for fear of contamination. Often the rituals and defensive precautions seem grotesque when compared with their ostensible purpose, as in the case of a patient who is perpetually putting himself to the greatest trouble in order to ensure that he never steps on a worm inadvertently; much of the grotesqueness disappears when it is discovered what the worm symbolises for him. Ludicrous as his behaviour may seem, it is often tragic in the distress, and indeed ruin, it may cause him. Another phobia is that which has fear as its object, *i.e.* the patient is afraid of any situation in which he may feel fear; some such patients do not leave their homes for years, because they fear they may have an attack of agoraphobia once they get outside. Obsessional *rumination* usually takes the form of endless questioning or search. The patient has to ask himself "Why" with pointless insistence about all manner of problems beyond his or anybody's grasp; or he has to keep casting round in his mind after some forgotten name or word which he could easily do without. Religious scruples sometimes fall into this category, as when a penitent is continually running to his confessor with some venial trifle he has come upon in his interminable self-questioning and doubt.

Obsessional patients are in most cases depressed; their illness is a depressing one. Besides this secondary depression, however, there is frequently an association of a more intimate kind, in which depression—or mania—is the essential or the main part of the illness, and the concurrent obsessions seem to be symptoms of this affective disorder. In such cases the obsessional illness is very often cyclical in its course. Anxiety is a common accompaniment of obsessions; in phobias it is most conspicuous. The anxiety is inseparable from the patient's struggle against the subjective compulsion which is so alarming to his feeling of integrity in self and mind, such a shock to his belief that he is a free agent. Schizophrenic symptoms may be in the offing, or actually present, when the obsessional ideas are of the magical kind, *e.g.* the patient feeling that the effect of his obscene thoughts upon others may be averted by some gesture, or when his rituals are carried to bizarre lengths, *e.g.* having to save the last drops of his urine because of some recurring doubt. Depersonalisation may occur in the course of an obsessional illness.

Diagnosis.—If the essential features, *i.e.* feeling of subjective compulsion and immediate resistance to this, be kept in view, it is seldom difficult to distinguish between obsessions, on the one hand, and delusions, hallucinations, ideas of reference or self-reproach, feelings of being influenced and schizophrenic stereotypies, etc., on the other. The only difference between obsessions and many schizophrenic phenomena towards which the patient retains insight and which he regards as alien to him, lies in the nature of the compulsion he experiences: in obsessions it is subjective—he feels that it comes from within his own mind, whereas in the schizophrenic phenomena he feels that it comes from without, it is imposed upon him. It is a difference, however, that may be obliterated, *i.e.* what was once obsessional may become schizophrenic, but this is an uncommon outcome when the obsessional disorder is definite and well-established. In differential diagnosis it must be remembered that obsessions may occur in the course of almost

any mental illness in a person of obsessional tendencies, and that the psychological mechanism for the production of obsessions, like that for hysterical symptoms, is present in almost everybody in varying degree. Consequently, an illness is not to be regarded as obsessional unless obsessions are the chief symptoms.

Course and Prognosis.—The outlook for recovery is worse if obsessional symptoms have been present since childhood, if they now fill up most of the patient's time, and if he is weakly resigned to his illness. The best outlook is when the obsessional illness comes on suddenly in a person who has not had conspicuous obsessional traits or who has had previous benign attacks. A cyclical course is not uncommon. The situation is ominous when the ritual gets more and more systematised and remote from what previously occasioned it. The development along schizophrenic lines, already mentioned, is more to be feared in such cases and in those with bizarre obsessional thoughts; the great majority of gross obsessionals, however, do not become schizophrenic or anything else than obsessional. About half the cases recover from an attack, which may, however, last for a year or even more. Many people are subject to brief attacks, lasting only a few days, and largely due to fatigue or physical illness reducing their mental health. Inter-current happenings influence the course of the illness, *e.g.* some men were free from symptoms during their period of war-service, with its routine and lack of responsibility or need for decision. The content of the obsessions is of little use prognostically. Old age is not in itself an adverse factor, but attacks in childhood suggest a strong constitutional bias and are therefore unfavourable on the whole. Few obsessionals give way to anti-social impulses, *e.g.* to suicide, homicide, delinquency. It is true that obsessionals who are also depressed may kill themselves, and that obsessionals who are irritable and angry may injure others; but obsessionals rarely yield directly to an impulse they have resisted, or need to have "irresistible impulse" urged in extenuation of a crime. Sexual offences and perversions are rarely obsessional.

Treatment.—Patients should be encouraged to continue at their occupation and not to test themselves, or try to overcome their obsession, by repeatedly putting themselves in a situation in which it will occur. So long as their impulses are not likely to get them into trouble, they should be encouraged to give way to them, rather than to "fight." The physician must aim at getting a patient well by putting an end to his anxiety and struggle; if that is not wholly attainable, the patient must be educated to deal with his obsessional tendencies by acknowledging their existence, their psychological origins, and their harmlessness in those very respects in which he thought them most harmful, *e.g.* obscenity. Frank recognition of obsessional tendencies, which everyone has in some degree, is an important step in learning to control them. In some patients the obsessional attack is so cyclical and almost self-limited that a brief rest and general care are all that is needed. In others, whose affection is chronic, recovery is out of the question, but advice about the management of their lives, varying according to their individual circumstances, helps them greatly. These patients, so prone to rumination and endless questioning, often clamour to be psycho-analysed. There is no evidence that psycho-analysis, however prolonged, benefits them more than methods that are not so exigent of time and money.

Obsessional children usually respond well to changes in their human environment, advised after the physician has inquired into the family and school situation; temporary separation from an obsessional parent or treatment of the latter often proves remarkably beneficial for the child. Discussion of his problems with the child (especially if they centre round secret sexual play) is an important adjunct of such treatment, just as it would be with an obsessional adult.

EDWARD MAPOTHER.
AUBREY LEWIS.

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a less extent to a myxœdematous state of the heart muscle. The degree of cardiac failure is not always related to that of enlargement. The rate of the circulation is diminished. On screening, in addition to possible enlargement of the heart, there is diminished pulsation. Changes in the electro-cardiogram are the most constant features (see p. 1029).

There may be indications of co-existing chronic myocardial disease, atheroma or hypertension.

A diagnosis may be made from the general symptoms of hypothyroidism, together with the clinical features described and an electro-cardiogram; and the response to appropriate treatment.

Treatment consists of thyroid therapy, given cautiously. The beneficial effect is very quick, including the cardiac enlargement and the electro-cardiogram.

FREDERICK W. PRICE.

CLINICAL ELECTRO-CARDIOGRAPHY

By means of the electro-cardiograph it is possible to obtain graphic records of the movements of both auricles and ventricles, to study the time-relations of their contractions, and to measure the function of conductivity not only of the auriculo-ventricular junctional tissues but also that below the division of the auriculo-ventricular bundle into two branches. In addition, it tells us the point of origin of the impulse formation and also the path along which the wave of excitation travels.

All the various forms of irregular action of the heart can be identified with certainty. The electro-cardiograph affords the most precise means of investigating the function of the myocardium. Disease of the myocardium may, by interfering with the normal path of the wave of excitation, modify the form of the ventricular complex. The instrument is often of great value in the diagnosis of coronary disease and infarction of the heart; it contributes the most certain sign of transposition of the heart; it gives evidence of left- or right-sided preponderance when either exists; it is sometimes of value in the diagnosis of chronic valvular disease and congenital morbus cordis; and during the administration of quinidine in the treatment of persistent auricular fibrillation and persistent auricular flutter, the changes in the cardiac rhythm induced by the drug may be followed, and the dosage controlled accordingly.

It has been known for a considerable time that changes in electric potential take place in muscle when it contracts, and, further, that a record of these changes may be obtained by connecting the muscle with a sensitive galvanometer by means of electrodes.

A. D. Waller, in 1887, employed a capillary electrometer to register the changes in electric potential in the human heart during contraction. He demonstrated that these changes were distributed through the body, and he used the moist skin surfaces of the arms and legs as leads, connecting them with a galvanometer.

Einthoven employed the string galvanometer to register the changes

in electric potential in the human heart. He modified this instrument, the Einthoven string galvanometer being now generally employed in physiological and clinical investigations. In the Einthoven galvanometer there is an exceedingly fine silvered glass fibre, which is suspended in a box and placed vertically between the poles of a powerful electro-magnet. When a current passes through this fibre, minute movements of the fibre take place. The shadow of the oscillating fibre is magnified, and projected on to a photographic screen, and in this way a photograph of these movements is obtained. The sensitiveness of the fibre can be adjusted by altering the tension, the standard generally adopted being that of Einthoven, *i.e.* that when a difference of potential of one millivolt is passed through the fibre, this gives a deflection on the plate of 1 cm. in amplitude.

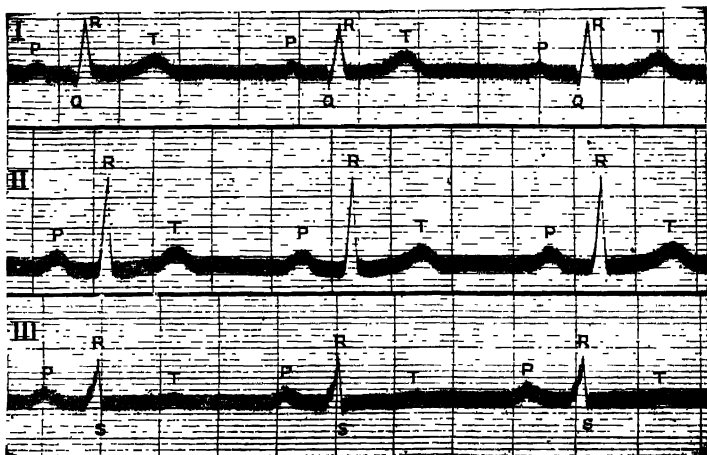


FIG. 46. —A normal electro-cardiogram of leads I, II, and III. The first deflection, *P*, is due to the contraction of the auricles: the others are the result of the contraction of the ventricles, and are termed *Q*, *R*, *S*, and *T*. In normal subjects the amplitude of all the deflections is usually greatest in lead II, especially the deflection *R*.

Recently some workers employ valve modification and a mirror galvanometer instead of the string galvanometer.

Both varieties of electro-cardiographs are now made in reliable portable form, which can be taken to the bedside of the patient.

Formerly the common practice was to accept three leads and the following nomenclature, *i.e.* lead I, or the transverse, a lead from the right and left hands; lead II, or the axial, from the right hand and left foot; and lead III, or the left lateral, from the left hand and left foot. Now a fourth or chest lead is also employed, in which one electrode is applied over the præcordium and another over some distant part of the body. This lead, on the joint recommendations of the Cardiac Society of Great Britain and Ireland and the American Heart Association, is arranged as follows: One electrode is placed over the extreme left border of the apex-beat, or, if this

cannot be determined by palpation, it may be applied in the fifth intercostal space immediately outside the left border of percussion dullness, or just outside the left mid-clavicular line. The other electrode is applied either to the left leg or to the right arm. The leads so arranged are referred to as leads *IV F* and *IV R* respectively. In using either of the foregoing connections, the correct polarity is obtained as follows: The lead switch is turned to lead 1; the left arm terminal is connected to the præcordial electrode; and the right arm terminal is connected to the distant electrode—to the left leg for lead *IV F*, or to the right arm for lead *IV R*.

The record of the changes in electric potential which take place in the heart during contraction is called an electro-cardiogram.

If a normal electro-cardiogram of leads I, II, and III be studied, certain

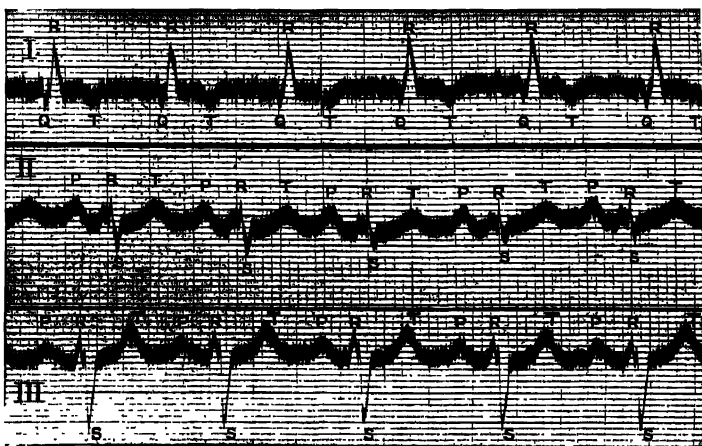


FIG. 47.—Electro-cardiogram, showing inversion of *T* in lead I. There is also left-sided preponderance.

upward and downward deflections or waves are seen in each cardiac cycle (Fig. 46). Following Einthoven, the deflections are called *P*, *Q*, *R*, *S*, and *T*, in some instances *T* being followed by *U*. *R* and *T* are the most constant deflections, the former especially so; *Q* and *S* are not infrequently absent, particularly the first; while the *U* deflection is of very uncommon occurrence.

P, *R*, *T*, and *U* are upward deflections, while *Q* and *S* are downward ones.

The *P* deflection is due to the contraction of the auricles. *Q*, *R*, *S*, and *T* are due to the contraction of the ventricles. *U*, if present, occurs early in diastole, and its significance is not fully understood.

That portion of the electro-cardiogram from the beginning of *P* to the commencement of *Q* is called the auricular complex. *P* represents the spread of the wave of excitation over the auricles. It is an upward, small, and usually rounded deflection, but it may be pointed. It is succeeded by an

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isoelectric interval indicated by the string either remaining at the zero level, or descending or ascending slightly, in which event usually the former.

That portion of the electro-cardiogram from the beginning of *Q* to the end of *T* is designated the ventricular complex. *Q*, *R*, *S* constitute the initial group of deflections, while *T* is the terminal or final deflection. *Q* passes at once into *R*, and *S* follows immediately upon the latter. *Q* and *S* are downward steep deflections, and usually very small. *R* is an upward, conspicuous, sharp spike, and of greater amplitude than any of the other deflections. The *S-T* interval usually exhibits a pronounced zero-line, which however may descend or ascend slightly, but, on the other hand, there may even be no zero-line. *T* is an upward, prominent, broad and rounded deflection, but in lead III it not infrequently points downward (see p. 1004).

In normal subjects, the amplitude of all the deflections, especially *R*, is

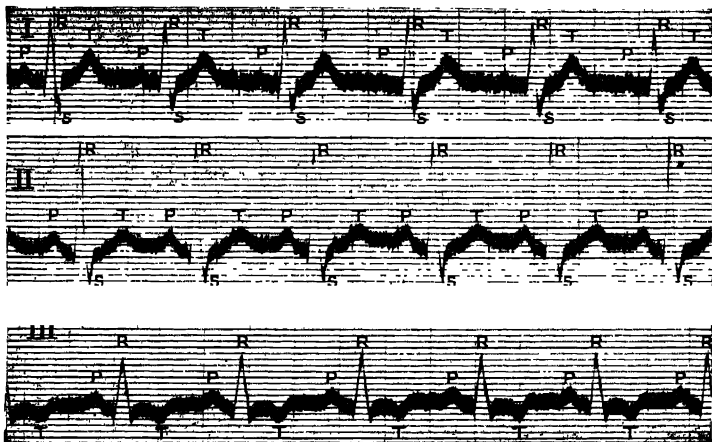


FIG. 48.—Electro-cardiogram showing inversion of *T* in lead III.

usually greatest in lead II, while those in lead III are not infrequently of small amplitude.

The time-distance between the beginning of *P* and the commencement of *Q*, or between *P* and *R*, as the case may be, is an index of the *A-s-Vs* interval, that is, the interval separating the commencement of auricular and ventricular contraction, and is a measure of the function of conductivity of the auriculo-ventricular node and bundle above its division into two branches; it is called the *P-Q* or *P-R* interval. The rule is to employ the latter, on account of the frequent absence of the *Q* deflection. The *P-R* interval is a more reliable indication of the rate of conduction of the wave of excitation than the *a-c* interval in a polygraphic tracing, because the presphygmic interval and the period between the opening of the aortic valves and the carotid pulse are not included. In normally acting hearts the interval varies between 0.12 and 0.18 second. If it exceeds the latter, and certainly if more than 0.20 sec. (Fig. 78), we may conclude that the

function of conductivity is definitely depressed. The *P-R* interval is diminished in auriculo-ventricular nodal rhythm and in functional bundle-branch block (see Fig. 82).

The period of time occupied by the ventricular complex is approximately that of the ventricular systole. That occupied by the *QRS* group of deflections is of much importance. It should not exceed one-tenth second. If it does it indicates a delay in the conduction of the wave of excitation through the ventricular muscle. This occurs in bundle-branch block, arborization block, ventricular extrasystoles, in the ventricular variety of paroxysmal tachycardia, and, it may be, in extreme preponderance of either ventricle.

The *Q-T* or *R-T* duration varies considerably. With a normal cardiac rate, it probably averages between 0.32 to 0.35 second. It is diminished by an increased cardiac rate, and *vice versa*. When the cardiac rate is unusually frequent the duration of the diastolic interval is so shortened that the deflection *T* approaches more and more to the following *P*, and *P* and *T* may even coincide and are superimposed (Fig. 49).

In auricular hypertrophy, such as in mitral stenosis and congenital

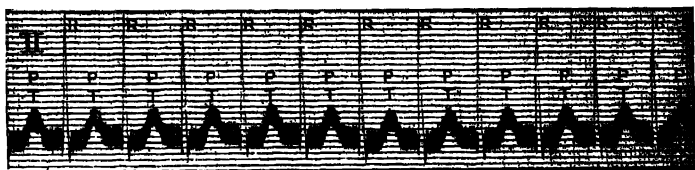


FIG. 49.—Electro-cardiogram in which *P* and *T* coincide and are superimposed.

pulmonary stenosis, especially the former, the *P* deflection, particularly in lead II, is of increased amplitude (Figs. 53 and 54), and not infrequently is also broad and has a flat top, and may be notched or bifurcate (Figs. 55 and 56). Some writers are of opinion that a mere increase in the amplitude of *P* is occasionally met with in normal hearts.

A large *Q* in lead III is often a feature of the *T3* type of infarction, either recent or old. It should be noted, however, that it may also occur with a horizontal or transverse position of the heart due to a high diaphragm, as, for example, in ascites, pregnancy, or obesity. In the differential diagnosis, whether there is also a *Q* of somewhat smaller amplitude in lead II and a characteristic alteration in the *R-T* (*S-T*) interval and in the form of the *T* deflections are of cardinal importance. In addition, a large *Q* due to upper displacement of the diaphragm is diminished or abolished by holding the breath after a deep inspiration. A *Q* in lead I is strongly suggestive of an old infarct of the wall of the left ventricle near the apex and the adjacent part of the interventricular septum.

Notching or splintering or slurring of the *QRS* complex of slight degree may be found in normal conditions, and in preponderance of either ventricle. These in pronounced degree and also notching which is irregular signify that the wave of excitation is spreading in the different parts of the ventricular musculature in an abnormal manner. They may be met with in bundle-

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branch block, arborization block, ventricular extra-systoles, and in the ventricular variety of paroxysmal tachycardia.

Diminution in the amplitude of *T* occurs during forced expiration, and it may be with advancing age. The *T* deflections may be flattened or inverted in the three leads in hypothyroidism (see Fig. 95). Diminution of *T* in lead II is frequently met with in marked left- or right-sided preponderance. Inversion of *T* in lead III alone (Fig. 48) is not infrequently found in health, and therefore its significance in a single examination is uncertain and it is of essential importance to know whether the deflection was formerly upright. Among pathological conditions, it is found in marked right-sided preponderance. Inversion of *T* in lead III and lead II may occur during forced expiration. Apart from this, it is probably pathological.

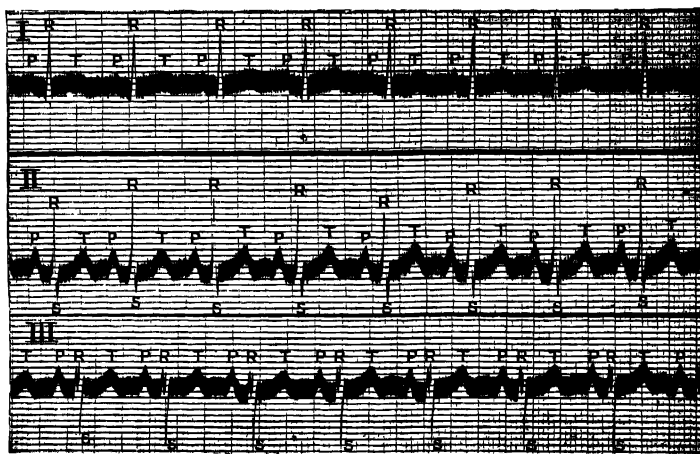


FIG. 50.—Electro-cardiogram showing predominant hypertrophy of the left ventricle.

Inversion of *T*₁ and *T*₂ may be regarded as pathological, suggesting coronary or myocardial disease. Inversion of *T*₁ is also to be met with in gross left-sided preponderance.

The changes in the *T* deflections which occur in bundle-branch block, coronary occlusion, and with full doses of digitalis are described elsewhere.

The characteristics of a normal fourth lead are as follows (Fig. 51). The first deflection, *P*, is upright, and is rather smaller than in the other standard leads but less so in lead IV *F*. The initial group of ventricular deflections is of larger amplitude than in leads I, II, and III, and is diphasic, the first wave, *R*, being positive and the second, *S*, negative, and they are approximately of equal size. The *S-T* interval, as in the other three leads, is usually isoelectric but it may, on the other hand, show a slight inclination to rise above or to fall below the zero line. The *T* deflection is positive, and is of increased amplitude.

The form of electro-cardiographic curves depends upon the point of origin of the impulse formation, the paths along which the wave of excitation travels, and the manner of conduction of the latter; any departure from the normal in respect of one of these will result in an alteration in the form of the electro-cardiogram.

A typical *P* signifies that the impulse arises in the sino-auricular node and that the wave of excitation spreads over the whole of the auricles along the normal paths. When the auricle contracts in response to impulses not generated at the sino-auricular node, almost always the *P* deflection is of abnormal form. The degree of this depends upon the distance of the site of origin from the normal, for example, if near the node *P* may be almost normal. Often the deflection is inverted. The various forms of abnormal *P* deflections which may be found in auricular or auriculo-ventricular nodal extra-systoles, auricular or auriculo-ventricular nodal paroxysmal tachycardia, auriculo-ventricular nodal rhythm, and auricular flutter are described later. A diphasic or inverted *P* in lead III alone may occur in left-sided preponderance and in the absence of any pathological condition.

Similarly, a typical ventricular complex signifies that the contraction of

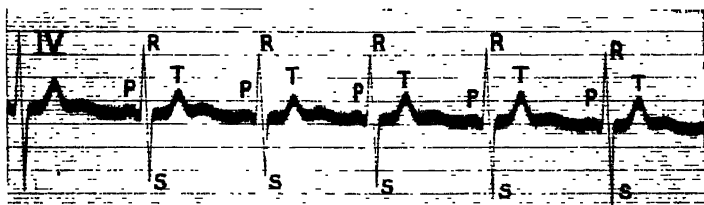


FIG. 51.—A normal electro-cardiogram of lead IV.

the ventricles is supra-ventricular in origin, *i.e.* they have contracted in response to an impulse which arises above the division of the auriculo-ventricular bundle, and that the wave of excitation proceeds along the normal paths. An atypical ventricular complex means that the contraction of the ventricles is ventricular in origin, *i.e.* the stimulus arises below the division of the auriculo-ventricular bundle. When the ventricular contraction is *aberrant* (see p. 847), the complex, while fundamentally typical, differs from the normal to some extent, this varying from a slight degree to even, rarely, that in which the complex resembles one of ventricular type.

CARDIAC HYPERTROPHY.—It has been previously pointed out that in cardiac hypertrophy, while both ventricles are more often affected than one alone, one ventricle is frequently involved to a greater degree than the other. This predominant hypertrophy or preponderance of either ventricle is revealed by the electro-cardiograph. If in cardiac hypertrophy an electro-cardiogram does not indicate either right- or left-sided preponderance, we may assume that the hypertrophy involves both ventricles approximately equally.

In left-sided preponderance, the amplitude of *R* is greater in lead I than in lead III, and the amplitude of *S* in lead III is greater than in lead I (Figs. 47 and 50). The deflections of greatest amplitude in leads I and III, there-

fore, point away from each other. In right-sided preponderance, the amplitude of *S* is greater in lead I than in lead III, and that of *R* in lead III is greater than in lead I (Fig. 52), and so the deflections of greatest amplitude point towards each other.

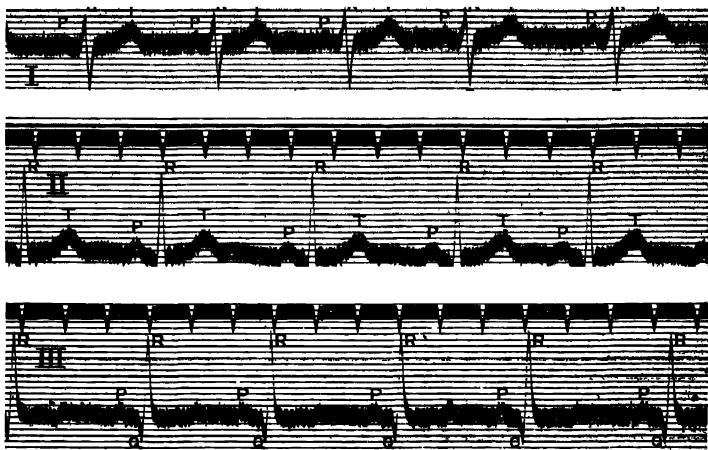
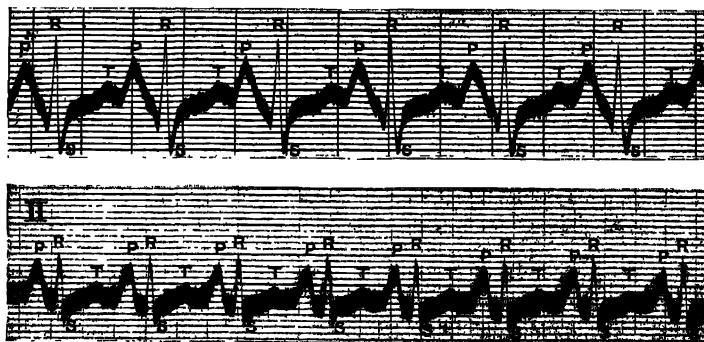


FIG. 52.—Electro-cardiogram showing predominant hypertrophy of the right ventricle.



FIGS. 53 and 54.—Electro-cardiograms from two different subjects showing increased amplitude of the deflection *P*.

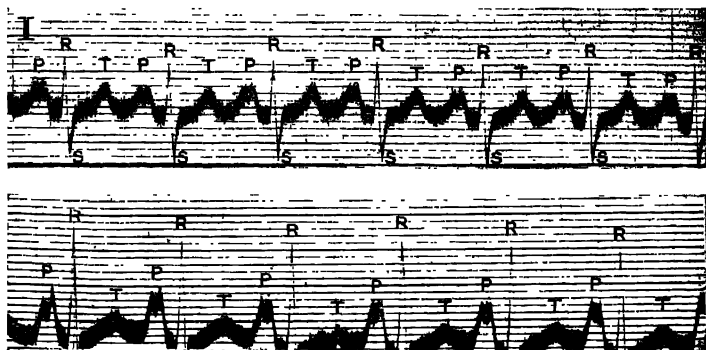
In marked preponderance of the left side, there is often inversion of *T* in lead I; and in that of the right side, inversion of *T* in lead III. Barnes and Whitton have suggested that these changes in the *T* deflection result from mechanical strain on one ventricle rather than actual disease of the myocardium other than hypertrophy. In extreme preponderance of either

ventricle, the period of time occupied by the *Q*, *R*, *S* group of deflections may be increased, exceeding one-tenth of a second.

In the diagnosis of preponderance of either ventricle it is necessary to exclude displacement of the heart, for the following reason. The electrical axis of the organ is influenced by the anatomical axis, so that displacement may give rise to electro-cardiographic curves of right- or left-sided preponderance. Thus, a horizontal or transverse position of the heart due to a high diaphragm tends to produce a curve of left ventricular preponderance; while a vertical position of the organ with a low diaphragm, as seen in asthenic subjects, tends to produce that of right ventricular preponderance.

It is also necessary to distinguish between predominant hypertrophy of the left or right ventricle and a lesion of the left or right main branch of the auriculo-ventricular bundle (new nomenclature) respectively. This is referred to on p. 1020.

CHRONIC VALVULAR DISEASE.—In aortic valvular disease there is usually



FIGS. 55 and 56.—Electro-cardiograms from two different cases of mitral stenosis. The deflection *P* is increased in amplitude, and is also broad, has a flat top, and is bifurcate.

left-sided preponderance. In mitral stenosis there is generally right-sided preponderance; and the *P* deflections may show the changes indicative of auricular hypertrophy described on p. 1003. The latter, when present, are of diagnostic value, especially when there is also right-sided preponderance. When auricular fibrillation supervenes, the curves will present features characteristic of that condition.

CONGENITAL HEART DISEASE.—The amplitude of the deflections is sometimes greater than in the acquired form of valvular disease. There is frequently right-sided preponderance, often of marked degree, especially in pulmonary stenosis.

In dextro-cardia all the deflections of a curve from lead I are inverted, while lead III is equivalent to the normal lead II, and lead II to the normal lead III (Fig. 57). This form of electro-cardiogram is pathognomonic of the condition. It is to be noted that if there is also a lesion giving rise to enlargement of the right ventricle, the electro-cardiogram will be that of left-sided preponderance with inversion of *P* in lead I. In uncomplicated wide patency of the interauricular septum there may be deviation of the electrical axis

to the right. In patency of the interventricular septum the electro-cardiogram is usually normal, except when the defect in the septum involves the

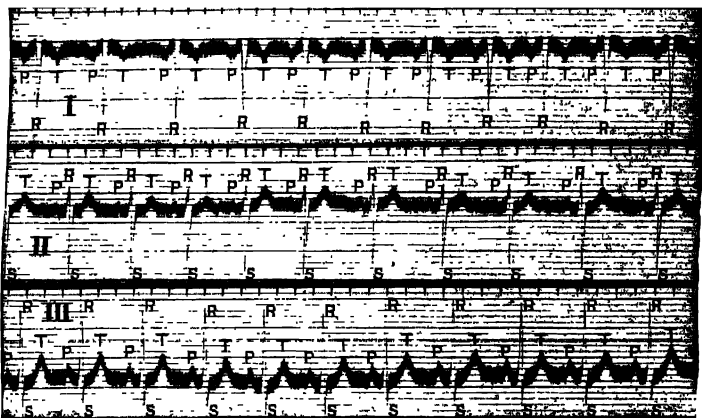
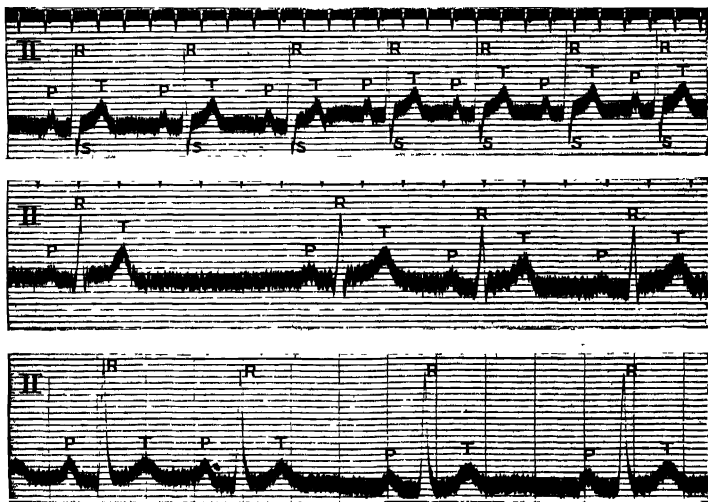


FIG. 57.—Electro-cardiogram from a case of transposition of the heart. All the deflections in lead II are inverted.



Figs. 58, 59 and 60.—Electro-cardiograms from three different subjects showing sinus irregularity. The auricular and ventricular deflections are of normal form, but there is a variation in the length of the intervals between T and P.

auriculo-ventricular bundle and so gives rise to congenital heart-block. In congenital pulmonary stenosis the electro-cardiogram will reveal a marked right ventricular preponderance.

SINUS ARRHYTHMIA.—The electro-cardiogram of this rhythm may readily be identified (Figs. 58–60). There is merely a variation in the length of the diastolic periods, *i.e.* the intervals between *T* and *P* (see p. 885).



FIG. 61.—Electro-cardiogram showing ventricular extra-systoles, marked *Ex.S.* The corresponding *P* deflections are embedded in the diaphasic variations. There is also inversion of *T* in lead I, and left-sided preponderance.

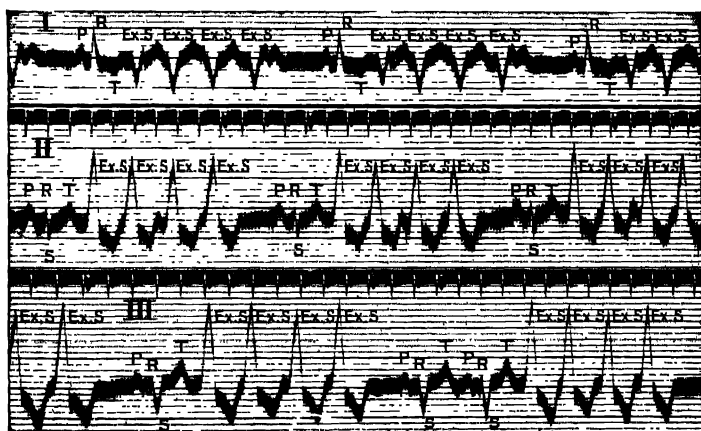


FIG. 62.—Electro-cardiogram showing rapid successions of ventricular extra-systoles. There is also inversion of *T* in lead I and left-sided preponderance.

EXTRA-SYSTOLES.—Extra-systoles may be readily recognised by means of the electro-cardiograph, and usually their site of origin.

In the ventricular variety (Figs. 61 and 62) the ventricular complex occurs

earlier than the anticipated time. As the ventricular contraction is ventricular in origin and therefore the wave of excitation travels along abnormal channels, the ventricular complex is wholly atypical: it is diphasic, and is of increased amplitude. It is of the same duration as that of the rhythmic contraction.

There are two main varieties of ventricular extra-systoles, and it is usually possible to differentiate them by means of the electro-cardiograph. In one, the ventricular complex consists of an upward, tall and pointed deflection, and then of a downward and broader deflection, in lead I; and of a downward, deep and pointed deflection, and then of an upward and broader deflection, in lead III. In the other variety, the ventricular complex consists of a downward, deep and pointed deflection, and then of an upward and broader deflection, in lead I; and of an upward, tall and pointed deflection, and then of a downward and broader deflection, in lead III. It was formerly thought, largely as the result of experiments in animals, that the first variety has its origin in the left ventricle or apical portion of the heart, and the second in the right ventricle or basal portion. Recent evidence has been adduced, based partly on direct observations on the exposed human heart, which strongly suggests that the opposite is the case. The direction of the deflections in lead II is usually the same as in lead III, but the reverse may be the case.

In the ventricular variety of extra-systoles, the auricle maintains its usual rhythm and contracts as the result of the normal stimulus from the sinus. It follows, therefore, that the corresponding *P* deflection appears at the anticipated time and is normal in form. But it is usually embedded in the ventricular complex, although it may be detected in this part of the electro-cardiographic curve in some cases, and occasionally is nearly separate.

If the ventricular extra-systole takes place after the normal auricular contraction, and the wave of contraction from the auricle has reached the ventricle and met that of the premature ventricular contraction in the ventricular wall, the ventricular complex of the premature contraction of the ventricle will present both typical and atypical features.

The site of origin of a ventricular extra-systole may occasionally be at one time in the basal or right portion of the ventricle, and at another in the apical or left portion, in the same subject.

A ventricular extra-systole is usually followed by a compensatory pause, which is complete. Sometimes, when the cardiac rate is slow, an interpolated extra-systole occurs.

In the auricular variety of extra-systole (Fig. 63) the *P* deflection takes place before the anticipated time. As the point of origin of the stimulus for contraction is at a site other than the sino-auricular node, almost always the *P* deflection is of abnormal form. The degree of difference depends upon the distance of the site of origin of the impulse from the sino-auricular node; and if near or at the node, *P* may be practically normal. Often the deflection is inverted (see lead II). The premature contraction of the auricle may take place so early as to coincide with the ventricular contraction of the preceding cycle, in which case *P* and the preceding *T* are superimposed. The *P-R* interval may be increased. The *P* deflection is usually followed by a premature ventricular complex. This is of typical form, since the ventricular contraction is of supra-ventricular origin and therefore the wave of excitation to the ventricle travels along the usual paths,

but it may be aberrant. Almost always aberrant ventricular beats are found only when there is diminished auriculo-ventricular conductivity. In some cases, the stimulus for contraction does not reach the ventricle at all, in which case the premature contraction of the auricle is not followed by a premature contraction of the ventricle—"blocked auricular extra-systole" (third extra-systole in lead I of Fig. 63).

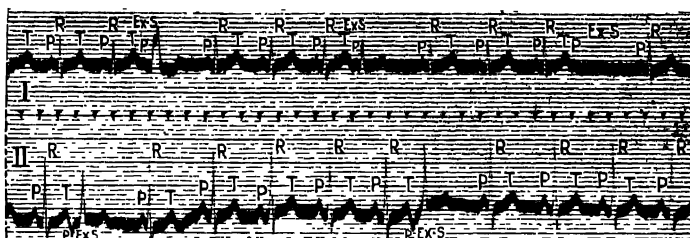


FIG. 63.—Electro-cardiogram showing auricular extra-systoles, marked *Ex.S.* The premature *P* deflections are of normal form in lead I, and of abnormal form—being inverted—in lead II. The premature ventricular complexes are of abnormal form. The third extra-systole in lead I is blocked.

In auricular extra-systole the compensatory pause is rarely complete.

In the auriculo-ventricular nodal variety of extra-systole (Fig. 64) there is prematurity of the *P* deflection and also of the ventricular complex. In cases in which the contraction of the auricle and ventricle is absolutely synchronous, the *P* deflection coincides with, and is embedded in, the *Q, R, S* complex (see lead II). When the contraction of the auricle begins before that of the ventricle, the *P* deflection precedes *R*, and the *P-R* interval is

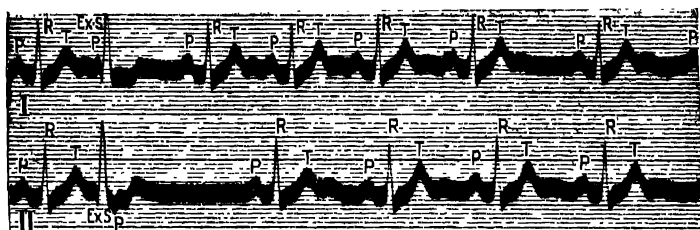


FIG. 64.—Electro-cardiogram showing auriculo-ventricular extra-systoles, marked *Ex.S.* In that of lead I the premature *P* deflection occurs before that of the ventricular complex, while that in lead II is embedded in the ventricular complex.

diminished (see lead I). When the auricular contraction commences after that of the ventricle, the *P* deflection follows the *R* deflection. When the *P* deflection is to be observed, as the auricular contraction is due to an impulse which arises at an abnormal point, it is almost always abnormal in form, often being inverted. As the ventricular contraction is supra-ventricular in origin, the ventricular complex is of typical form but may be aberrant.

In the auriculo-ventricular variety of extra-systole, the compensatory pause may, or may not, be complete.

Occasionally the beat immediately following an extra-systole arises from the same site as that of the premature contraction.

PAROXYSMAL TACHYCARDIA.—The first *P* deflection of the paroxysm is premature, and usually the paroxysm is followed by a long pause.

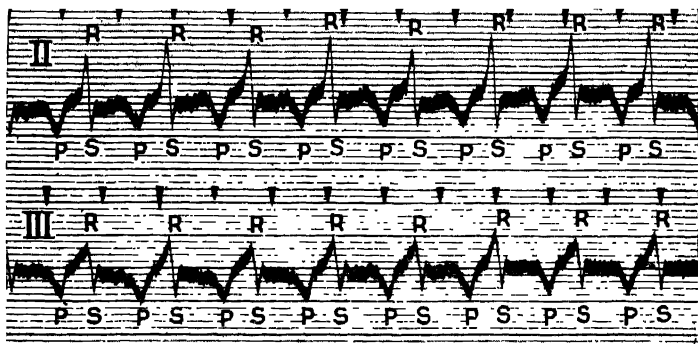


Fig. 65.—Electro-cardiogram showing the auricular variety of paroxysmal tachycardia.

There is no essential difference between the complexes of the individual beats during a paroxysm and those of single extra-systoles in the same individual.

In auricular paroxysmal tachycardia (Fig. 65) there is a rapid succession of *P* deflections occurring at regular intervals. As in the case of auricular extra-systoles, these are almost always of abnormal form, often being inverted. The contraction of the auricle may take place so early as to coincide with the ventricular contraction of the preceding cycle, in which

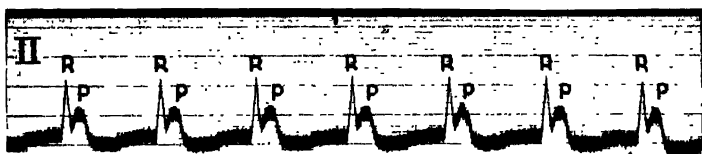


Fig. 66.—Electro-cardiogram showing the auriculo-ventricular nodal variety of paroxysmal tachycardia.

case *P* and the preceding *T* may be superimposed. The *P*–*R* interval may be increased. The *P* deflections are usually followed by ventricular complexes of the same form, or approximately so, as those preceding and following the paroxysm. There may be auriculo-ventricular block or bundle-branch block. In the latter case the ventricular complexes may resemble those of ventricular paroxysmal tachycardia. In this connection, in the differential diagnosis between the ventricular complexes of supra-ventricular and of ventricular origin, it is important to note whether any part of the *P* deflection

can be detected in the ventricular portion of the electro-cardiographic curve or not.

In auricular paroxysmal tachycardia, the rhythm is regular.

In auriculo-ventricular nodal paroxysmal tachycardia (Fig. 66) the electro-cardiogram is often difficult to determine. There is a rapid succession of auricular and also of ventricular complexes. In cases in which the contractions of the auricle and ventricle are absolutely synchronous, the *P* deflections coincide with and are embedded in the *Q*, *R*, *S* complexes. When the contractions of the auricle begin before those of the ventricle, the *P* deflections precede the *Rs*, and the *P-R* interval is diminished. When the auricular contractions commence after those of the ventricle, the *P* deflections follow the *Rs*. When the *P* deflections are to be observed, they are

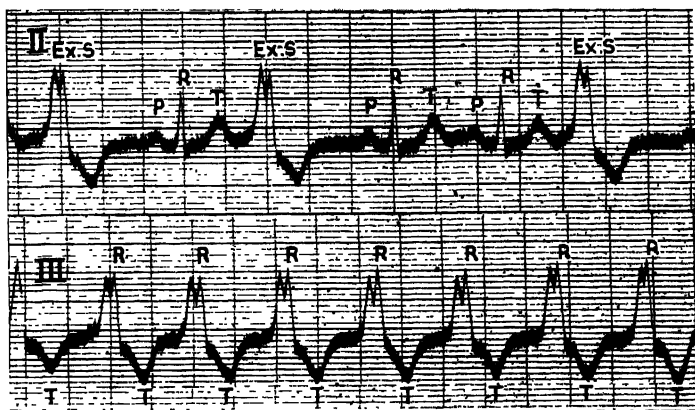


FIG. 67.—Electro-cardiogram of leads II and III. Lead II shows three isolated extra-systoles of ventricular origin. Lead III shows the ventricular variety of paroxysmal tachycardia, the complexes of which are of the same form as those of the isolated extra-systoles in lead II.

usually of atypical form, often being inverted. The ventricular complexes are of typical form but may be aberrant.

In auriculo-ventricular nodal paroxysmal tachycardia the rhythm may be regular or irregular.

In ventricular paroxysmal tachycardia (Figs. 67 and 68) there is a rapid succession of ventricular complexes. Usually each of these is of the same form as those of isolated extra-systoles arising in one ventricle (see p. 1010). In some cases, however, the point of origin of the beats is sometimes in one ventricle and at other times in the other, resulting in complexes of different form; and this may occur in the case of every other beat, so that complexes of different form alternate, which condition is sometimes called bi-directional ventricular tachycardia, and is of very serious significance. Sometimes the ventricular complexes are of indefinite form.

In the ventricular variety of paroxysmal tachycardia, the auricle generally maintains its usual rhythm, contracting as the result of an impulse arising

in the sino-auricular node. It follows, therefore, that the *P* deflections occur at the usual intervals and also are of normal form. They are generally embedded in the ventricular complexes, but in some cases they may be detected in this portion of the electro-cardiographic curve and occasionally they are nearly separate. In some cases, however, the ventricular rhythm gives rise to retrograde auricular beats, either with each ventricular contraction, or, less frequently, *i.e.* retrograde heart-block. Rarely there is auricular fibrillation, and more rarely auricular paroxysmal tachycardia, or auricular flutter.

In ventricular tachycardia, any existing irregularity of rhythm is usually so slight that it is detected only by means of graphic methods.

In the differential diagnosis of paroxysmal tachycardia by the electro-cardiograph, it is to be observed that, as the difference in the form of the complexes of the abnormal and the normal rhythms may be only slight it may be necessary to compare carefully the complexes of the paroxysm with those of the beats which either preceded or followed it—indeed, an analysis of either transition periods may be necessary.

AURICULO-VENTRICULAR NODAL RHYTHM.—In this condition, when the

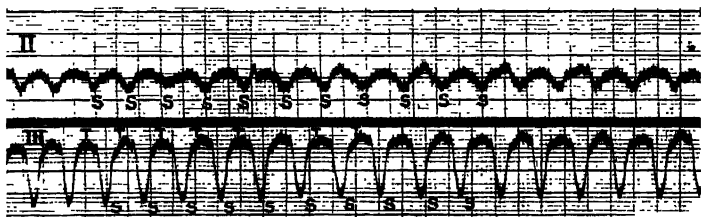


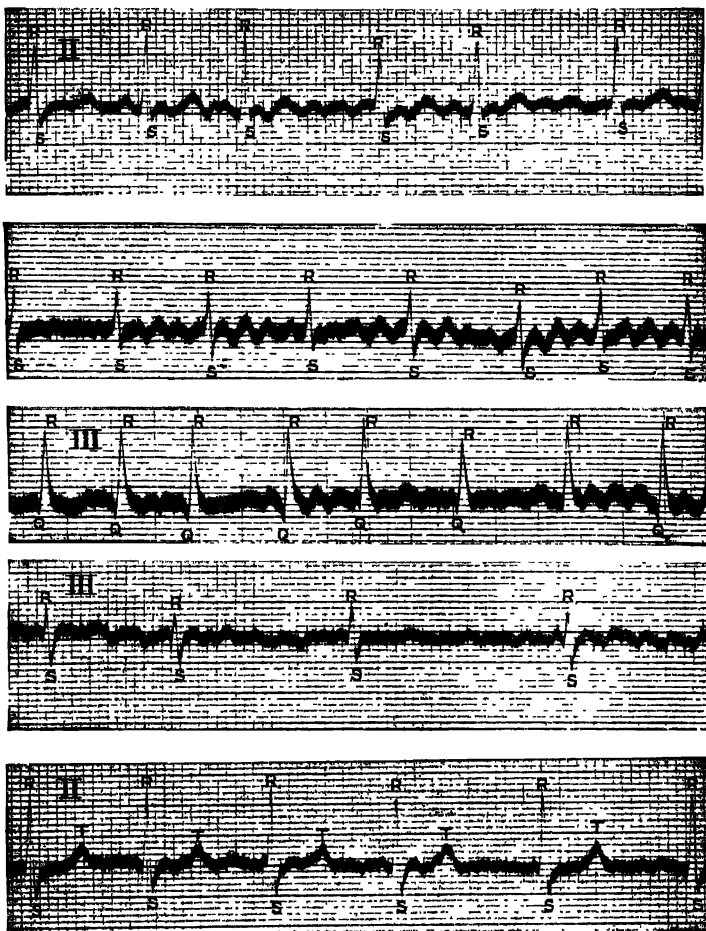
FIG. 68.—Electro-cardiogram of leads II and III showing the ventricular variety of paroxysmal tachycardia.

contractions of the auricle and ventricle are absolutely synchronous, the *P* deflections coincide with and are embedded in the *Q*, *R*, *S* complexes. When the contractions of the auricle begin before those of the ventricle, the *P* deflections precede the *Rs*, and the *P*-*R* interval is diminished. When the auricular contractions commence after those of the ventricle, the *P* deflections follow the *Rs*. When the *P* deflections are to be observed, they are usually of abnormal form, often being inverted. The ventricular complexes are of typical form but may be aberrant.

AURICULAR FIBRILLATION.—In auricular fibrillation the electro-cardiogram is characteristic (Figs. 69-73).

There is an absence of *P* deflections. There are, on the other hand, oscillations caused by the fibrillating auricle, called fibrillary waves, during ventricular diastole, at a rate ranging from 300 to 600, and usually about 450 times, per minute. They occur at irregular intervals, and their form and size vary, the latter being sometimes very minute and sometimes considerable. They are most evident in cases of slow cardiac action. They may coincide with the *T* deflections, in which case the outline of the latter is altered. Apart from the very rare cases in which complete auriculo-ventricular block is present, the ventricular rhythm is completely irregular, *i.e.* the *R* deflec-

tions occur at irregularly irregular intervals; their amplitude varies from cycle to cycle; and often there is no relationship between the length of a pause and the amplitude of the *R* deflection which follows it. When the



FIGS. 69-73 — Electro-cardiograms from cases of auricular fibrillation.

ventricular rate is slow or very rapid, this may be difficult to determine. As the ventricular beats are supra-ventricular in origin, the complexes are of typical form but they may be aberrant. There may be superadded ventricular extra-systoles, usually during the administration of one of the digitalis group of drugs (see p. 868).

In the differential diagnosis between the electro-cardiograms of auricular fibrillation and those of auricular flutter, in the former almost always the ventricular rhythm is completely irregular, while in the latter, it will be found that such is not the case, even when the rhythm is very irregular. Further, the deflections due to auricular systole in auricular flutter may be distinguished from the fibrillary waves in auricular fibrillation in that they are less frequent, rhythmic, of larger amplitude, and almost invariably of constant form and size.

AURICULAR FLUTTER.—In this condition it is of particular importance to analyse the three leads (Figs. 74–76). The *P* deflections may range from 180 to 360, the usual rate being about 300, per minute. In all the leads the deflections occur at regular intervals, and as soon as one terminates the next

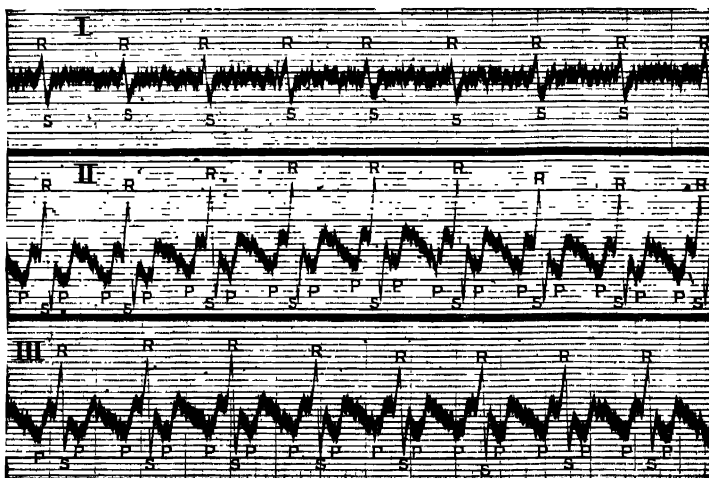


FIG. 74.— Electro-cardiogram from a case of auricular flutter, with 2 : 1 heart-block. The rate of the auricle is between 320 and 330 per minute.

one commences. In each lead of any given case they are almost invariably of constant form. In lead I the amplitude is comparatively small. As the impulse arises at an abnormal point, the deflections are atypical in form. The ascending limb is rather sharp, and the descending more gradual, and the summit may be dome-shaped. It is difficult to say at what part the deflection begins. As the ventricular complexes are supra-ventricular in origin, they are of typical form. They are superimposed upon the *P* deflections, modifying their outline. In some cases *T* may be detected. Excepting in the very rare cases of 1 : 1 rhythm, in which event there is an equal number of *P* deflections and ventricular complexes, there are two or more *P* deflections to each ventricular complex, according to the degree of auriculo-ventricular block (see p. 900). When the response of the ventricle to auricular contraction is at irregular intervals, the ventricular rhythm may be even very irregular.

It is necessary to differentiate the electro-cardiograms of auricular flutter from those of auricular fibrillation. This is dealt with on p. 1016.

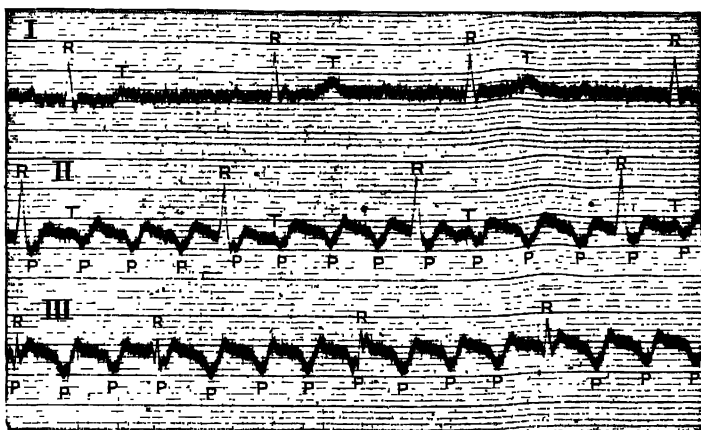


FIG. 75.—Electro-cardiogram from a case of auricular flutter, with 4 : 1 heart-block.

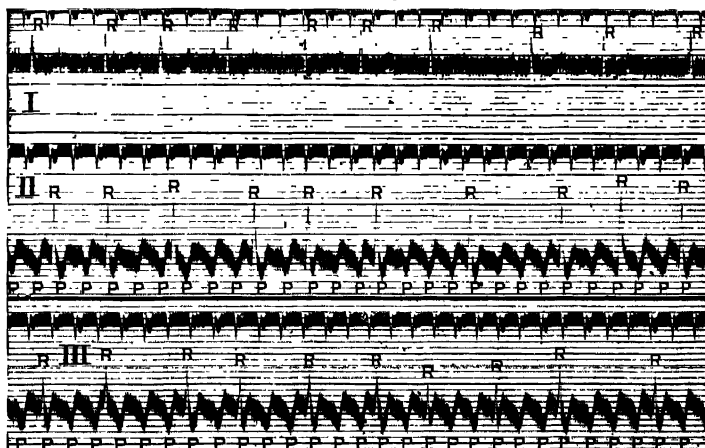


FIG. 76.—Electro-cardiogram from a case of auricular flutter. The auriculo-ventricular ratio is sometimes 2 : 1, at others 3 : 1, and at others again 4 : 1. The response of the ventricle to auricular contraction being at irregular intervals, there is irregularity of the ventricular rhythm.

VENTRICULAR FIBRILLATION.—The ventricular complexes are replaced by oscillations, occurring at very rapid and irregular intervals, and their form and size vary.

SINO-AURICULAR BLOCK.—In this condition there is an absence of both the auricular and ventricular complexes during an abnormally long pause. (See p. 904 and Fig. 77.)

AURICULO-VENTRICULAR BLOCK.—In the first grade (leads I and III of Fig. 78) there is merely an increase of the *P-R* interval, it exceeding 0.18

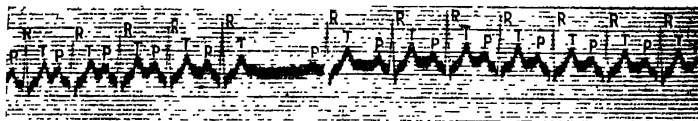


FIG. 77.—Electro-cardiogram showing an abnormally long pause due to sino-auricular block.

second. It may be increased to such a degree that *P* coincides with the preceding *T*.

In the second grade (lead II of Fig. 78) the *P* deflections are found at regular intervals and are of typical form. Sometimes the *P* deflections are not followed by ventricular complexes, the frequency of such depending

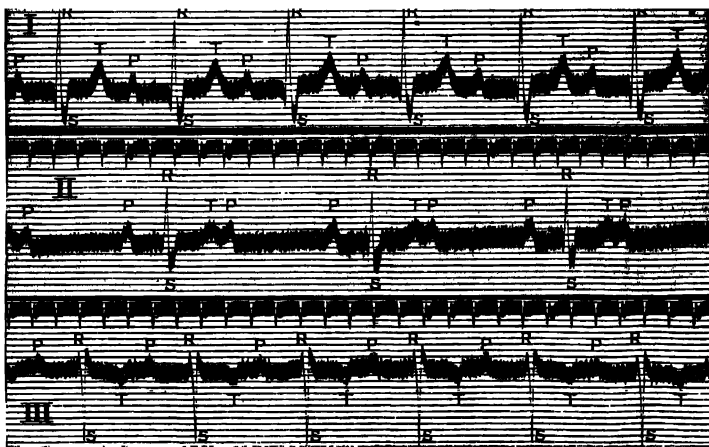


FIG. 78.—Electro-cardiogram showing partial heart-block. In each lead there is an increase of the *P-R* interval, and in lead II there is also continuous 2:1 rhythm, every other stimulus from the auricle failing to reach the ventricle. There is also inversion of *T* in lead II† and left-sided preponderance.

upon the degree of block, as described on p. 905. Unlike complete heart-block, on each occasion the ventricular complex is preceded by a *P* deflection. In the case of dropped beats, almost always there is a progressive increase of the *As-Vs* interval preceding and a progressive shortening of the interval following each dropped beat, so that the prolonged pause during a dropped beat is not equal to two regular pulse-beats. As the ventricular beats

are of supra-ventricular origin, the ventricular complexes are of typical form.

In complete heart-block (Fig. 79) the *P* deflections are found at regular intervals, and are of typical form. They are more frequent than the ventricular complexes. In addition, the time-relation between the *P* deflec-

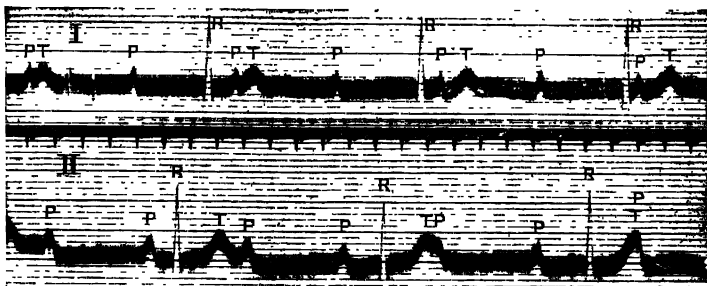


FIG. 79.—Electro-cardiogram of leads I and II showing complete heart-block, or dis-association of the auriculo-ventricular rhythm, the auricles and ventricles beating independently of each other.

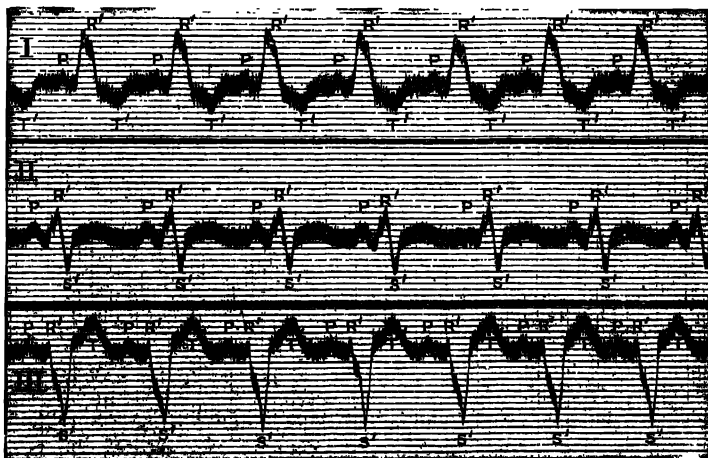


FIG. 80.— Electro-cardiogram showing a lesion of the left main branch of the auriculo-ventricular bundle (new nomenclature).

tions and the ventricular complexes is a constantly varying one, the former at one time preceding, at another following, and sometimes, again, coinciding with the latter. In the last case the *P* deflection is superimposed upon the ventricular complex. As the ventricular beats are of supra-ventricular origin, the ventricular complexes are of typical form. Sometimes there are superadded ventricular extra-systoles.

BUNDLE-BRANCH BLOCK.—A lesion of either of the two branches of the auriculo-ventricular bundle may be recognised by means of the electro-cardiograph (Figs. 80 and 81).

The ventricular complex is diphasic and of increased amplitude. The initial group of deflections (*Q*, *R*, *S*) is of increased duration, exceeding one-tenth second and comprising more than one-third of the whole complex, and usually exhibits pronounced or irregular notching. The terminal deflection (*T'*) points in the opposite direction to the initial group of deflections in leads I and III. In lead II, *Q*, *R*, *S* is usually of less amplitude and is often diphasic; *T'* may point in either direction. In the diphasic ventricular complexes of leads I and III there is usually no iso-electric period between *Q*, *R*, *S* and *T*. The *P*-*R* interval may be increased.

Two types of curves may be recognised: (1) The common type, in which

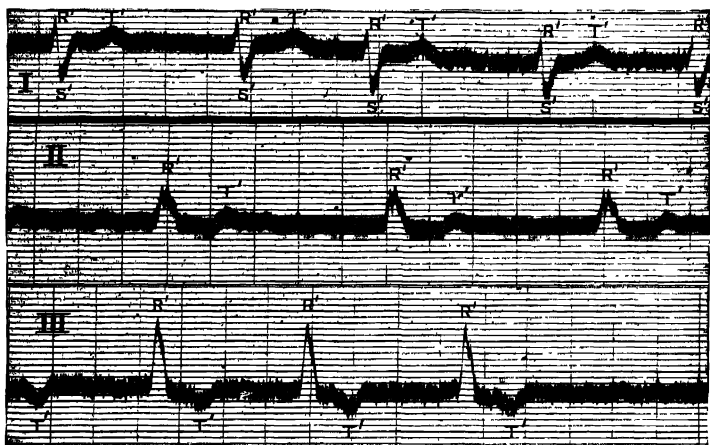


FIG. 81.—Electro-cardiogram showing a lesion of right main branch of the auriculo-ventricular bundle (new nomenclature). There is also auricular fibrillation.

there is a large *R'* in lead I and a large *S'* in lead III, *T'* pointing downwards in lead I and upwards in lead III. (2) The rare type, in which there is a large *S'* in lead I, and a large *R'* in lead III, *T'* pointing upwards in lead I and downwards in lead III. It was formerly supposed that the first type was indicative of right bundle-branch block, and that the second signified left bundle-branch block. It is now believed that the opposite is the case.

It is necessary to distinguish between a lesion of the left or right main branch (new nomenclature) and preponderance of the left or right ventricle respectively. The distinguishing features are that in bundle-branch block the initial group of deflections is of increased duration and usually exhibits pronounced or irregular notching, and *T* points in the opposite direction to the main initial deflection in leads I and III. As already noted, increased duration of the *Q*, *R*, *S* complex may be met with in extreme preponderance of either ventricle. But taking cases of preponderance as a whole, the

period of time is materially less. In addition, pronounced or irregular notching does not occur, and *T* is not of increased amplitude and, apart from exceptional cases, points in the same direction.

FUNCTIONAL BUNDLE-BRANCH BLOCK.—A condition, which is rare, in which there is increased duration of the *Q*, *R*, *S* group of deflections together

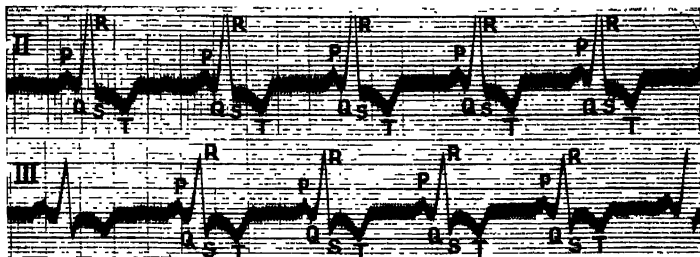


Fig. 82.—Electro-cardiogram showing functional bundle-branch block.

with a diminished *P*-*R* interval (Fig. 82) has been termed functional bundle-branch block.

ARBORIZATION OR INTRA-VENTRICULAR BLOCK.—The initial group of deflections is of increased duration and usually exhibits pronounced or irregular notching, and is of low voltage (Fig. 83). Some believe that occasionally the *T* deflection points in the opposite direction to the initial

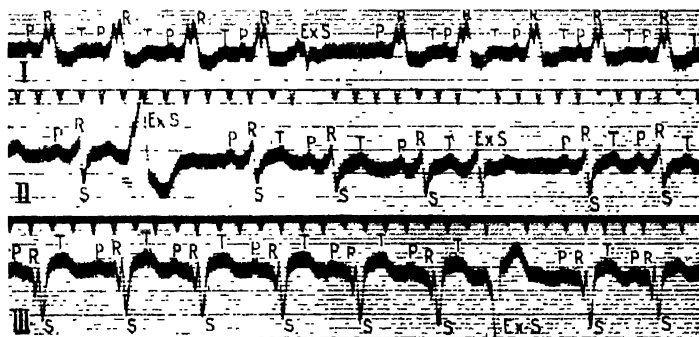


Fig. 83.—Electro-cardiogram showing arborization block. There is an extra-systole, marked Ex.S., in lead I, two extra-systoles in lead II, and one in lead III.

deflection in leads I and III. Even if such should be the case, its form is not so abnormal.

It should be pointed out that some writers are of opinion that the electro-cardiogram just described indicates a delay of the wave of excitation along the right or left main division of the auriculo-ventricular bundle, *i.e.* incomplete or partial bundle-branch block.

ALTERNATION OF THE HEART.—This may sometimes be recognised by means of the electro-cardiograph, by an alternation in the amplitude of the deflections due to the contraction of the ventricle (Fig. 84). Both the *R* and *T* waves may be affected, or one more than the other. It should be noted that alternation of the heart is sometimes shown in a sphygmogram without any corresponding evidence in an electro-cardiogram, while rarely the opposite holds good. It should be further noted that the alternation in a sphygmogram and electro-cardiogram does not always correspond; i.e. the smaller ventricular deflections correspond with the larger pulse-wave.

ANGINA PECTORIS.—Left-sided preponderance is usual. In a proportion of cases the ventricular complexes are abnormal. Most varieties of these

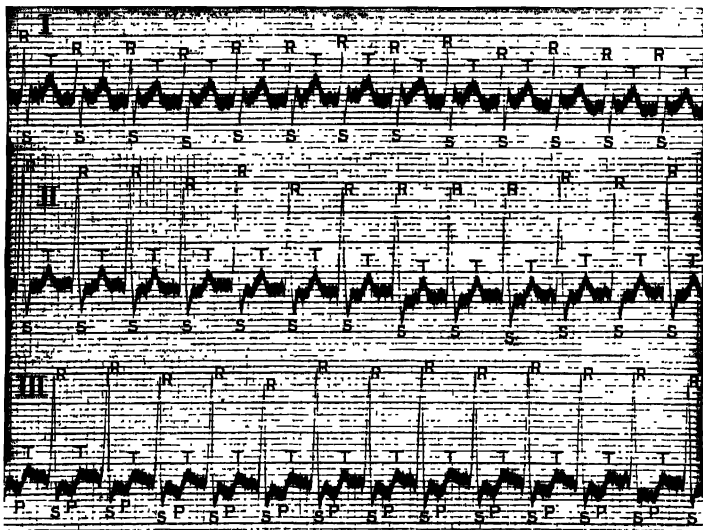


FIG. 84.—Electro-cardiogram from a case of paroxysmal tachycardia. There is alternation in the amplitude of the *R* deflections.

abnormalities may be met with, including flattening or inversion of *T* in lead I or II, or both; increased duration, and notching of the *Q*, *R*, *S* group of deflections; bundle-branch block; and a large *Q* deflection in lead III (see p. 1025). There may be extra-systoles and some degree of heart-block. Other abnormalities of rhythm are exceptional. The changes in the ventricular complexes may be of considerable value in doubtful cases. Negative findings, on the other hand, are of no importance.

Transient modifications of the electro-cardiograms similar to those of coronary occlusion with infarction have been observed in some cases of angina pectoris during the attacks. Such are of much diagnostic significance.

CORONARY OCCLUSION WITH INFARCTION OF THE HEART.—Electro-cardiograms of this disease are usually characteristic and of great diagnostic value. They are as follows:

Within a few hours there is usually a deviation of the *R-T* or the *S-T* segment. This portion of the curve commences from the *R* or *S* deflection either above or below the iso-electric level and proceeds in a more or less horizontal direction, resulting either in a plateau-shaped elevation or in a depression respectively. This alteration is generally most noticeable in

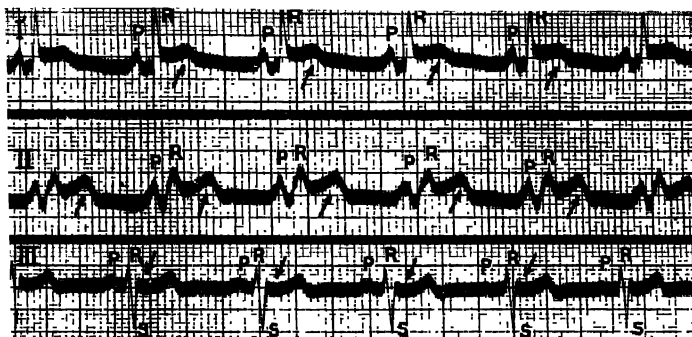


FIG. 85.—Electro-cardiogram from a case of the T^1 type of infarction of the heart taken early after the onset of symptoms. The *R-T* portion of the curve commences above the zero level in leads I and II, and slightly below the zero level in lead III, as indicated by arrows.

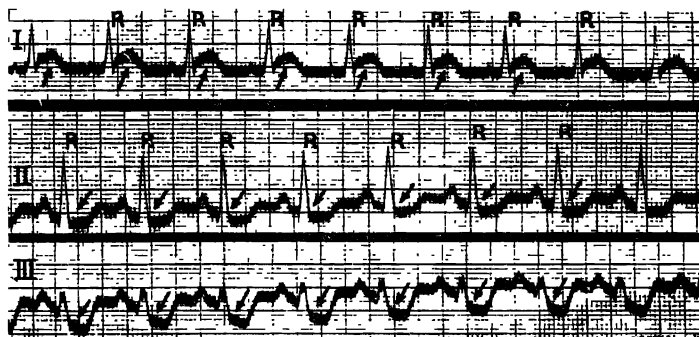


FIG. 86.—Electro-cardiogram from a case of the T^1 type of infarction of the heart taken early after the onset of symptoms. The *R-T* portion of the curve commences above the zero level in lead I, and below the zero level in lead III and rather so in lead II, as indicated by arrows.

leads I and III. In this event, the corresponding portions of the curves in these leads are divergent. Thus, if there is *R-T* elevation in lead I there is *R-T* depression in lead III (Figs. 85 and 86); and *vice versa* (Figs. 87 and 88). Sometimes the deviation of the *R-T* segment is best observed in leads III and II, or in leads I and II; or the alteration may be present in one lead only. The foregoing features are perhaps pathognomonic of the condition.

After a few days or more, the *R-T* portion of the curve gradually returns to the iso-electric level, and the *T* deflections gradually reappear. The latter assume a direction opposite to that to which the *R-T* segments were



FIG. 87. —Electro-cardiogram from a case of the T^3 type of infarction of the heart taken early after the onset of symptoms. The *R-T* portion of the curve commences above the zero level in lead III and slightly so in lead II, and below the zero level in lead I, as indicated by arrows.

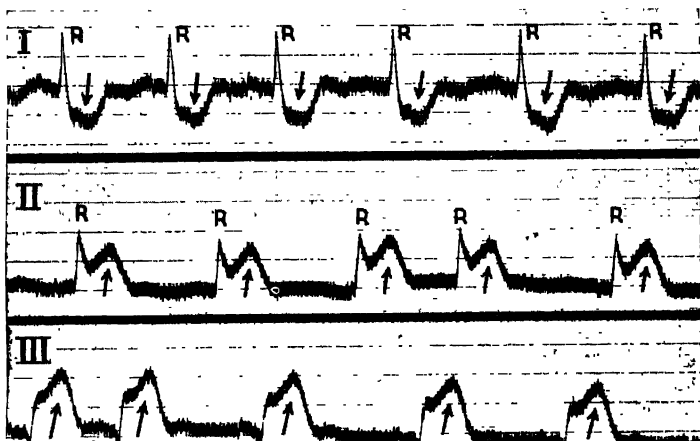


FIG. 88. —Electro-cardiogram from a case of the T^3 type of infarction of the heart taken a few hours after the onset of symptoms. The *R-T* portion of the curve commences below the zero level in lead I and above the zero level in leads II and III, as indicated by arrows.

previously deviated. Thus, inversion of the *T* deflections follows elevation of the *R-T* segments, and upright *T* waves follow depression of the *R-T* segments. (Figs. 89-91.) The *T* deflections are usually sharply defined, and their amplitude is often large. The *R-T* intervals preceding the

altered *T* waves frequently exhibit convexity or concavity. The foregoing features are not so characteristic as are those of the first stage, but, taken together with the clinical features, they afford strong corroborative evidence of the disease.

In both stages, sometimes there is diminished amplitude and increased duration of the initial group of ventricular deflections (*Q*, *R*, *S*), and the latter is generally associated with notching or splintering.

There are various types of curves, the two commonest being: (1) Lead I shows *R-T* elevation and, later, inversion of the *T* deflections; and in lead III, the *R-T* segments are depressed, with the subsequent development of upright *T* waves. (Figs. 85, 86 and 89). (2) Lead III shows *R-T* elevations and, later, inversion of the *T* deflections; and in lead I, the *R-T* segments are depressed, with the subsequent development of upright *T* waves (Figs. 87, 88, 90 and 91).

The first type, which is the more common, is associated with occlusion

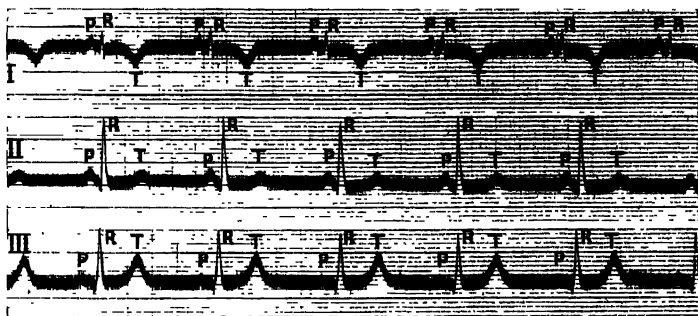


FIG. 89.—Electro-cardiogram from a case of the T^1 type of infarction of the heart. The *T* deflections in lead I are inverted, and are rather sharply defined and of increased amplitude. Those in lead III are upright, and are sharply defined and of increased amplitude.

of the descending branch of the left coronary artery and infarction of the wall of the left ventricle near the apex, especially the anterior part, and the adjacent part of the interventricular septum; and is termed the T^1 type. The second is associated with occlusion either of the right coronary artery or the circumflex branch of the left and infarction of the posterior wall of the left ventricle near the base; and is designated the T^2 type.

In both types of curves the *T* deflections in lead II are often slightly inverted or flattened.

Some changes in the *T* deflections towards the normal usually supervene within a few weeks or months, and ultimately the *T* deflections in all leads may become normal, but sometimes those in one lead remain inverted and sharply defined, and even these changes are very suspicious.

A large *Q* deflection in lead III (see Figs. 90 and 91) is often a noticeable feature of infarction of the posterior wall of the left ventricle, either recent or old. It is to be noted, however, that it may also occur in other conditions (see p. 1003).

Lead IV is particularly valuable in occlusion of the descending branch of the left coronary artery, *i.e.* the T^1 type. This lead alone may be affected, while in some cases the changes are more evident, or occur earlier and last longer than in the three other leads. They resemble those of lead I of this type. Thus, there is elevation of the $R-T$ intervals, and subsequently in-

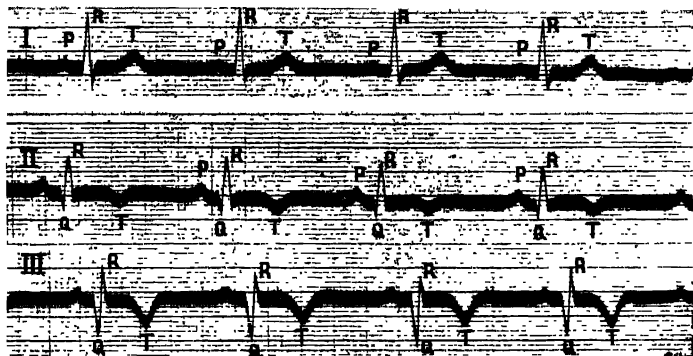


FIG. 90.—Electro-cardiogram from a case of the T^3 type of infarction of the heart. The T deflections in lead II are inverted. Those in lead III are inverted, and are sharply defined and of increased amplitude. There is a large Q in lead III.

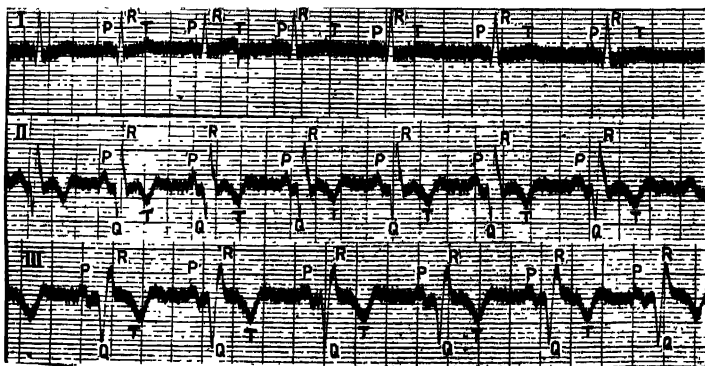


FIG. 91.—Electro-cardiogram from a case of the T^3 type of infarction of the heart. The T deflections in leads II and III are inverted, and are sharply defined and of increased amplitude. There is a large Q in leads III and II.

verted T waves. (Figs. 92 and 93.) Lead IV is of less value in infarction of the posterior wall of the left ventricle, *i.e.* the T^3 type. There may be no changes, or if present they are less marked. If there are changes, they generally resemble lead I of this type. Thus, there is depression of the $R-T$ intervals and, later, upright T deflections. The latter are of increased amplitude, and may be huge. (Fig. 94.)

The foregoing changes in the electro-cardiograms of coronary occlusion with infarction of the heart are not constant. But they are frequent, and when they do occur are of great diagnostic value. It is necessary to point out that it is the successive changes in the curves which are especially

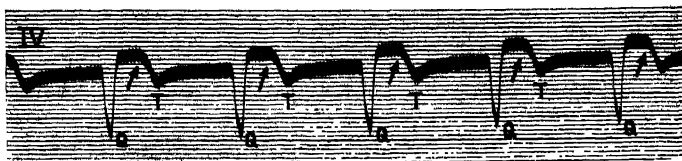


FIG. 92.—Electro-cardiogram of the fourth lead from a case of the T^1 type of infarction of the heart. There is R - T elevation, as indicated by arrows, and inversion of T . There is also a large Q .

important. For this reason, serial records taken over a period of time are of much greater value than a single one, for in cases in which the latter does not reveal the characteristic changes, later curves may do so. It is unusual to find an absence of these changes during the whole of the first two weeks



FIG. 93.—Electro-cardiogram of the fourth lead from a case of the T^1 type of infarction of the heart. There is slight R - T elevation, and the T deflections are inverted, sharply defined and of increased amplitude. There is also a large Q .

after the onset of symptoms, though transient changes may have disappeared in later electro-cardiograms.

In conclusion, it is to be noted that a similar deviation in the R - T (or S - T) interval has also been recorded in rheumatic carditis, pericardial

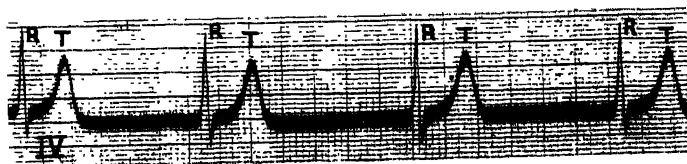


FIG. 94.—Electro-cardiogram of the fourth lead from a case of the T^3 type of infarction of the heart. The T deflection is positive, sharply defined and huge.

effusion, uræmia, and pneumonia. The clinical features of these conditions, however, do not resemble those of coronary occlusion.

It has been pointed out that transient changes in the electro-cardiograms similar to those of coronary occlusion have been observed during attacks of angina pectoris.

Low Voltage.—The term low voltage is used when no part of the *Q*, *R*, *S* complex exceeds five millimetres in the three leads. It is almost always of

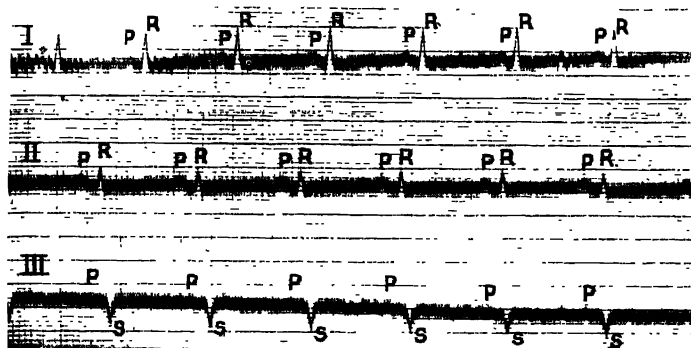


FIG. 95.—Electro-cardiogram from a case of hypothyroidism. The *T* deflections are absent in the three leads; and there is also diminished amplitude of the *Q*, *R*, *S* complex.

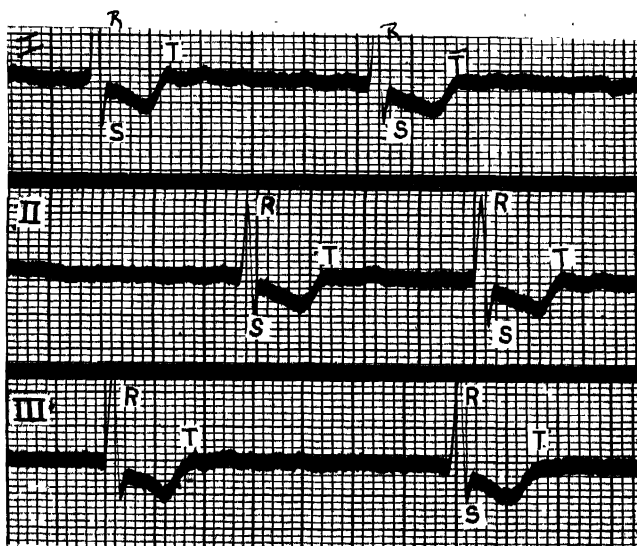


FIG. 96. — Electro-cardiogram from a case of auricular fibrillation fully under the influence of digitalis.

pathological significance, and may be met with in marked chronic myocardial disease, severe congestive failure from any cause, coronary occlusion, pericardial effusion, and hypothyroidism. In the last named there are also

changes in the other deflections (see below). As stated on p. 1021, low voltage is one of the various features found in arborization or intraventricular block.

HYPOTHYROIDISM.—The *T* deflections are flattened or inverted in the three leads; and frequently there is also diminished amplitude of the *Q*, *R*, *S* complex. See Fig. 95.

THE EFFECT OF DIGITALIS.—Electro-cardiograms of patients fully under the influence of digitalis may show depression of the *R-T* interval with flattening or inversion of the *T* deflections (see Fig. 96). This should be distinguished from the *R-T* deviation due to coronary occlusion. In the former the corresponding portions of the curves always point in the same direction; while in the latter usually they point away from each other in leads I and III.

FREDERICK W. PRICE.

DISEASES OF THE BLOOD VESSELS

The term arterio sclerosis, which means no more than arterial hardening, has led to much confusion as it has been employed by various authors in different senses to describe different pathological conditions of the arteries. Thus, it has been used to comprise the following conditions:

(a) Diffuse hyperplastic sclerosis, characterised by intimal hyperplasia and hypertrophy of the media;

(b) Degeneration of the middle coat of the artery, known as Mönckeberg's medial sclerosis, in which lime salts are deposited more or less symmetrically in rings around the artery; and

(c) Arterial degeneration of the intima, known as atheroma, or, because of the tendency to calcification, as atherosclerosis.

Or it is confined to the first and second. Or it is restricted to the first. Or it is employed to include the three mentioned above and, in addition, arterial inflammation, which when chronic is most often the result of syphilis but may be caused by other infections. Or, finally, it is employed by a few writers to include even other pathological conditions, such as infiltration. It is, therefore, clear that the term arteriosclerosis should not be used except in a very general sense.

It is proposed to adopt the following classification of diseases of the arteries:

- (i) Arterial inflammation
 - (a) Acute arteritis
 - (b) Chronic arteritis
 - (c) Thrombo-angiitis obliterans
 - (d) Polyarteritis nodosa or periarteritis nodosa
- (ii) Diffuse hyperplastic sclerosis
- (iii) Mönckeberg's medial sclerosis
- (iv) Fatty Degeneration of the Media
- (v) Atheroma (Atherosclerosis)
- (vi) Other Degenerations allied to atheroma
- (vii) Arterial infiltration.

And the following will be dealt with separately : (1) The syndrome of intermittent claudication, caused by several of the foregoing pathological conditions ; (2) Aneurysms, whether (a) saccular, most frequently caused by syphilis ; (b) aneurysmal dilatation, most often due to atherosclerosis ; or (c) the less common forms of aneurysm.

ARTERIAL INFLAMMATION

The arteries may be infected from their intima, either by micro-organisms settling on the surface or by the arrest of an infective embolus within the lumen. They may also be infected by micro-organisms reaching the media or adventitia through the vasa vasorum or by direct inward spread of inflammation from the surrounding tissues.

ACUTE ARTERITIS

Acute arteritis was formerly described as a common event in many diseases, the staining of the intima being mistaken for inflammation. Acute inflammation of the arteries is, however, a rare disease, and is usually met with as a complication in the acute infections. The intima of the aorta may be infected in cases of septicæmia and pyæmia, and most commonly in cases of progressive septic endocarditis, when the organisms usually found are streptococci. Vegetations may be seen upon the intima and the inflammation rapidly involves the subjacent coats. Occasionally the aorta may be infected in a septicæmia or pyæmia, through embolism of the vasa vasorum, or the ascending aorta may be infected by spread through the vessel wall from a pericarditis. The wall of the aorta may rupture, or an aneurysm be formed, but this is rare. Acute multiple arteritis is most frequently seen as a sequel of typhoid fever, but cases have been observed after small-pox, scarlet fever, influenza, and pneumonia. In many cases the organisms of the disease have been found in the vessel wall.

Symptoms.—The symptoms depend upon the vessels affected. In the case of the femoral artery, there may be severe pain in the course of the vessel with sometimes redness and swelling in the part affected. The pulse below is obliterated. The limb becomes pale and cold, and then livid. Gangrene may or may not follow ; it depends upon the rapidity with which the vessel is blocked. In some cases where the onset is severe and the symptoms suggest that gangrene will follow, the circulation improves and colour returns to the limb. In other cases, several of the arteries may be infected at the same time, with high fever and symptoms of an acute infection. Some cases of cerebral thrombosis in which a young or middle-aged patient makes a good recovery and lives for twenty or thirty years without any other vascular catastrophes are probably of this type.

Prognosis and Treatment.—In acute arteritis treatment, other than rest and general and local measures for the relief of pain, is of little avail. The condition is a very severe one. If the artery involved is a large one, every effort should be made to avoid infectious gangrene, and in some cases a surprising return of circulation may be observed.

CHRONIC ARTERITIS

Ætiology.—Acquired syphilis is by far the most common cause of chronic arteritis, and less frequently congenital syphilis. Tuberculous endarteritis is not uncommon in the small pulmonary arteries and in the arteries of the brain in tuberculous meningitis. Endarteritis obliterans may also be caused by infection with pyogenic organisms of a subacute or chronic type. Moreover, changes in the adventitia of the small arteries are also found in polio-encephalo-myelitis and in encephalitis lethargica.

Pathology.—Chronic arteritis is a focal affection, and is found in muscular and elastic arteries of all calibres. It is common in the aorta and large elastic arteries, and it also frequently attacks the small arteries. The large muscular arteries, however, are but rarely affected by syphilis. Chronic inflammation of the arteries has been divided into—(1) Endarteritis, where the intima is affected; (2) mesarteritis; and (3) periarteritis, where the external coat is involved. In the great majority of cases of inflammation of the smaller arteries all coats are involved. The muscular and elastic fibres tend to be destroyed and this may result in direct rupture. The changes in the intima are very conspicuous. Its layers become very much thickened by inflammatory infiltration and proliferation. In the early stages round cells are seen, and later spindle-shaped fibroblasts, definite granulation being thus formed, while in cases of syphilis plasma cells and eosinophil leucocytes are often present. The result of this thickening in such small arteries is to narrow the vessel, and the condition is often termed endarteritis obliterans. The lumen may finally become completely blocked, leading in the brain to cerebral softening, and in other tissues to fibrosis. The adventitia is also greatly thickened in chronic syphilitic arteritis and consists of inflamed tissue infiltrated by lymphocytes, plasma cells, and occasional eosinophil leucocytes.

Syphilis of the aorta or *syphilitic mesaortitis* is a focal inflammation, but it may implicate almost the whole length of the aorta. The inflammation extends from the adventitia. The vasa vasorum proliferate and in most cases pass into the intima. About these vessels is a zone of granulation tissue, usually consisting only of plasma cells, lymphocytes, eosinophil leucocytes, and fibroblasts, but occasionally there are gummata with giant cells. Endarteritis of the vasa vasorum is found only in the more intense reactions. The elastic fibres of the media are completely destroyed in the areas of granulation tissue; occasionally the media is necrosed between areas of granulation tissue. The *Sp. pallida* has been demonstrated in the lesions. The intima of the aorta is usually thickened over the areas of inflammation, and this thickening has been in the past confused with the degeneration of the intima that we know as atheroma. The inflammatory thickening of the intima due to syphilis can, in its earlier stages, be distinguished by the naked eye from atheromatous thickenings by its sharper demarcation, pearly colour, rubber-like consistency, crenated outline, pitted surface, and freedom from fatty degeneration. The weakening of the vessel wall on account of the replacement of the middle coat frequently results in dilatations, varying in size from minute stellate patches to large aneurysms. The scarring and pitting are due to fibrous tissue replacing the inflamed media and are characteristic. In the later stages atheroma usually occurs

in the thickened intima over the areas of inflammation; also syphilitic mesaortitis and primary atheroma may co-exist, especially in later life.

The *coronary* circulation is often found affected in these cases, with the result that there are attacks of angina pectoris. Owing to the mesaortitis of the ascending aorta, the orifices of the coronary arteries become narrowed or blocked, but the disease does not spread far down the arteries, in this way providing a striking contrast with atheroma. It may cause extensive necrosis or fibrous patches in the myocardium, and the heart is not uncommonly enlarged. Frequently the aortic ring has expanded, or the aortic valves have become involved by the syphilitic inflammation, so that aortic regurgitation is a common sequel.

The *arteries of the brain* are frequently involved by syphilis, which causes endarteritis obliterans and consequent cerebral softening. This change is also present in syphilitic meningitis, where the syphilitic inflammatory reaction is more intense. The kidneys are but rarely affected, though occasionally gummata are found. The eyes are not infrequently attacked and a condition of syphilitic choroido-retinitis may be seen on ophthalmoscopic examination. In other organs, such as the liver and testicle, gummatous necrosis, followed by fibrous changes, is found. In rarer instances the trachea and lungs are affected.

Symptoms.—The symptoms that result from syphilitic vascular disease depend upon the organ affected.

In the aorta, aneurysm is a common result from syphilis. Aortic regurgitation, with its usual effect on the heart, is even more common. The symptoms of necrosis or fibrosis of the myocardium are severe anginal pains, often resulting in sudden death. Occasionally the heart muscle may give way, with the usual signs of chronic cardiac failure. If the brain is affected by arterial thrombosis, the symptoms depend upon the area affected; hemiplegia, aphasia, and hemianopia may all result. Albuminuria is quite uncommon in syphilitic vascular disease. Iritis may be present, and more commonly choroiditis, in which white patches surrounded by pigmented areas give a striking appearance to the fundus oculi. The optic disc is often white and atrophied. In syphilitic meningitis, œdema of the optic papilla is often present. The radial artery is practically always normal to the touch in cases of syphilitic vascular disease.

Course.—The course of syphilitic arterial disease is very variable. The symptoms resulting from endarteritis and subsequent thrombosis in medium-sized arteries such as the cerebral, are generally present in the acute secondary stage, from 2 to 5 years from the date of infection, but the vascular disease may exist with exacerbations all through life, and the important and characteristic cardiovascular changes in the first part of the aorta generally follow 15 to 25 years after the original infection.

Prognosis.—The prognosis in cerebral syphilitic vascular disease is on the whole good, if the condition be taken early enough and treated properly. It has to be remembered, however, that vascular syphilis is often complicated by parenchymatous syphilis, where the spirochætes are not only found in the walls of the vessels, but also in the cerebro-spinal tissues themselves, and this complication certainly increases the gravity of the condition. In the later cardiovascular syphilis the prognosis is bad unless the diagnosis is made early before any serious cardiac symptoms have developed.

Treatment.—Prophylaxis is of the greatest importance in syphilitic vascular disease. With regard to treatment, in the acute stages of syphilitic vascular disease it is generally thought that mercury is the best drug to use. Mercurial inunctions or soluble intramuscular mercurial injections should be given for 2 or 3 weeks before any intravenous injections are prescribed. Moreover, iodides should be given by the mouth, to absorb as far as possible the inflammatory products in the intima of the vessels. The indiscriminate use of arsphenamine and neoarsphenamine in the acute stages of syphilitic vascular disease has been followed by most disastrous results, their administration causing further swelling of the intima and further blocking and thrombosis of the small vessels. After mercury and iodides have been given for 3 or 4 weeks, neoarsphenamine, should be administered cautiously in small doses and gradually increased.

THROMBO-ANGITIS OBLITERANS

This name was suggested by Buerger in 1908 for a disease characterised by acute inflammation of the deep arteries and veins, and sometimes by a migratory inflammation of the superficial veins in the extremities. Thrombosis develops and the vessels become occluded.

Ætiology.—The malady is found most commonly in middle-aged male Hebrews. The cause is unknown, but the pathological changes suggest that it is due to an infection. Syphilis is usually not present, the Wassermann reaction being negative. Excessive tobacco smoking has been suggested as a predisposing factor.

Pathology.—The deep vessels of the arms and legs, especially the latter, are occluded by thrombosis in various stages of organisation; their walls are traversed by vessels, and show a little inflammatory infiltration. In about a quarter of the cases this is associated with a migratory phlebitis in the superficial veins of the limbs. The condition is characterised by extensive progressive thrombosis, with organisation, with little inflammation of the vascular coats.

Symptoms.—The onset is gradual with pains in the feet and toes. The patient is unable to walk for more than a few minutes without severe cramp-like pain in the legs (intermittent claudication, see page 1041). The thrombi in the superficial veins, when they occur, are also very tender. Redness of the extremity, especially when in a dependent position, is often noted, while blanching occurs when the limb is raised. Diminution or loss of pulsation in the arteries, such as the radial or dorsalis pedis, is often present. In the later stages, the cramp-like pain becomes intense, and the disability in walking often leads to marked mental depression. Trophic changes appear in the skin, with gangrene, and fissures and ulcers may occur.

Diagnosis.—(1) Raynaud's disease more often attacks females; the upper extremities are most affected; and X-rays show marked atrophy of the bones of the hands. This is not present in Buerger's disease. (2) In erythromelalgia the limbs become red and flushed, but the arteries pulsate forcibly; gangrene does not occur. (3) In gangrene due to Mönckeberg's sclerosis, the calcified arteries may be well seen by means of the X-rays.

Prognosis.—The course of the disease varies. Some cases progress

rapidly, while others last for years. Gangrene may require high amputation of the limb.

Treatment.—As the cause is not known, there is no specific treatment. With regard to drugs, iodides and glyceryl trinitrate are used. Gentle massage is useful, and diathermy sometimes helps. Ultra-violet therapy has been tried. Buerger recommends *passive postural exercises*, and employs the following method: "The affected limb is elevated, with the patient lying in bed, to from 60 to 90 degrees above the horizontal, being allowed to rest upon a support for from thirty seconds to three minutes, the period of time being the minimum amount of time necessary to produce blanching or ischæmia. As soon as blanching is established, the patient allows the foot to hang down over the edge of the bed for from two to five minutes, until reactionary hyperæmia or rubor sets in, the total period of time being about one minute longer than that necessary to establish a good, red colour. The limb is then placed in the horizontal position for about three to five minutes, during which time an electric heating pad or a hot-water bag is applied, care being taken to prevent the occurrence of a burn. The placing of the limb in these three successive positions constitutes a cycle, the duration of which is usually from six to ten minutes. These cycles are repeated over a period of about one hour, some 6 to 7 cycles constituting a séance" (Leo Buerger, *The Circulatory Disturbances of the Extremities*). Others claim that the maintenance of a high venous pressure and stimulation of the heart are the most effective lines of treatment. Pain is relieved by rest in the recumbent position, and heat applied to the painful limb by electric pads or electric light baths is also useful. Ulcers of the legs should be treated surgically.

POLYARTERITIS NODOSA OR PERIARTERITIS NODOSA

Polyarteritis nodosa is a rare complaint characterised by prolonged fever and the occurrence of nodular swellings, and in some cases aneurysms of the medium-sized arteries. It may affect arteries in almost any part of the body, producing a very diverse symptomatology.

Ætiology.—Young adults are most commonly affected, and males more often than females. The cause is unknown. The Wassermann reaction is negative, but the pathological changes and course of the disease suggest an infectious agent.

Pathology.—The medium-sized arteries are usually affected, especially those of the heart, kidneys, and intestines. A remarkably focal acute inflammation extends through all the coats of the artery with hyaline or fibrinoid necrosis of the arterial wall, and within the lumen are often found thrombi, which may become organised. Aneurysmal dilatation is usually present. There may be many polymorphonuclear leucocytes and some mononuclear cells, lymphocytes, and plasma cells; eosinophil cells are a characteristic but inconstant finding. White or yellowish-white nodules, from the size of a pin's head to that of a pea, can be seen on the arteries. Owing to the alteration in the lumen of the vessels, necrosis and infarcts occur in the organs supplied.

Symptoms.—The disease may start in almost any way, even with bronchial catarrh or with epigastric pain. There is tachycardia and irregular fever, with marked prostration. Some cases may start as an acute illness, simulating nephritis or rheumatic carditis; in others long continued malaise is the main

presenting symptom. Acute abdominal pain may be caused by disease of the mesenteric arteries—indeed, even perforation of the intestine and peritonitis have followed. If the arteries of the heart are involved, evidence of myocardial disease will be present; and when the kidneys are affected, blood and casts appear in the urine. Occasionally bronchial asthma, cough and hæmoptysis have been noted. Later, in a small proportion of cases, nodular swellings, varying in size up to a pea, may be felt in the subcutaneous tissues of the abdomen, thorax, and limbs. Examination of blood shows the anæmia and a moderate leucocytosis. Blood cultures are sterile.

Diagnosis.—This is extremely difficult, owing to the variable symptoms displayed. In most cases, a pyrexial infection, of unknown origin, has its nature revealed only post-mortem. Rarely, however, where a node was felt in the subcutaneous tissues and excised during life, the diagnosis has been made before death. The diagnosis should now be made more often if it is suspected in any obscure and varied illness in which there is some evidence of arterial involvement.

Prognosis.—It has been said that in most cases death occurs within a few weeks to a few months after the onset of symptoms. Now, however, when more cases are recognised, it is found that many recover and that the disease may run an intermittent course for many years.

Treatment.—This is the same as that of any acute infection. Arsenic, mercury, and quinine should all be tried, though the results up to now have not been encouraging. In the main the treatment should be symptomatic as the parts involved are so varied.

DIFFUSE HYPERPLASTIC SCLEROSIS

The diffuse hyperplastic sclerosis of Jores and Evans, formerly known as arterio-capillary fibrosis of Gull and Sutton, is characterized by intimal hyperplasia, especially of the smaller arteries and arterioles, and hypertrophy of the media, particularly of the medium-sized arteries.

Ætiology.—Diffuse hyperplastic sclerosis is common in late middle age, and is by no means a scule change. In the great majority the causes are those of hyperpiesia (see p. 1065). The affection may also be due to gout and lead poisoning, and chronic nephritis. In rare cases associated with chronic interstitial nephritis, it may occur in young children. It is more common in males than in females. The inherited constitution is of great importance as shown by the high familial incidence in certain cases. Syphilis has no part in the causation of this condition, though, of course, it may be present in syphilitic cases.

Pathology.—The condition is widespread, frequently involving the whole arterial system. It affects the smaller arteries and the arterioles, and the medium-sized arteries. The kidneys and spleen are most commonly affected, especially the first; the next most frequently, the brain; while the pancreas, liver, suprarenal glands, stomach and intestines are less often implicated. The characteristic lesion consists of a cellular proliferation of the intimal cells and increase of hyaline material. At a later stage there is fatty degeneration in the arterioles alone. The thickening of the intima may lead to obliteration of the lumen of the vessels. There is hypertrophy of the media of the medium-sized arteries, *e.g.* the radials, with little or no

change in the intima. In the case of the smaller arteries and arterioles, a patchy ischæmic fibrosis of certain organs, *i.e.* the kidneys and brain, may take place. Hæmorrhages may also occur.

There may be co-existent atheroma, *e.g.* of the cerebral and coronary arteries, and, it may be, of the aorta, due to age or, in the opinion of some, the result of the hypertension.

Symptoms.—In diffuse hyperplastic sclerosis the vessel wall may be felt to be uniformly thickened—the so-called “whip-cord” artery. The degree of hardening of the arteries is found on palpation to vary at different times. The artery feels hardest when the vessel is most contracted and consequently smallest. As the muscular arteries are those chiefly affected, the radial, brachial, and temporal arteries are involved. The systolic blood-pressure in cases of simple hyperpiesia may be 160–240 mm. or more; it may reach even 300 mm. in cases associated with chronic interstitial nephritis. Symptoms of cardiac hypertrophy are common in the early stages, but in favourable cases they may not appear for years. In some cases cardiac hypertrophy is followed by dilatation, with its attendant symptoms. Headache, of a throbbing and bursting character, and generally in the occipital region, is an early symptom of arterial hypertrophy, and giddiness and fullness in the head are frequently complained of. Transient paralysis may occasionally be met with, and this has been attributed to spasm of the hypertrophied arteries. Later on, cerebral hæmorrhage may occur, with the production of hemiplegia. Albuminuria and casts in the urine may be found, while profuse renal hæmorrhage may occur. Uræmic symptoms do not occur frequently and then not till late unless the condition is secondary to chronic nephritis. Gastro-intestinal symptoms are often present. The patient may first complain of dyspepsia. A chronic diarrhoea without obvious cause in an elderly man should lead to a careful examination of the arteries and kidneys. Bronchitis and emphysema may mask a cardiovascular hypertrophy, and the enlargement of the heart may be overlooked unless there is a careful examination of the blood-pressure. The changes in the fundi are numerous and characteristic, and have been discussed in the section on hypertension (see pp. 1066, 1067). Intermittent claudication is not uncommon in these cases.

Prognosis.—The prognosis depends very largely on the degree of renal involvement. In hyperpiesia the condition may last for many years and only be terminated by hæmorrhage into the brain, the ischæmic fibrosis of the kidney, which accompanies it, being of no clinical importance. If, however, there is evidence that the cardiovascular hypertrophy and high blood-pressure are complicated by true nephritis, the condition is a grave one and uræmia may ensue; and when well-marked albuminuric retinitis is present death usually occurs within six months, though very rare cases have been recorded where a certain amount of ocular change has persisted for years.

Treatment.—The first indication is to remove the cause of the condition as far as possible. As a rule the most important thing is the regulation of the patient's life, the removal of anxiety and the reduction of his work and activity to a reasonable amount. The reduction of alcohol and tobacco is often needed, and it is the average daily consumption that counts in this direction. Any gouty tendencies should be corrected by the administration of alkalis and intestinal antiseptics, such as sodium benzoate. All sources of focal sepsis, whether in tonsils, teeth, nasal sinuses, or genito-urinary tract,

should be carefully sought for and, if found, removed. Moderation in food and drink, with regular exercise and care in promoting diaphoresis, is essential. Turkish and vapour baths may be given cautiously. The administration of a purgative pill once a week, and the use of a mild saline purgative each morning are indicated ; while in acute crises the abstraction of one pint of blood from the arm has often saved life.

MÖNCKEBERG'S MEDIAL SCLEROSIS

This form of degeneration is accompanied by a deposition of lime salts in the middle coat.

Ætiology and Pathology.—The cause is undoubtedly a senile degeneration of the elastic tissue and the muscle of the large muscular arteries, and a deposition of masses of lime salts in the dying tissue. It has no relation to syphilis. This degeneration has been caused experimentally in animals by a great variety of toxins. In man the causation is obscure, but it is common in diabetes and in old people. The lime salt is deposited, more or less symmetrically, within the media in plaques, which encircle part or all of the lumen. The affection is very common in the arteries of the leg below the bifurcation of the femoral ; occasionally the radial and ulnar arteries are affected ; rarely the aorta.

Symptoms.—The symptoms are coldness and œdema of the legs, as the result of defective circulation through them, and finally, and not uncommonly, gangrene results, this form of degeneration being usually present in senile and diabetic cases. The arteries feel like pipe stems, and sometimes crackle when rolled beneath the finger. They can be well seen by means of the X-rays.

Prognosis.—This depends partly upon the amount of gangrene present and partly upon the associated conditions.

Treatment.—It is clear that if amputation has to be undertaken a local amputation is of little value, and the limb should be amputated above the knee, as the arterial degeneration almost always extends to the bifurcation of the femoral artery.

FATTY DEGENERATION OF THE MEDIA

Fatty degeneration of the media occurs commonly in all arteries. *It tends to occur in cases of high blood pressure, and is consequently a common and important secondary complication of diffuse hyperplastic sclerosis.* It is probably the result of toxins or lack of nourishment, and is frequently present in cases of severe anæmia. It also occurs in old age, and is associated with cardiovascular hypertrophy. The muscle fibres become lost, being replaced by fibrous tissue. The vessel wall usually becomes weakened and is liable to rupture.

ATHEROMA

Synonyms.—Atherosclerosis ; Degeneration of the Arterial Intima.

Definition.—Atheroma is a variety of arterial degeneration which affects

and is almost confined to the intima. It is characterised by the accumulation of debris, which is at first fatty and later becomes impregnated with lime salts. The Greek word was used by Galen to signify a swelling full of gruel-like material.

Ætiology.—There is no doubt that atheroma is found more frequently and is more widespread with advancing age. Long life is a question of the blood vessels, and it has been well said that a man is only as old as his arteries. The quality of the arterial tissue that has been inherited may be poor, and a tendency to the development of atheroma at about the same age is often seen in all the members of certain families, thus showing the influence of heredity in the production of the condition. Atheroma also results from the amount of wear and tear to which the vessels have been subjected. It appears as a secondary change in cardiovascular hypertrophy, so that it tends to be more frequent in patients with hypertension than in other subjects of the same age. The affection is much more common in men than in women, and in subjects who have hard and prolonged mental or physical work. Moreover, it is much more common in the aorta than in the pulmonary artery, and when it does occur here it is nearly always associated with high pulmonary pressure, *e.g.* in mitral stenosis and in pulmonary fibrosis. The severity of the disease increases with the length of time during which the high blood-pressure has existed. Over-eating and stress and strain of modern life are probably factors in the ætiology of the condition. It has been said that the most important cause of atheroma is chronic poisoning. Acute degeneration of the media has been found after typhoid fever in young people, and has been caused experimentally by the injection of bacterial toxins. Chronic lead poisoning and gout are also ætiological factors. On the other hand, syphilis has no connection with atheroma, though the condition of the aorta known as syphilitic mesaortitis was for a long time confused with the chronic intimal degeneration we now know as atheroma. Disease of the kidneys probably has no direct relation with atheroma, though the high blood-pressure of chronic interstitial renal fibrosis and of secondary contracted kidney is an important factor in producing atheroma in the large elastic arteries.

Pathology.—Atheroma occurs in the large elastic and muscular arteries. The condition is usually most marked in the aorta. The coronary, cerebral, retinal, radial, brachial, and temporal arteries are frequently affected. The peripheral arteries may be normal to the feel in cases where there is advanced atheroma of the coronary or cerebral arteries. In the slighter degrees, minute yellow flecks or patches on the aorta may be observed by the naked eye. In the later stages, yellow plaques or buttons are conspicuous, and under the microscope masses of large fatty crystals, with a covering layer of fibrous tissue, are to be noted. Atheromatous plaques may ulcerate and the contents be discharged into the aorta, and thrombi are often deposited on the surface of these atheromatous ulcers. As the atheroma may be associated with degeneration of the media, a general dilatation of the aorta is very common. On the other hand, circumscribed aneurysm due to severe medial degeneration very rarely occurs. The aortic valves are frequently affected by atheromatous degeneration, and aortic stenosis or aortic regurgitation may result. A yellow atheromatous patch is commonly seen on the anterior flap of the mitral valve. Atheroma often causes great narrowing of the

lumen of the vessels, and eventually a thrombosis may form and complete occlusion result; this is the most dangerous result of atheroma; it frequently occurs in the large divisions of the coronary arteries, especially in the anterior interventricular branch of the left, and is not uncommon in the vessels of the brain. With regard to the heart, atheroma has a most profound influence, owing to the fact that it is one of the commonest causes of fibrosis of the myocardium; but the chief danger is a sudden blockage of one of the coronary arteries, generally the anterior ventricular branch of the left coronary. In these cases, if death does not follow immediately, a sudden softening of the heart muscle (*myomalacia cordis*) may occur, and an aneurysm of the heart may result, and in certain rare cases actual rupture of the heart wall has followed—a broken heart. In the brain, atheroma results in cerebral thrombosis, and is the commonest cause of this condition in old people, and in middle-aged people who have not had syphilis. Very commonly, however, especially in those cases of atheroma where the blood-pressure is raised, hæmorrhage may occur. In most cases of atheroma the kidneys are not involved, though occasionally atheromatous plaques may be found on the branches of the renal artery. Should, however, one of these plaques be large enough to cause much narrowing of an interlobar artery, a wedge-shaped red area of fibrosis in the distribution of the artery will occur. The renal changes, however, are relatively unimportant, and they rarely lead to symptoms during life.

Symptoms.—The blood pressure is only raised if the atheroma happens to complicate cardiovascular hypertrophy. In the aorta a diffuse dilatation, with pulsation in the supra-manubrial notch, may be present. The radial, brachial, and temporal arteries are often irregularly thickened and tortuous and can often be seen pulsating beneath the skin, but this dilatation has none of the important effects that so often follow a saccular aneurysm. Atheroma of the coronary arteries frequently gives rise to cardiac failure, and more often to angina pectoris; and sudden death is not uncommon, owing to a sudden thrombosis of a large branch. In the brain, hemiplegia usually results from hæmorrhage and more rarely from thrombosis. Ocular symptoms are rare in atheroma of the retinal arteries, which may be seen with the ophthalmoscope to be irregularly swollen and tortuous, but swelling of the optic disc and retinitis are not present.

Prognosis.—The course and prognosis are extremely uncertain. Circulation through the diseased vessels may proceed fairly satisfactorily for a long time, but thrombosis may occur with alarming suddenness, and with the direst results, if a cerebral or cardiac artery is affected.

Treatment.—The treatment of atheroma is unsatisfactory. Prophylaxis exists in the removal of the cause when possible. Great attention should be paid to diet, and repletion should be studiously avoided. Alcohol and tobacco should be taken with greatest moderation, and attention should be paid to regular exercise, and the action of the skin should be assisted by warm baths. It is doubtful if drugs are of value in the treatment of atheroma; but small doses of potassium iodide seem to be of some use in absorbing the degenerative products and assisting in the circulation of the blood through the obstructed areas.

OTHER DEGENERATIONS ALLIED TO ATHEROMA

There are closely allied intimal degenerations, in which this accumulation of fatty debris is absent.

In *fibrotic degeneration*, muscle fibres and to a less extent elastic fibres disappear after little or no fatty degeneration, and the intima becomes fibrotic. This may be found in any artery, but is very common in the smaller arteries, for instance, the interlobular and afferent arteries of the kidneys, where atheroma is very rare.

Hyaline degeneration affects the ultimate arterioles in the kidney and other organs; the muscle and elastic fibres disappear rapidly, and the intima becomes swollen and hyaline, and usually fatty. It is common with high blood pressure, but is otherwise rare.

Both these forms of intimal degeneration are of importance, because intimal fibrosis is much less focal than atheroma, and both forms, in affecting arteries of small calibre, lead in the same way as atheroma to a narrowing of the lumen sufficient to cause ischæmic destruction of the tissues. They may therefore be considered together with atheroma in their clinical effects.

ARTERIAL INFILTRATION

The commonest form of infiltration is amyloid infiltration.

Ætiology.—Amyloid or lardaceous disease occurs in cases of long supuration due to pyogenic organisms, and is frequently associated with the secondary pyogenic infections that occur in tuberculosis of the bones and joints, in chronic syphilitic ulceration, and in actinomycosis.

Pathology.—The amyloid substance is extracellular and is deposited beneath the endothelium of capillaries, the reticulum of adenoid tissue and the pulp of the spleen, and in the smaller arteries and veins, especially in their middle coats. The affected organs are firm to the touch and have a waxy appearance. The amyloid substance can be demonstrated macroscopically by pouring tincture of iodine on the affected organ, the waxy material being stained a deep mahogany colour. Microscopically, an iodine staining may be used, or a methyl-violet stain, which colours the amyloid substance pink and the parenchymatous cells blue. In the kidney, the small arterioles in the glomeruli, those around the convoluted tubules and those in the medulla, are first attacked. In the intestine, the arterioles in the villi stand out clearly. There are two forms of amyloid infiltration of the spleen: the diffuse waxy spleen, where the venous sinuses are outlined and the central artery of the Malpighian capsule is affected; and the sago spleen, where the Malpighian capsule is greatly enlarged by the amyloid infiltration—its central artery is untouched, but its branches into the capsule are greatly swollen by the waxy material. In all these organs the parenchymatous cells are unaffected directly by the amyloid infiltration, but in the later stages necrose, owing to interference with their nourishment.

Symptoms.—The patient is pale, but often has a waxy complexion with a bright colour in the cheeks. Chronic, profuse and painless diarrhœa is common. The urine contains a large quantity of albumin, and is usually fair in

amount and of low specific gravity. The liver and spleen are enlarged, and ascites and oedema of the legs are often present.

Prognosis.—If the chronic suppuration can be cured, the condition may sometimes disappear; but in the majority of cases this is not possible, as the septic condition is engrafted on to a chronic tuberculous or other granulomatous condition, which is almost always impossible to eradicate.

Treatment.—This consists in trying to remove the cause.

INTERMITTENT CLAUDICATION

So far the various conditions discussed have been classified according to the underlying pathology. Intermittent claudication is only a symptom, but it provides such a characteristic syndrome that it is best taken separately here. The term "intermittent limp" or claudication is applied to a condition in which severe pain, in one or both legs, comes on after walking for a certain distance.

Ætiology and Pathology.—In the large majority of cases this syndrome occurs in elderly men, who have well-marked calcification of the middle coat of the arteries of the lower limbs (Mönckeberg's degeneration, *q.v.*). In rarer cases it may be present in atheroma or diffuse hyperplastic sclerosis or thrombo-angiitis obliterans (*q.v.*). It may be associated with high blood pressure, gout, diabetes, syphilis, or excessive indulgence in tobacco.

The symptoms are due to the arteries of the leg being unable to supply the muscles with the increased flow of blood that the limb requires during walking. It is one of the earliest signs of partial impairment of the arterial flow.

Symptoms.—The characteristic pain comes on after walking a certain distance and is brought on more quickly by faster walking. It may be accompanied by cramp in the calves, and also by numbness or tingling sensations. It causes the patient to limp and finally to stop. After resting for a minute or two, he is able to continue walking, but the symptoms again recur after he has walked for a further period. Sometimes the pain may only be described as tiredness or may only be felt in the feet and unless a careful examination is made the pain may be attributed to flat feet. Often the patient may suffer from angina pectoris, the causation of the two conditions being very similar, or his anginal pain may seem to disappear as his intermittent claudication appears, really because he is no longer able to walk quickly enough to provoke the anginal pain. In nearly all cases of long standing there is absence of pulsation in the dorsalis pedis artery, or in the posterior tibial of the affected limb, which often shows signs of circulatory disturbance, being swollen, congested, and mottled, while the toes may be white and cold; but in early cases the arteries may still carry enough blood when the patient is resting so that it is more difficult to confirm the diagnosis. The amount of calcification in the arteries, which is often very extensive, may be determined by X-ray examination. In many cases dry gangrene of the limb has supervened.

Prognosis.—The prognosis is bad, but the attacks may persist for years before more serious results, such as gangrene, appear.

Treatment.—Exercise must be limited, and the patient warned to move

slowly and avoid hurrying in his walks. The production of intermittent venous occlusion may give helpful results. Diathermy has been used to relieve the pain. Heart muscle extracts and preparations of the pancreas given hypodermically have been tried, but without much success. Dry gangrene may require amputation of the limb, and when threatened may be relieved by removing the sympathetic nerves round the femoral artery.

ANEURYSM

Definition.—The word aneurysm is derived from the Greek to widen or dilate, and may be said to include any dilatation of an artery.

Aneurysms are generally divided into—

1. **TRUE ANEURYSMS.** in which the walls of the dilatation are formed by the coats of the artery. These may again be divided into—

(a) *Diffuse aneurysm.*—These are general dilatations of an artery. The dilatation is generally not great, and is of little clinical significance except that it indicates medial degeneration. The artery is sometimes tortuous in addition. The so-called cirroid aneurysm is a very extreme example of this.

(b) *Circumscribed aneurysm.*—These are limited to a segment of an artery or to a part of its circumference.

(c) *Dissecting aneurysm.*—These are caused by the splitting of the coats of the artery, the blood having passed through the lumen into the wall of the artery, separating one coat from another.

(d) *Arterio-venous aneurysm.*—In these there is a communication between an artery and a vein; there are two varieties in this group—(1) aneurysmal varix, and (2) varicose aneurysm.

2. **FALSE ANEURYSMS** are those following a wound or rupture of an artery, with the formation of a diffuse or circumscribed hæmatoma, and are bounded by tissues external to the wall of the artery.

When a true aneurysm ruptures and gives rise to a false aneurysm the resulting structure is known as a mixed aneurysm.

Ætiology and Pathology.—The two main factors in the causation of aneurysm are—(1) loss of the muscular and elastic fibres in the wall of the artery; (2) strain. The latter is generally brought about by repeated and prolonged muscular effort, and high arterial pressure may sometimes be an additional factor. The importance of strain as a causal factor is borne out by the fact that aneurysm is much more frequent in men than women—about five to one—and occurs more frequently in the fourth decade of life than at any other period. It also occurs much more frequently in hard manual workers, such as dock labourers, soldiers and sailors. *By far the most common cause producing weakening of the large elastic arteries is syphilitic inflammation. In persons dying of aneurysm, examination of the aorta in the neighbourhood of the aneurysm will generally reveal mesaortitis* (see page 1031). In the first part of the aorta this is nearly always so, but as one gets farther away from the aortic valves an increasing number of aneurysms are due to atheroma and non-syphilitic degeneration of the media; in the abdominal aorta less than half are syphilitic. Small aneurysms may also result from erosion of the walls of the arteries in cases of septic endocarditis—the so-called **mycotic aneurysm**. Frequently these are multiple; they occur most often

in the cerebral arteries or in the peripheral arteries. Extensive growth of streptococci and septic granulations may be seen in the neighbourhood of the dilatations.

Aneurysm may also be the result of congenital defects in the media of the vessel, which is very commonly seen about the circle of Willis, at the junction of the anterior communicating artery with the anterior cerebral. The aneurysms vary from about the size of a pin's head to that of a pea, and not infrequently their rupture gives rise to a diffuse subarachnoid hæmorrhage, the origin of which is often overlooked unless careful search is made for the aneurysm. Congenital aneurysm has also been described in the aorta, at the point of insertion of the ductus Botalli, and in cases of coarctation of the aorta, in which condition there is great narrowing of the aorta just below the origin of the left subclavian artery.

Loss of support by surrounding tissues also appears to lead to the production of aneurysm, *e.g.* at the base of a gastric ulcer a small aneurysm often projects as a nodule and is liable to rupture. Peptic erosion may also be a cause of weakening the walls of such arteries. In the cavities of the lungs, occurring as the result of pulmonary tuberculosis, it is quite common to find an aneurysm on the walls of the arteries lying in such cavities.

It is very doubtful if external trauma alone is ever the cause of true aneurysm, but injury to the artery by penetrating wounds by knives or bullets may certainly cause it.

Slight medial degeneration leads to diffuse aneurysm, and severe medial degeneration may cause circumscribed aneurysm. It is the usual cause of circumscribed aneurysms of muscular arteries, such as the popliteal, but is a very rare cause of circumscribed aneurysm of the aorta. Atheroma itself does not lead to aneurysm, but it may be complicated by medial degeneration. Continued high blood-pressure is an important contributory factor in the formation of a diffuse dilatation of the aorta, but this has few clinical resemblances to saccular or even to fusiform aneurysm.

One of the most striking appearances in an aneurysm is the coagulation of blood in the sac itself. This does not occur in diffuse dilatation of the aorta, but in those cases of sacculated aneurysm where the wall has become roughened. The sac becomes lined with fibrinous deposits, and occasionally an aneurysm may be cured by the deposition of successive layers of fibrin, so that the sac becomes almost completely filled. On the other hand, thrombus in aneurysms may form emboli and so lead to infarcts. Again, in many fatal cases of aneurysm, rupture and hæmorrhage have taken place, the deposition of fibrin having failed to prevent the blood reaching the surface. Rupture may take place externally, or into any of the hollow viscera or the serous cavities. Rupture into the pleura is common, as also is rupture into the trachea or into the œsophagus. In these cases, death is usually sudden, though oozing may have taken place some time before the final rupture. When the hæmorrhage forces itself into the connective tissues or muscles, it takes place much more slowly, and in the cases of the limbs may allow time for treatment. An aneurysm often exercises pressure on the other organs and structures in its neighbourhood. The heart is displaced away from the aneurysm; and the blood vessels are often narrowed so that circulation through them is impeded. The trachea, the bronchus, the œsophagus, and the nerves passing near the aneurysm also suffer. When the aneurysm meets

bony tissues, absorption of the bone takes place and the vertebræ are frequently eroded in this way, the bone being absorbed more rapidly than the intervertebral cartilage. When the aneurysm presses against the anterior surface of the chest, the ribs and sternum are pushed forward and finally are absorbed and perforated.

I. ANEURYSM OF THE THORACIC AORTA AND ITS BRANCHES

These are two types—(1) The diffuse aneurysm, or general dilatation, which occurs in medial degeneration of the aorta ; and (2) the circumscribed, usually saccular, aneurysm, almost always the result of syphilitic inflammation.

GENERAL DILATATION OF THE AORTA (DIFFUSE ANEURYSM)

Symptoms.—In general dilatation of the aorta the enlargement is extensive, but never reaches a very great size. The symptoms are generally due to atheroma and the medial degeneration that accompanies it. Very often there is interference with the coronary circulation, and this leads to diminution of the circulation of blood through the heart and consequently to cardiac pain. The aorta may sometimes be felt pulsating in the supra-manubrial notch, and the X-ray photograph will show general dilatation of the aortic arch. When the aortic ring is not stretched, the dilatation of the aorta beyond it may lead to the formation of a systolic murmur. Occasionally these patients die suddenly, on account of thrombosis of an atheromatous branch of a coronary artery. Provided there is no aortic regurgitation or coexisting coronary disease, patients with a dilated aorta may live for many years without serious discomfort.

CIRCUMSCRIBED ANEURYSM

These are generally saccular but may be fusiform.

Symptoms.—ANEURYSM OF THE ASCENDING PART OF THE ARCH OF THE AORTA.—An aneurysm of the ascending part of the arch tends to grow forward and outwards, and to produce a pulsating tumour that is palpable and audible at the level of the second or third interspace ; hence it is often called the *aneurysm of physical signs*. It often erodes the ribs and sternum. The tumour is tender and is often the seat of pain, which may be constant but is increased by exertion. A soft systolic murmur may be heard over it. If syphilitic aortitis spreads to the aortic valve or if the part adjacent to the aortic valves is affected, the aortic ring may be dilated and aortic regurgitation will take place. There will then be hypertrophy and dilatation of the left ventricle, with an aortic diastolic murmur, and the symptoms of aortic regurgitation will be added.

The pressure effects produced by an aneurysm of the ascending aorta are as follows : The heart is displaced downwards and to the left. The superior vena cava is pressed upon ; this may result in cyanosis of the head and neck, and œdema of the arms, and enlarged veins may occasionally be seen coursing over the front of the thorax. Very rarely the aneurysm may rupture into the superior vena cava, in which case the symptoms noted may come on quite suddenly and are very marked. There is often a systolic thrill, and on auscultation a continuous murmur, which is increased during

systole, is of great diagnostic value. The aneurysm may press upon the right bronchus, causing a chronic cough, due to irritation of the bronchus and stasis of its contents, and deficient or absent breath sounds over the upper lobe of the right lung. It may occasionally press upon the pulmonary artery and in rare cases, actually open into it, causing great dilatation of the right ventricle and auricle. Such aneurysms may present themselves to the left of the sternum rather than in the usual place, the right. Aneurysm of the ascending aorta has been known to perforate into the right ventricle and much more frequently into the pericardial sac. When the aneurysm comes forward it may irritate the pleura; and in some cases a loud pleuritic rub, audible over the aortic area, may be one of the early signs. Not uncommonly this form may rupture into the pleura or sometimes externally.

ANEURYSM OF THE TRANSVERSE ARCH OF THE AORTA.—As the arch of the aorta passes from right to left it also passes from before backwards, and consequently aneurysms arising from the transverse and descending parts of the aortic arch are situated more deeply in the chest than those arising from the ascending portion. Aneurysms of the transverse and descending parts of the aortic arch have been called by Broadbent *aneurysms of symptoms*, because their presence has often to be inferred by the pressure symptoms that they produce, while a pulsating tumour is only present in the very late stages and may not appear at all.

1. *Pain.*—Pain is one of the commonest and earliest symptoms of aneurysm. It may often occur behind the sternum or across the back and pass down the left arm and be very severe: it then lasts longer than true anginal pain and is more liable to occur at night; this form of pain occurs when the ascending aorta is distended. When, however, the transverse arch is affected the pain is sometimes felt on the left side of the neck and even in the occipital region; it is probable that this pain in the neck is a reflected pain caused by abnormal afferent impulses reaching the cervical spinal cord as a result of the distension of the transverse arch. A boring, persistent pain in the chest is probably the result of direct pressure of the aneurysm. Sometimes there may be true angina pectoris owing to involvement of the mouths of the coronary arteries.

2. *Respiratory Symptoms.*—Dyspnoea is common in aneurysm, and apart from associated heart disease is usually caused by pressure upon a bronchus. There is often stridor, which in this case is heard both in inspiration and in expiration. Hæmorrhage also occurs as a result of leaking of an aneurysm through the bronchus. It may at first be slight, but often a huge gush of blood supervenes, causing death. It is said that reflex irritation of the vagi will occasionally cause bilateral adductor spasm of the vocal cords and marked dyspnoea. In this case the stridor is only heard with inspiration and disappears if a little chloroform is inhaled. Patients with aneurysm often have a ringing, rough, brassy cough. If the pressure on the bronchus has been gradual, secondary changes occur in the lung, and compression of the left bronchus in the early stages may produce over-distension of the left lung with diminished or absent breath sounds, so as to lead to a suspicion of pneumo-thorax; later on bronchitis occurs with dilatation of the bronchus with expectoration of purulent phlegm. Pressure on the trachea may occasionally be observed in cases of aneurysm of the aortic arch, the larynx being drawn downwards and backwards with each cardiac pulsation. A

physical sign described by Surgeon-Major Oliver is known as *tracheal tugging*. The patient should be placed in the erect position, and directed to close his mouth and elevate his chin. The cricoid cartilage should be grasped between the finger and thumb and gentle steady upward pressure be made upon it. If there is an aneurysm, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand. This sign may occur in aneurysm before other symptoms are evident, but is not very reliable.

3. *Pressure on nerves.*—(a) The left recurrent laryngeal nerve.—This nerve courses round the arch of the aorta and passes up behind it, and is consequently often involved in aneurysm of the transverse arch. The abductor fibres of the recurrent laryngeal nerves succumb to the effect of pressure before the abductor fibres and consequently the vocal cord is at first in the position of adduction. During respiration, the right vocal cord moves up to and meets the adducted left vocal cord and respiration is unaffected. During phonation, the right vocal cord again comes up to and meets the left vocal cord, and the voice may be quite normal. It is therefore clear that a laryngoscopical examination may reveal the early stage of pressure on the left vocal cord before there has been any alteration in the voice. As the pressure increases, the adductor muscles are affected and the left vocal cord remains motionless in the cadaveric position half-way between full inspiration and full expiration. The voice is now hoarse, and it not uncommonly happens that hoarseness is the first symptom for which the patient presents himself. (b) The sympathetic nerve.—Pressure upon the sympathetic nerve causes first of all irritation and later on a paralysis of the cervical sympathetic fibres. When the sympathetic is irritated, the pupil on the same side is dilated, and there may be sweating and flushing of the same side of the face and ear. When the cervical sympathetic is paralysed, the pupil is smaller than on the opposite side, the eyeball may be sunken into the orbit, and there may be a slight degree of ptosis. Unequal pupils in aneurysm are, however, frequently found without any of the other symptoms of sympathetic irritation or paralysis, and in these cases Doctors Wall and Walker suggest that the difference in the pupils is due to the pressure of the aneurysm upon the arteries passing to the neck. They have shown that in a condition of low blood pressure the pupils are dilated, whilst in a condition of high blood pressure they are contracted, and that pressure upon one common carotid will cause dilatation of the pupil on the same side. They point out that under these conditions the dilated pupil is constantly on the same side as the smaller temporal pulse, and they consider that the dilated pupil and the small pulse are due to the same cause, namely, pressure upon the arteries supplying that side of the neck. Unequal pupils also occur as the result of syphilitic disease of the nervous system. We may get bilateral pin-point pupils, or the pupils may be unequal and irregular in outline. In both cases the reaction to light is lost, while the reaction to accommodation remains. (c) Pressure upon the intercostal nerves occasionally results from an aneurysm that presses backwards and erodes the vertebræ and posterior portions of the ribs. In these cases the pain is very severe along the affected nerves. In the distribution of the nerve itself the skin may be anæsthetic—the so-called *anæsthesia dolorosa*. (d) In some cases the aneurysm presses upon the brachial plexus, shooting pain occurring in the head and neck and down the right arm.

4. *Pressure upon the branches springing from the aorta.*—This is not uncommon, and may result in the absence of one radial or temporal pulse, or inequality between the two pulses. If the innominate artery is pressed upon, the right radial and temporal arteries may be small and imperceptible, while in an aneurysm of the transverse arch of the aorta, the left subclavian artery is compressed, in which case the left radial pulse may be affected. A sphygmographic tracing is often of value in demonstrating the difference between the two pulses, and a forced expiration, by increasing the intrathoracic pressure, will often accentuate the difference between the two sides. When the pulses are markedly unequal, the blood-pressure is also diminished on the side of the feebler pulse; a difference of pressure of over 30 mm. between the two sides is in favour of aneurysm provided it is not due to coarctation of the aorta.

5. *Pressure on the œsophagus.*—This may result in slight difficulty in swallowing but the dysphagia is very rarely important. Occasionally the aneurysm may ulcerate into the œsophagus so that death takes place from a sudden rupture.

ANEURYSM OF THE DESCENDING PORTION OF THE ARCH.—In these cases the sac frequently projects backwards and erodes the vertebræ from the third to sixth thoracic, causing great pain and occasionally compression of the spinal cord, resulting in paraplegia. Dysphagia is more common, and sometimes a tumour appears in the region between the scapula and the spine, and may attain a very large size.

ANEURYSM OF THE DESCENDING THORACIC AORTA may occur close to the diaphragm. Aneurysm of this form is frequently overlooked, pain in the back being the most prominent early symptom.

Diagnosis.—*Inspection.*—This is most essential. Abnormal pulsation should be looked for in the thorax, and can often be seen when the patient is seen obliquely in a good light. Posterior pulsation is generally observed to the left of the spine. Enlarged veins over the chest, suffusion of the face, and alteration in the pupil may be noted. The apex-beat is often displaced from its normal position, especially when the sac is large, this being due to pressure of the aneurysm on the thorax; the heart itself is seldom hypertrophied, unless there is a leakage through the aortic valves.

Palpation.—Palpation may reveal the area and degree of the abnormal pulsation. There may only be a diffuse impulse, but if the sac has perforated the chest wall, a forcible heaving and expansile impulse may be felt. Occasionally a diastolic shock is to be noted. This has been thought by some to be due to the forcible closure of the aortic valves producing an effect within the aneurysmal sac; another explanation is that the contraction of the heart draws in the ribs during systole at the point where they are adherent to the aneurysm and the diastolic shock is produced by the elastic recoil of the ribs and costal cartilages. Occasionally a systolic thrill may be felt.

Percussion.—A dull area may in some cases be made out in the second right interspace in cases of aneurysm of the ascending aorta. Much more rarely an aneurysm of the arch may press forwards and to the left, and produce dullness below the left clavicle. Pressure upon a bronchus may at one period result in hyper-resonance from lung distension, and later, owing to absorption of air in the lung, the percussion note may become dull.

Auscultation.—There may be no murmur, even in a large aneurysm, but a systolic murmur is not uncommon. When both systolic and diastolic murmurs are heard, aortic regurgitation is present in addition to the aneurysm. Accentuation of the aortic second sound is a most constant auscultatory sign of aneurysm, but it occurs with syphilitic aortitis before an aneurysm has developed.

Reference has already been made to alteration in the radial and temporal pulses. Exceptionally, in a large aneurysm of the descending aorta there may be absence of pulsation in the abdominal aorta and peripheral arteries of the legs, the dilatation of the thoracic aorta being sufficient to convert the intermittent into a continuous stream.

Examination by radioscopy is most important in every case where aneurysm is suspected and may give valuable information. The chest should be examined from the anterior, the posterior and the right oblique positions. The pulsation of the tumour and its relation to the aorta may actually be seen, but photographs should always be taken, both in the anterior-posterior and oblique positions, as much information is obtained from the density of the shadow cast by the aneurysm. If the latter is very dense, it can be reasonably inferred that deposition of the laminated clot has taken place within the sac.

Complications and Sequelæ.—The main complication causing death is rupture, which may take place either externally, into the pericardium, into the pleura, into the œsophagus, into the bronchus, or into the lung tissue itself. Pressure on the trachea, causing stridor and respiratory obstruction, is a very distressing complication. Bronchitis may occur during the course of the illness, and may be recovered from more than once. Broncho-pneumonia and gangrene of the lung not infrequently occur when there is pressure upon a bronchus, and empyema may occasionally result. Tuberculosis of the lung may coexist with aneurysm, but death from hæmoptysis, the result of perforation of a deep-seated aneurysm into the bronchus, has often been mistaken for the profuse hæmorrhage of tuberculosis. Cardiac failure is responsible for a large number of deaths. This may be the result of interference with the circulation through the coronary arteries, and in other cases it is due to the aortic regurgitation. Cerebral embolism sometimes occurs in cases of aortic aneurysm, a portion of the clot within the artery becoming displaced and passed up to the brain.

Course.—Most cases live from 2 to 5 years from the time when the first symptoms have appeared. Occasionally life may be prolonged for several years by treatment, provided the diagnosis is made early. Spontaneous cure may be obtained by deposition of laminated clots within the cavity of the aneurysm, but this is comparatively rare.

Diagnosis.—Intrathoracic aneurysm is sometimes difficult to diagnose from intrathoracic neoplasm. In both there may be an externally projecting tumour, but in aneurysm the pulsation may be seen to be expansile. The diastolic shock indicates an aneurysm. Systolic murmurs may occur in both conditions, but the ringing aortic second sound is of great importance, and is rarely heard in tumours. Tracheal tugging is in favour of aneurysm, while progressive wasting and enlargement of glands in the neck are in favour of neoplasm. Aneurysm, as a rule, occurs in apparently healthy men between 45 and 60 years of age, whereas malignant growth in the chest is associated

with emaciation and pallor. In aneurysm there is a greater likelihood of the pupils and pulses being unequal, while in neoplasm oedema of the upper extremities and chest wall is not uncommon. In all cases an X-ray examination should be made, and will nearly always clear up the diagnosis. Clinical evidence of infection by syphilis and a positive Wassermann reaction are obviously important.

A violently pulsating thoracic aorta, either in association with aortic regurgitation or with violent throbbing of the heart, may lead to the unfounded suspicion of an aneurysm.

In cases where an empyema is pointing on the left side in the region of the heart, the tumour may pulsate. The throbbing is usually diffuse and widespread, and there is a coexistence of a pleural effusion. Exploration with a fine needle will usually settle the diagnosis. It must, however, be remembered that occasionally an empyema may be the result of extension of septic trouble from a bronchus which has become compressed by an aneurysm.

Prognosis.—In aneurysm this is always difficult. Complete cure is very unlikely, although pain and other unpleasant symptoms and physical signs may give way to treatment. The presence of aortic regurgitation is unfavourable, while an aneurysm progresses much more slowly in people of a placid disposition and those who lead a quiet life. Even in cases where treatment has apparently been most successful and pain and dyspnoea have been apparently relieved, sudden death from rupture may occur.

Treatment.—The recognition that the main cause of aneurysm is the weakening of the wall by syphilitic mesaortitis has brought anti-syphilitic remedies into the forefront of modern treatment of aneurysm of the thoracic aorta. Mercurial inunctions and injections have been but little tried, but in many cases a good deal of benefit has been observed. The iodides have been given in aneurysm for many years—long before syphilis had been recognised as a cause of the condition. The most striking effect of iodide is the relief of pain, and this may be obtained by even small doses, such as 5 grains three times a day. In all cases, however, large doses, such as 20 grains three times a day, should be given a trial. Neoarsphenamine has been given intravenously in many cases with great success; it should always be tried when the diagnosis is made early enough, but should never be used if congestive failure or paroxysmal nocturnal dyspnoea have developed.

Efforts to produce clotting within the sac should be tried in early cases of aneurysm. Tufnell's method—that of a complete rest and restricted diet—is only of historic interest. The patient had to lie in a quiet and secluded room for several months. Few patients put up with such a rigid diet and rest. Secondly, the administration of lime-salts, such as the chloride or lactate of calcium, should also be tried, in an endeavour to promote clotting within the sac. The gelatin treatment has been abandoned.

Many patients with intrathoracic aneurysm do better if, after a preliminary rest with the treatment appropriate to their stage, they are allowed to follow their general vocations, provided their work be not too strenuous for body or mind. Nearly always it will be necessary for their activity to be curtailed. Patients should be cautioned to take things as easily as possible, to avoid alcohol, to eat with great moderation, and to avoid any sudden exertion. At least 10 hours should be spent in bed. A certain amount of tobacco may be smoked.

Special symptoms may have to be treated. For severe pain, cyanosis and dyspnœa, venesection will often give marked relief. Amyl nitrite and iodide of potassium are of great service in relieving the anginal pain of aneurysm. Severe paroxysmal dyspnœa is nearly always due to direct pressure on the trachea; both inspiratory and expiratory stridor are present. The inhalation of chloroform does not give relief, and tracheotomy is useless. In some very rare cases there may be a bilateral abductor spasm of the vocal cords, due to irritation of the vagi, and relief may be obtained by chloroform. Intubation of the larynx is preferable to tracheotomy, which should never be performed to relieve the dyspnœa of aneurysm.

Surgical Treatment.—This may be considered under four heads—(1) ligature of the vessels arising from the arch of the aorta; (2) the passage of wire into the sac with or without galvanism; (3) needling the sac; and (4) ligature of the neck of the sac. There are few cases where the outlook under medical treatment is so bad that the certain risks and uncertain benefits of surgical treatment can be advised with wisdom.

Ligature of vessels has been of little service in the case of aneurysm of the aorta. In aneurysm of the innominate artery, combined simultaneous ligature of the right common carotid and subclavian arteries may be tried, but even this may be insufficient to prevent the flow of blood through the sac. Moore's method of introducing silver or zinc wire into the sac through a cannula has been used, but the best results have been in cases of abdominal rather than intrathoracic aneurysm. Puncture of the aneurysm and scratching its wall with the point of a needle, as advocated by Sir William M'Ewen, has sometimes been partially successful. Ligature of the neck of the sac may be undertaken when it appears to be small, but suitable cases are very rare.

ANEURYSM OF THE INNOMINATE ARTERY.—This is not uncommon. It forms a pulsating tumour, which can sometimes be felt above the right clavicle, and nearly always produces marked diminution in the right radial and temporal arteries. In this form of aneurysm, paralysis of the right recurrent laryngeal nerve occurs not infrequently, the right vocal cord being paralysed instead of the left.

ANEURYSM OF THE CAROTID AND SUBCLAVIAN ARTERIES is mainly of surgical interest. It has been thought to be most frequent in the common carotid, especially in women, but in many of these cases there is really a kinked carotid artery, due to hypertension and athero-sclerosis, that simulates an aneurysm. Subclavian aneurysm is nearly as frequent as carotid aneurysm. Syphilis is found in nearly all cases near the aorta but trauma becomes more important towards the periphery.

II. ANEURYSM OF THE ABDOMINAL AORTA AND ITS BRANCHES

Aneurysm may occur in any part of the abdominal aorta, but it is much less common than aneurysm of the thoracic aorta. A forcible dynamic pulsation of the vessel is often mistaken for aneurysm and no case should be diagnosed as aneurysm unless a tumour can be grasped between the fingers. Often in true aneurysm there is evidence of syphilis and the Wassermann reaction is positive. A systolic thrill can sometimes be felt, and a systolic murmur is, as a rule, audible. The complications in abdominal aneurysm are

many. Death may result from complete obliteration of the lumen by clots, or by erosion of the vertebrae and compression of the spinal cord, resulting in paraplegia. Occasionally the superior mesenteric artery may become blocked by a clot and acute intestinal obstruction result. The commonest complication is rupture, which generally takes place into the retro-peritoneal tissues, with the formation of a large rapidly-growing tumour in the flank. More rarely death takes place from rupture into the peritoneum or duodenum.

Treatment.—The treatment of abdominal aneurysm is the same as that of thoracic aneurysm. In cases where medical treatment is unsuccessful after a fair trial, surgical measures should be undertaken and are more likely to be successful than in thoracic aneurysm.

ANEURYSM OF THE SPLENIC ARTERY is very rare. A tumour can be felt near the spleen and it may perforate into the colon. If the diagnosis can be made, removal of the aneurysm and of the spleen should be undertaken.

ANEURYSM OF THE MESENTERIC ARTERY, which is also rare, generally results in plugging of the vessel or its branches, with the result that acute intestinal obstruction takes place and death occurs from this cause.

ANEURYSM OF THE HEPATIC ARTERY is very rare.

ANEURYSM OF THE RENAL ARTERY has occasionally been noted, and in some cases successfully removed.

ANEURYSM OF THE BRACHIAL ARTERY used to be common, but is now rarely seen.

ANEURYSM OF THE FEMORAL ARTERY is much more common, and is most often traumatic.

ANEURYSM OF THE POPLITEAL ARTERY is one of the most common of the peripheral aneurysms. It has been suggested that this is due to the fact of the exposure to stress and strain to which the popliteal region is subjected during violent lifting efforts.

In all cases of peripheral aneurysm not due to trauma, a syphilitic basis should be investigated; but the aneurysms of the muscular arteries of medium size are almost invariably due to medial degeneration, and a syphilitic aneurysm would be most unusual. The treatment is mainly surgical. Distal or peripheral ligature and excision have all been tried, and more recently Matas has suggested treatment by *Reconstructive Endoaneurysmorrhaphy*. After rendering the limb exsanguine, he freely opens the arterial sac and by a process of suturing reconstructs a channel between the afferent and efferent artery of the sac. This is theoretically the best treatment, but in practice it is often found impossible.

When the symptoms warrant surgical treatment, proximal ligature is probably the method most frequently used for a large artery and excision of the sac for a smaller one. Proximal ligature as close to the aneurysm as possible has been the classical operation since the time of John Hunter. It should be combined with distal ligature, as this does not increase the risk of gangrene and diminishes the risk of spreading infection or of an embolus becoming detached.

Until the War of 1914–1918 great care was taken to avoid injury to the vein, but it was found that proximal and distal ligature of the vein as well as of the artery reduced the risk of gangrene. This should therefore be the routine surgical treatment.

III. DISSECTING ANEURYSM

This may originate in an atheromatous ulcer. Very often the dissection of the coat is small, especially when the blood-pressure is not high. When, however, there is a very high blood-pressure and much degeneration of the media, an extensive dissecting aneurysm may occur. The degeneration of the media may cause a small split in the intima and the dissection separates the intima from the media, so that in some cases there may be a double tube instead of a single aorta. Extensive dissection frequently causes sudden death, but in other cases the patient may live on, and the association of a rapidly beating heart and a feeble pulse in the lower limbs has been suggested as a clinical sign by which the condition may be recognised. The immediate picture is very similar to that of a coronary thrombosis. If the dissection spreads up the common carotids, symptoms from interference with the cerebral circulation may follow and this soon after an attack, suggesting coronary thrombosis may reveal the diagnosis. Occasionally the patient makes a complete recovery and lives for years with the circulation through the new channel made by the dissection.

In many cases of extensive dissecting aneurysm of the aorta, due to medial degeneration, vascular hypertrophy is well marked, and consequently the blood pressure was high during life. Less commonly dilatation, rupture and the dissecting aneurysm of the aorta are apparently due to congenital weakness, histological evidence of degeneration or inflammation being absent.

IV. CIRROID ANEURYSM

Cirroid aneurysm is a condition in which an artery is dilated and tortuous. Occasionally it is due to medial degeneration of muscular arteries, particularly the splenic and temporal, but this form is of little clinical importance. More often it is due to defective development of the walls of arteries and their branches, and this form has been called serpentine angioma. The arteries, their branches, the capillaries, and even the efferent veins dilate progressively, causing destruction of the intervening soft tissues and erosion of bone. The superficial temporal, posterior auricular, and occipital arteries are most commonly affected. It also occurs in the brain, pancreas, orbit, and limbs. It is most common between puberty and 30 years of age. Although the condition is generally congenital it may develop greatly after some local injury such as a blow.

Pathology.—The arteries are dilated, thinned and very tortuous, and the disease tends to spread towards the capillaries and also along the arteries that feed the aneurysm. The skin over the aneurysm is often atrophied and may become ulcerated, leading to very dangerous hæmorrhage.

Symptoms.—There is an ill-defined pulsating tumour on the scalp, in which the tortuous vessels may be felt. In rare cases the tumour may be slow in its growth, but this is generally rapid and the skin over it ulcerates, leading to hæmorrhage.

Treatment.—This is very difficult. In limited cases the tumour may be excised. Generally, however, ligature of the peripheral arteries of the growth is more satisfactory. Electrolysis and injections of perchloride of iron into the

mass have been tried, with some success. Occasionally spontaneous cure follows some infection.

V. ARTERIO-VENOUS ANEURYSM

Arterio-venous aneurysms, in which an artery and a vein communicate, are of two kinds: (1) aneurysmal varix, where the two vessels anastomose directly; and (2) varicose aneurysm, where the sac separates the connecting vessels.

(1) ANEURYSMAL VARIX.—The aneurysm is usually traumatic in origin and used to be frequent at the elbow, as the result of venesection. The artery is wounded at the same time as the vein and they become connected, the result being that the vein becomes markedly dilated and tortuous.

The varix forms a soft, compressible, ill-defined tumour, which pulsates. Pain in the tumour is not uncommon. A marked thrill can often be felt and a loud bruit may be heard over the tumour. If the limb is raised the tumour shrinks, while it becomes large and congested if the limb is held downwards. The limb below the tumour is often œdematous.

In some cases the aneurysm remains stationary, and all that is required is an elastic support. If, however, it tends to increase in size, the artery should be ligatured above and below its communication with the vein, and the vein should also be ligatured.

An intrathoracic aneurysm may become adherent to a vein and perforate into it. The most common site is when an aneurysm of the ascending arch of the aorta perforates the superior vena cava, but even this is rare. The latter vessel becomes greatly distended and an arterio-venous aneurysm is formed. There is often a sudden onset when the lumen of the two vessels becomes connected; there is congestion of the head and neck and upper limbs, great distension of the veins, and often œdema. On auscultation over the tumour, a continuous humming murmur is heard, with marked accentuation during systole.

(2) VARICOSE ANEURYSM.—This occurs when an artery and vein are simultaneously wounded. A false aneurysmal sac is formed in the tissues and communicates both with the artery and vein. The symptoms are similar to those of an aneurysmal varix, but in addition there is a pulsating tumour, which can be distinguished from the dilated vein. This form of aneurysm should be excised by open operation combined with four-fold ligature of the artery and of the vein. It is not often seen in civil life but is fairly common in war-time, mainly as a result of gun-shot wounds.

ARTERIO-VENOUS ANEURYSM OF THE ORBIT OR PULSATING EXOPHTHALMOS.—This is a form of aneurysm by anastomosis, due to a communication having formed between the cavernous sinus and internal carotid artery as it passes through it. It is generally the result of a fracture of the base of the skull. It is usually unilateral, but may be bilateral, and the communication may take place immediately after the fracture, or evidence of the lesion may only appear days or weeks after the injury.

The main symptom is protrusion of the eyeball, the globe being displaced outwards and downwards. It may be seen to be visibly pulsating, but if not, slight pressure upon the globe of the eye will bring out pulsation. A loud bruit, either continuous or increased during systole, may be heard anywhere

over the head ; this roaring sound is generally very distressing to the patient. There is great dilatation of the veins around the eyelids, conjunctivæ, and fundus. Headache is common

The condition may last for years, and in a few cases spontaneous recovery has taken place.

The treatment is either compression or ligature of the carotid artery. The great danger in ligaturing the carotid artery is the occurrence of cerebral softening and hemiplegia. To avoid this, it is better to ligature the artery temporarily and see if any cerebral symptoms tend to develop ; if they do, the ligature should be removed after 24 hours ; but if they do not, the ligature may be tightened and the artery completely occluded.

DISEASES OF THE PULMONARY ARTERIES

The pulmonary artery is much less frequently the seat of disease than is the aorta, but it is liable to be affected by pathological changes of a similar character to those that are found in the systemic arteries.

Ætiology and Pathology.—Four main pathological changes are generally recognised, namely

1. **HYPERTROPHY.**—This condition is associated with an increase in the blood pressure in the lesser circulation and is found in cases of disease of the lungs, such as emphysema, pulmonary fibrosis, and bronchiectasis, and also in disease of the heart, such as mitral stenosis. The intima of the pulmonary arteries hypertrophies and is prone to degeneration, so that patches of fatty change (atheroma) appear in the hypertrophied tissue. This form of intimal degeneration is superimposed upon hypertrophy, which is the result of obstruction to the lesser circulation and is independent of inflammatory change.

2. **INFLAMMATION.**—There is no doubt that *syphilitic* inflammation may attack the pulmonary arteries as well as the aorta. The larger trunks may be affected by mesarteritis, and saccular aneurysms of the main branches have been described, but are rare. The smaller arteries and arterioles in the lungs may also be affected by syphilitic arteritis, with endarteritis obliterans. The endarteritis, either by itself or in combination with thrombosis, may lead to complete occlusion of the lumen. Ayerza and his pupils have emphasised the importance of syphilis as an ætiological factor in the production of cyanosis and congestive heart failure in the absence of the usual causes of these conditions, such as emphysema and fibrosis of the lungs, or mitral stenosis. Fatty or calcareous changes may occur as secondary changes in the walls of the inflamed arteries.

Tuberculous inflammation of the branches of the pulmonary arteries is also common. Tuberculous endarteritis obliterans is frequent in phthisis. Tuberculosis of the lung or a bronchial gland may extend through the wall of a large artery to its intima, giving an intimal tubercle which when softened can lead to a general dissemination. The wall of a pulmonary artery exposed in a tuberculous cavity is frequently weakened by tuberculous or pyogenic invasion from without, and an aneurysm results. The profuse hæmoptysis found in the latter stages of chronic pulmonary tuberculosis results from the rupture of one of these aneurysms.

3. **DEGENERATION.**—Degeneration of the intima secondary to intimal hypertrophy or to inflammation has been described above. Slight primary intimal degeneration, or atheroma, is not uncommon in elderly subjects. C. F. Coombs and others have described cases of a severe atheroma of the pulmonary artery in young subjects, in whom there was no evidence of syphilis or of pulmonary or cardiac lesions, and suggest that in these cases there is an inherited tendency to intimal degeneration.

4. **CONGENITAL MALFORMATIONS.**—In the majority of cases a pulmonary stenosis is present, but in rare instances dilatation of the vessel has been found. These conditions are described under congenital heart disease (*q.v.* p. 968).

Symptoms.—The symptoms of disease of the pulmonary artery are those of obstruction of the lesser circulation.

Dyspnoea is often an early symptom, and may occur on exertion or in nocturnal paroxysms. In the later stages it becomes constant, with attacks of orthopnoea. Cyanosis is one of the most characteristic manifestations, but it varies in intensity and also in the stage of the disease at which it appears. Cyanosis is due to an imperfect oxygenation of the blood passing through the lungs at each cardiac cycle. It will, therefore, be most marked in those cases in which only a small proportion of the blood passes through the lungs at each beat, as in congenital pulmonary stenosis, or those in which the capillary area in the lungs has been so reduced by emphysema or the lung itself has been so damaged by fibrosis that the circulating blood is imperfectly oxygenated. In mitral stenosis the degree of cyanosis is remarkably variable and probably depends as to whether secondary changes in the lungs have developed. In Ayerza's disease (syphilitic inflammation of the pulmonary arteries) the cyanosis may be extreme and the patients may have almost a black appearance (*cardiacos negros*). This may be due to the endarteritis obliterans of branches of the pulmonary artery, or to a coexisting syphilitic obliterating bronchitis, or a syphilitic pneumonia causing fibrosis of the lung.

Hæmoptysis may occur before cyanosis has become established or in the later stages. It may be slight or profuse, and may be associated with attacks of pulmonary artery thrombosis. Cough, with mucopurulent expectoration, is common, and attacks of vertigo may occur. Somnolence is not infrequently found when marked cyanosis is present. The fingers are not clubbed, except in cases resulting from bronchiectasis or fibroid lung.

The pulse is usually regular and the heart is much enlarged, especially the right ventricle. If mitral stenosis is present diastolic murmurs may be heard at the apex. There are no constant physical signs in the lungs, but if emphysema, fibrosis, or bronchiectasis has been the determining factor, the physical signs characteristic of these conditions will be found. Œdema is often present and may be extreme and the liver enlarged. The spleen is not palpable. The blood shows an increase in the number of red cells, up to 8,000,000, the number varying with the degree of cyanosis.

The radioscopic findings are characteristic. The right ventricle is enlarged, the pulmonary artery often dilated, and the branches of the pulmonary artery show more clearly than usual and can be followed into the lung and in some cases can be seen to pulsate. The electrocardiogram shows a marked right ventricular preponderance and alterations in the *P* wave, suggesting right auricular hypertrophy.

Diagnosis.—Cases of pulmonary artery affections secondary to pulmonary or cardiac disease can be distinguished by the presence of the symptoms and signs of the underlying lesion. There is no sure method of diagnosis between syphilitic and non-syphilitic cases. In syphilitic pulmonary arteritis the patients are usually between 30 and 50 years of age and may give a history of syphilitic infection. The Wassermann reaction in the blood is positive.

Course.—There may be a history of pulmonary symptoms, such as cough and dyspnoea, for many years. Later the intense cyanosis may develop, and this may last for 4 or 5 years. Some of these patients die in their sleep, but in others myocardial failure, with advanced anasarca, is the cause of death. Others die of complications, such as broncho-pneumonia.

Prognosis and Treatment.—The outlook depends on the causative factor. In early cases in which syphilis has been established as the cause of the inflammation of the pulmonary artery, anti-syphilitic treatment will retard the progress of the disease. In paroxysms of cyanosis, venesection gives marked relief. The usual treatment for congestive heart failure must be adopted, when this has supervened.

PHLEBITIS

Phlebitis or inflammation of the veins may be sharply divided into two great classes—(1) non-suppurative or plastic, and (2) suppurative. The terms endo- and peri-phlebitis have been used to indicate inflammation of the internal and external coats. Peri-phlebitis results from invasion of the veins by inflammatory processes outside it, or from injury. It may extend inwards towards the lumen of the vein, and result in endophlebitis and generally clotting of the blood within the vein. Endophlebitis is usually the result of poisons or microbes circulating within the vein. Inflammatory changes of a plastic type occur in the endothelium, and in consequence a clot or thrombus is set up within the vein. The clot may adhere to the vessel wall and completely obliterate it. Organisation of the clot by fibrous tissue may occur, the vein being transformed into a hard fibrous cord. In other cases the clot may become softened and broken down, and the circulation may be resumed through the vein. In certain cases changes in the composition of the blood may lead to clotting in a vein, and the presence of this clot itself may give rise to a plastic phlebitis; this is sometimes called *thrombo-phlebitis*. In other cases the vein and contained clot may become invaded by pyogenic organisms, and leucocytes will enter the clot and cause it to break down into a purulent fluid.

PLASTIC PHLEBITIS

Ætiology.—(1) Traumatic phlebitis; (2) the formation of a non-infective clot—thrombo-phlebitis; (3) gouty phlebitis may accompany an attack of gouty arthritis or may occur independently; (4) typhoid fever not infrequently causes phlebitis and thrombosis; (5) in pneumonia and influenza phlebitis is not an uncommon complication; (6) post-operative phlebitis is not at all uncommon in cases of operation on the lower abdomen and the

bladder; and (7) puerperal phlebitis or phlegmasia alba dolens frequently follows parturition.

Phlebitis may attack any vein, but is most common in the lower limb, particularly in the saphena vein.

Symptoms.—Phlebitis is accompanied by pain and tenderness in the course of the affected vein, which can be felt as a hard cord. The skin may become reddened over the superficial veins, and the limb is often œdematous when thrombosis has taken place. There is usually more or less febrile disturbance. In gouty phlebitis, the pain is often severe, the areas of inflammation are often multiple; there is a great tendency for more than one vein to be attacked at once, and, in opposition to most forms of phlebitis, the disease may be symmetrical.

Complications and Sequelæ.—The complications and sequelæ of plastic phlebitis are those of the thrombosis that accompanies it, and will be described under thrombosis.

Prognosis.—This is generally good apart from the risk of embolism or of the thrombosis spreading towards the large veins, such as superior or inferior vena cava. Treatment must be carefully carried out to minimize the risk of this.

Treatment.—Patients with phlebitis should be put to bed, and the limbs elevated and wrapped in cotton wool. All sudden movement, friction, or handling should be avoided. The bowels should be freely opened, as chronic constipation and stasis in the colon may impede the circulation in the iliac veins. In gouty phlebitis, the diet should be restricted to fish and light farinaceous foods, but when patients are marasmic and anæmic, the diet should be as liberal as their digestive powers will permit. In all cases of phlebitis foods containing much lime-salts, such as milk, should be limited. Potassium or sodium citrate with carbonate of ammonia and liquor ammonii acetatis are of service. Glycerin of belladonna smeared over the inflamed vein appears to ease the pain.

SUPPURATIVE PHLEBITIS

Ætiology.—Suppurative phlebitis is the result of infection of the walls of the veins with pyogenic organisms. The micrococci may be in the circulating blood, as in some cases of puerperal phlebitis, or they may reach the veins from a focus of suppuration around it, as in facial carbuncle, middle-ear disease, or inflammation of the portal veins—suppurative pylephlebitis.

Pathology.—The coats of the vein are infiltrated with leucocytes, the clot which has formed within the vessel breaks down into yellow pus, and abscesses are not infrequently found along the course of the vein. Not uncommonly the septic inflammation spreads along a vein, splitting up the coats.

Symptoms.—There is a throbbing, smarting pain in the region of the affected vein, and the part drained by it is œdematous. Not infrequently the septic process spreads along a vessel. There is often fever, a rapid pulse, a dry tongue, and delirium, and in many cases a succession of rigors indicates the development of pyæmia.

Complications and Sequelæ.—These depend upon the situation of the vein and the occurrence of emboli, owing to breaking away of the softened

clot. (See Thrombosis.) When the vein is superficial the diagnosis is easy, but when a deeply-seated small vein is affected the only symptoms may be those of the pyæmia to which it gives rise.

Treatment.—The prevention of this disease by asepsis is one of the great advances in modern surgery. As soon as the disease is recognised, a ligature should be placed upon the vein between the affected area and the heart, the inflamed vein should be thoroughly laid open, the septic clot removed and the cavity thoroughly cleansed. In some cases where numerous abscesses are formed, amputation is the only means of arresting the general infection.

Thrombosis of the portal vein is commonly the result of septic conditions within the abdomen, most often suppuration in the region of the appendix. The condition is not always acute. Peptic ulcers may lead to portal thrombosis, and tubercular glands along the course of the portal vein have been found to cause clotting within the lumen of the vessel. Dysenteric ulcers resulting from bacillary dysentery may also give rise to septic portal thrombosis, but typhoid ulceration very rarely does so.

The effect of portal thrombosis is to produce a portal pyæmia, portions of the clot passing into the liver and causing abscesses within the radicles of that organ. Occasionally the portal vein itself becomes converted into a sac containing pus, and the liver is then riddled with abscesses along the course of the portal branches. This condition is known as *portal pylephlebitis*.

Symptoms.—The symptoms of septic portal thrombosis are the occurrence of fever and of rigors. The liver becomes enlarged and tender, and jaundice of a slight degree is quite common.

As the condition is such a generalised one, recovery very seldom takes place, even if treatment is adopted.

Treatment.—This consists in removing the cause and incising any liver abscesses.

MAURICE CAMPBELL.

THROMBOSIS AND EMBOLISM

Thrombosis is the name applied to the coagulation of blood within living vessels, whether in the heart, the veins or the arteries.

Embolism is the process whereby a portion of clot or other substance, such as parasites, fat globules, masses of bacteria or particles of tumour, is carried from one part of the circulation to another, and is impacted when it arrives at a vessel too narrow for its further progress. An infarct is the degenerated or necrosed condition of the tissues due to interference with the circulation of blood within it, and can be caused by embolism, thrombosis, endarteritis, endophlebitis or strangulation of veins. An infarct is generally wedge-shaped in outline, with the base towards the periphery of the organ affected. As seen post mortem, it is either yellowish-white in colour (the white infarct), or blood-red in colour (the hæmorrhagic infarct).

In the case of the white infarct the tissue deprived of its blood supply becomes permeated with lymph from the surrounding living tissue, and coagulative changes take place in it. In the case of the kidney or spleen, the

coagulable material is sufficient to render the infarct hard. In the case of the brain, less coagulable lymph is poured out, and the area of the brain affected becomes softened. In the early stages there is often a zone of congested vessels around a white infarct; this is a reaction on the part of the surrounding living tissues to the presence of the dead material. Later the infarct becomes invaded by fibrous tissue and a scar results.

In hæmorrhagic infarct, coagulation and necrosis also take place, but to this is added hæmorrhage, by diapedesis of the red blood cells from vessels of the collateral circulation. A hæmorrhagic infarct is commonly seen in the lung; a cone-shaped area of lung tissue becomes hard and dark-red in colour. Should the embolus, instead of being aseptic, contain living micro-organisms, a septic process is set up within the infarct and an abscess results. Such purulent abscesses are commonly seen in the lungs, as the result of septic phlebitis, and occasionally in the systemic system, as the result of septic endocarditis.

THROMBOSIS

Ætiology.—The causes of thrombosis are—(1) altered conditions of the blood or increase in its coagulability; (2) slowing of the current of the blood within the vessels; and (3) a lesion of the lining membranes of the vessel or cavity of the heart. Thus, thrombosis may occur in cases of anæmia or after infections or operations, where the blood is more coagulable than normal; in the appendages of the dilated auricles of the heart where the movement of blood is feeble or in peripheral vessels or in the lungs when the circulation as a whole is feeble, *e.g.* with a failing heart; and it may result from inflammation of the lining of the vessel or degenerative changes in its endothelium, especially when there is atheroma associated with much narrowing of the channel obstructing the circulation.

(1) **INTRA-CARDIAC THROMBOSIS.**—This is one of the commonest forms of thrombosis and is very important. It occurs in the left auricle, when it has become extremely dilated as the result of mitral stenosis. The ante-mortem clot generally begins to form in the dilated appendix of the left auricle, but may extend by the deposition of excessive layers of fibrin to invade the auricle itself so that a large ball thrombosis be formed within the cavity. Portions of the ante-mortem clot may break away from the thrombus, and may be carried into the left ventricle and into the general circulation, and embolism may occur in the brain, spleen, kidneys, intestines and the main arteries of the limbs. This danger is mainly to be feared in the first few weeks after the deposition of the thrombus, but may of course recur as fresh thrombi form.

Ante-mortem clot is occasionally deposited among the meshes and cavities of the dilated left ventricle. In cases of cardiac fibrosis and myelomalacia cordis, the result of disease of the coronary arteries, and especially after coronary thrombosis and cardiac infarction, the lining of the heart may become affected and fibrinous deposits occur within either ventricle. Lastly, in septic endocarditis, large vegetations, consisting of clot and masses of micro-organisms, may occur. Inflammation of the ventricle and of the auricle may also be present, and ante-mortem clot may be deposited on these roughened surfaces. Portions of this clot may leave the ventricle and pass into the

general arterial circulation, where the effect produced will depend upon whether the emboli are aseptic or contain micro-organisms.

Thrombosis in the right auricle occurs in many conditions where there is gradual cardiac failure and dilatation of the right side of the heart. Portions of ante-mortem clot form in the right auricular appendix, and parts may break away and pass into the lungs and an embolism of the pulmonary artery result. If the embolus is sufficiently large to cause blocking of the artery or of one of its main branches, sudden death ensues, but if only one of the smaller branches is affected, a pulmonary infarct results. Much more rarely ulcerative endocarditis occurs on the right side of the heart, and portions of the valves or affected clot reach the lungs in the same way.

(2) **ARTERIAL THROMBOSIS.**—Arterial thrombosis is rarer than venous thrombosis. It is sometimes due to arterial embolism, but is also the result of trauma or disease of the arterial walls, such as atheroma or endarteritis.

Thrombosis of the coronary arteries is a very important condition, since it is a frequent cause of sudden death and of serious cardiac disability. It has already been described (pp. 991–994). The usual artery affected is the anterior interventricular branch of the coronary artery. Atheromatous plaques are constantly found within the thrombosed vessel, and sometimes atheroma has occluded the orifice of the artery. In cases where the circulation has been slowed and greatly diminished before the final clotting, changes in the wall of the left ventricle are very common. Syphilitic mesoarteritis sometimes occludes the orifices of the coronary arteries but this is much less common.

Thrombosis is very frequent in the small cerebral arteries, especially when they have become narrowed as the result of disease. In early middle-age this narrowing is usually the result of syphilitic endarteritis, but most patients are older and then the arterial lumen is diminished by atheromatous changes in the wall of the vessel.

Thrombosis of the main artery of a limb usually results in gangrene; the limb becomes first white and pallid, later mottled in appearance, and finally black. If the patient survives the immediate shock and the disease to which the thrombus owes its origin, a line of demarcation will form between the vital and devitalised tissues, and the limb should be amputated well above this level.

Thrombosis of the retinal artery is more common than embolism. There is a sudden painless loss of sight in one eye, and generally the blindness is permanent and complete. Within a short time there is opacity of the central parts of the retina, and the macula shows up by contrast as a bright cherry-red spot.

(3) **VENOUS THROMBOSIS.**—*Thrombosis of the lateral sinus* occurs in disease of the middle ear. The mastoid cells become infected with pyogenic organisms and the disease spreads to the petrosal or sigmoid sinus. The clot in the vein becomes softened by pyogenic organisms, and particles break away and are conveyed to the lungs, in which pyogenic abscesses are formed.

The symptoms of septic thrombosis of the lateral sinus—and its continuation of the jugular vein—are infiltration of the tissues of the neck, with a cord-like induration of the vein itself, with some restriction of the movements of the head. There is a history of a chronic and often offensive discharge from the ear of the same side. A high temperature and rigors,

due to flooding of the circulation by poison, occur when a portion of the septic clot is dislodged. In these circumstances the jugular vein should at once be ligatured below the clot, in order to prevent further portions of the clot gaining access to the blood stream. A radical mastoid operation should also be performed, the sinus opened and its septic contents removed.

Thrombosis of the longitudinal sinus of the brain occurs as the result of injuries and infected wounds of the skull. It is a common war injury, the vertex of the head having been injured by a bullet as the soldier passes along the trench. Thrombosis of the cranial sinuses also occurs in marasmic patients, but it is usually agonal.

In many of these cases, owing to the vertical position of the leg areas in the brain, a paraplegia is produced, while the arms are not affected. The condition should be treated by trephining and draining the cranial cavity. Occasionally the longitudinal sinus becomes thrombosed in septic conditions in children and also in chlorotic anæmia in adults.

Thrombosis of the cavernous sinus is not infrequently the result of the extension of a chronic suppurative process of the sphenoidal cells at the back of the nose. The cavernous sinus is also affected by septic processes on the face; a small boil on the nose or a mosquito sting on the face may produce a septic thrombosis of the angular vein; this vein communicates with the ophthalmic vein, and the septic clot may extend along the latter into the cavernous sinus. As the venous plexuses of the pterygoid and zygomatic fossæ communicate through the foramina in the middle fossa and by the inferior ophthalmic vein, purulent inflammation of the jaw and of the teeth sockets is sometimes a cause of cavernous thrombosis. The result is that a marked degree of exophthalmos and swelling of the lids, and œdema of the optic disc and extensive retinal hæmorrhages occur. Not infrequently the septic condition of one cavernous sinus spreads to that on the other side through the circular sinus, and the exophthalmos may be double. Death from pyæmia or meningitis results. Owing to the position of the sinus, operation is impossible.

Thrombosis of the central retinal vein is common in elderly patients with athero-sclerosis and high blood pressure. The loss of sight is not so sudden or complete as in blockage of the artery. There is often albuminuria. Glaucoma often develops, but if the thrombosis is in a tributary vein the degree of recovery may sometimes be fairly good.

Femoral thrombosis is perhaps the commonest form of thrombosis. It frequently occurs after parturition and in anæmic and marasmic states. It is met with after infectious fevers, especially after typhoid fever, more rarely after influenza and pneumonia. It also follows operations, especially if a septic condition has been dealt with, or results from the operation. The thrombosis generally occurs in the femoral vein, and there is often some rise of temperature and a slight rise in pulse rate. It is most common on the left, because of the pressure of the right common iliac artery on the left common iliac vein. The limb affected becomes œdematous and a hard cord is found in the course of the vein.

In other cases thrombosis of the veins may also occur in marasmic conditions secondary to carcinoma, tuberculosis and tertiary syphilis.

Complications and Sequelæ.—Collateral circulation is usually satisfactorily accomplished in femoral thrombosis. In cases where the arteries

as well as the veins are involved gangrene may occur. Occasionally the thrombosis may spread up into the iliac vein and into the inferior vena cava, in which case both legs may become swollen and œdematous. Even in cases where the inferior vena cava has become thrombosed, recovery may take place, collateral circulation being established by means of veins passing up from the legs into the axillæ. If, however, the clot reaches the entrance to the renal veins, death nearly always results from renal thrombosis. Embolism is not at all uncommon in femoral thrombosis, the clot passing into the right auricle, and then into the right ventricle and pulmonary artery.

Treatment.—Complete rest for at least 3 weeks, as a precaution against embolism, should be insisted upon. Limitation of foods containing quantities of lime salts, such as milk, and the administration of citrates and salts of ammonia will materially help in the treatment of the case. The leg should be elevated and wrapped in cotton wool and kept warm.

EMBOLISM

Embolism may occur in three main situations, namely—(1) in the systemic circulation; (2) in the pulmonary circulation; and (3) in the portal circulation.

Emboli in the systemic circulation are derived from ante-mortem clots in the left auricle and left ventricle. These clots are formed in cases of mitral stenosis and more rarely in mitral regurgitation, and also when the left ventricle is greatly dilated and hypertrophied. In these cases the emboli are aseptic. Systemic emboli also occur in septic endocarditis, when portions of the valve break away or masses of fibrin and micro-organisms pass into the general circulation. These emboli are septic, and when they reach their destination usually form abscesses.

Emboli in the pulmonary circulation may have their origin in clots formed within the right auricle and right ventricle, or more rarely from septic endocarditis of the tricuspid and pulmonary valves; they may also come from any part of the systemic venous system. A very important form of pulmonary embolism is met with after abdominal operations and after childbirth. About the tenth day after an apparently successful abdominal operation or an uneventful parturition, a pulmonary embolism may occur with appalling suddenness. Death may take place at once, or hæmoptysis and pleurisy supervene. The clot forms in the common iliac vein, at the junction of the internal and external iliaes. Not only may a clot pass along the veins, but we also get droplets of fat in fat embolism, air bubbles in air embolism, and masses of parasites in parasitic embolism.

Two recent studies, one of autopsy cases of hæmorrhagic infarct of the lung and one of post-operative cases of thrombosis and embolism, support the view that embolism is much the most common cause of pulmonary infarction (87 per cent.) and that this generally comes from a venous thrombosis. In 14 per cent. of the autopsy cases a massive pulmonary embolism was the main diagnosis and most of these were after operations or fractures. In 47 per cent. heart disease with the accompanying stagnation of the blood stream appeared to be a predisposing factor. A potential source for the

embolus was found in 75 per cent., most often in the pelvic or leg veins. Massive embolism was more likely to occur on the tenth day but was not infrequent at any time in the first fifteen days ; it was independent of sepsis and depended on the stagnation of the blood stream, either from the state of the heart or more often from the mechanical conditions of rest. Smaller infarcts were more closely associated with sepsis (which suggests that the embolism is more often due to the breaking off of a small part of the thrombus) and more erratic in the time of their appearance.

In embolism in the portal circulation portions of clot in the radicles of the portal vein are finally arrested in the liver.

Symptoms.—1. EMBOLISM OF THE CEREBRAL ARTERIES.—The onset is sudden, and the left side of the brain is rather more often affected than the right. Hemiplegia or aphasia is produced ; consciousness is lost only for a few minutes during the attacks.

2. EMBOLISM OF THE SPLENIC ARTERY.—The onset is sudden, with pain in the left side and sudden enlargement of the spleen, which is very tender. During the next few days, the spleen diminishes in size, but it often remains permanently enlarged.

3. EMBOLISM OF THE RENAL ARTERIES.—Sudden pain in the back is produced, and blood and a little albumin are present in the urine. The hæmaturia may last for a day or two, or longer if a larger branch was involved.

4. EMBOLISM OF THE SUPERIOR MESENTERIC ARTERY.—The patient is seized with sudden, violent abdominal pain and distension. The collateral circulation, in spite of the numerous vascular arteries that supply the intestine, fails, and gangrene of the small intestine results. There is a complete intestinal obstruction, and blood finds its way into the stools and into the peritoneal cavity. Operation should be undertaken at once, but owing to the large amount of bowel affected recovery is very rare.

5. EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.—This is not an uncommon event in cases of mitral stenosis and in septic endocarditis of the aortic and mitral valves. The patient is seized with pain in the eye and becomes suddenly blind on one side. The optic disk becomes pale and the retinal vessels small. Occasionally only a single branch of the vessel is affected. The same picture is found more often as a result of arterial thrombosis in elderly patients with athero-sclerosis or high blood pressure.

6. EMBOLISM OF A LARGE ARTERY IN A LIMB.—This sometimes occurs. There is acute pain in the limb, followed by numbness and loss of power. The pulse is imperceptible below the seat of the embolism, and gangrene results.

7. AIR EMBOLISM.—Air may enter the veins during operation on a large vein, or during intravenous injection of saline or other solutions, or after distension of the bladder and the urethra with air. Air embolism is undoubtedly capable of causing death, but a small quantity may enter a vein without any effect whatever, or, if some disturbance should arise, without fatal termination. The exact way in which air embolism causes death is doubtful ; it may be due to arrest of the pulmonary circulation or to cerebral anæmia.

The diagnosis is not difficult ; the respiratory embarrassment, convulsions, the feeble pulse and the characteristic sound upon air entry into the veins are usually sufficient.

Treatment consists in immediately occluding the vein into which the air has entered. Stimulants should be administered hypodermically and nitrite of amyl inhaled. Venesection may be used to relieve the embarrassment of the heart.

8. **FAT EMBOLISM**—Fat may reach the blood vessels in cases of fracture of bones and in cases of hæmorrhage into or rupture of the liver. The fracture is usually situated in the long bones, generally in the tibia or femur, and occasionally in the ribs. Fat embolism may occur within a few hours of fracture of the bones.

The fat droplets first lodge in the capillaries of the lungs. Occasionally they are forced on through the lungs into the general circulation, and the glomeruli of the kidney may be plugged with fat cells, and they may also lodge in the brain or spinal cord.

The patient becomes cyanosed, and crepitations from œdema of the lungs may be heard at the bases. The temperature remains normal. Cerebral complications, such as delirium, coma and rarely localised paralysis, may be found. The urine should always be examined, as oil drops have been detected by staining with osmic acid, and also the retina, as in one case the fat drops were recognised in the retinal vessels before death.

When fat embolism occurs within a few hours of fracture of the bones, it has to be diagnosed from the general shock of the accident. It is doubtful whether fat embolism in the lungs can alone cause death; more probably this is due to the disturbance of the kidneys or lesions of the brain.

The indication for treatment is to sustain the heart. Nitrite of amyl is often useful, and inhalations of oxygen should be given to lessen cyanosis.

9. **PARADOXICAL EMBOLISM**.—In certain cases of venous thrombosis, emboli occur not only in the lungs but also in the systemic arteries. It has been shown that in these cases the embolus has passed from the right auricle to the left auricle through a patent foramen ovale. These crossed or paradoxical emboli are often preceded by pulmonary embolism, which causes a rise in pressure in the right auricle and a fall in the left auricle, so that the embolus can pass from the right to the left side of the heart.

MAURICE CAMPBELL.

ARTERIAL BLOOD PRESSURE

HYPERTENSION

In hypertension the systolic and diastolic blood pressure readings are persistently—not necessarily permanently—above certain levels, from whatever cause. Authorities differ somewhat as regards these levels, for the limits of normal variation are fairly wide, and, moreover, it is now practically universally believed that the blood pressure does not normally increase with advancing age as much as was previously thought to be the case; but it is generally agreed that persistent readings above 150 and 90, or at any rate 160 and 90, in adult males, and 170 and 100 in the elderly are pathological.

It is important to note that a transient increase in the blood pressure

readings, especially of the systolic, may occur with nervous excitement, such as is not infrequently incidental to a medical examination.

The late Sir Clifford Allbutt introduced the term *hyperpiesia* to denote a clinical condition in which there is a persistently raised blood pressure independent of renal disease. Some writers use the term *essential* or *primary* hypertension, or *hypertensive cardiovascular disease* (Janeway). The increased blood pressure is the essential or primary condition. Any changes which may be in the walls of the blood vessels, the heart, the kidneys or other organs are either a secondary result of the hypertension, or co-existent with it, and in the latter case may or may not be due to the same cause as is the hypertension.

Ætiology and Pathology.—The ætiology and pathology of hyperpiesia is obscure, and constitutes one of the most important and difficult questions in clinical medicine.

An inherited constitution is an important factor, as revealed by the high familial incidence in certain cases. The malady is more common in late middle life; in those who tend to worry, to be over-anxious, to take things too seriously, or are of an excitable disposition; in those whose manner of life involves continued mental or emotional stress or strain; and in the obese. It may be the result of poisons circulating in the blood, which act as pressor substances, such as occurs in endocrine disturbances, for example, at the female menopause, in tumours of the adrenal cortex or medulla, with Cushing's Basophilism Syndrome (pituitary basophilism), and in acromegaly; in focal sepsis; and in chronic alcoholic excess and excessive smoking. The general consensus of opinion is that a high protein diet intake in itself is not a cause, but in my view it is.

With regard to the pathogenesis of hyperpiesia, it is generally agreed that the immediate cause of the condition is an increased resistance to the passage of blood through the smaller arteries, especially the arterioles, at first due to hypertonus on their part. If the latter does not cease, either spontaneously or because of therapeutic measures, then, sooner or later, cardiac hypertrophy, especially of the left ventricle, and diffuse hyperplastic sclerosis supervene (see page 1035). The latter gives rise to actual narrowing of the lumen of the blood vessels, which may even be great, and a resultant further increase in the resistance to the passage of blood.

The cause of the initiation of the hypertonus of the arteries cannot yet be explained.

It is to be noted that some now consider that hyperpiesia is primarily of renal origin. Among the reasons adduced for this view are that some cases, in the course of time, develop into what is termed "malignant" hypertension. It may here be observed that this only occurs in a small proportion of cases. The matter will be dealt with further later on. Again, experimentally produced renal ischæmia in dogs, the result of the gradual narrowing of the renal arteries by means of clamps, will give a condition similar to hyperpiesia in man. With regard to this, it may be pointed out that pressor substances circulating in the blood from any source, including the kidneys, will have a like effect.

By far the general consensus of opinion—which I myself share—is that it is not possible to explain hyperpiesia as being due to structural changes in the kidneys, in which connection the following observations may be

made. (1) In hyperpiesia the blood pressure becomes normal under ether anæsthesia, and is diminished during sleep. (2) Hyperpiesia and diffuse hyperplastic sclerosis may occur independently of any renal lesion. (3) While in hyperpiesia and diffuse hyperplastic sclerosis the kidneys generally become involved secondarily, it may be in marked degree, with consequent increased obstruction to the circulation and an increase in the hypertension, in a large proportion of cases there is generally little or no impairment of renal function, which is one of the most characteristic features of hyperpiesia, and less than 10 per cent. die from renal failure. (4) There is frequently an absence of hypertension in polycystic and tuberculous disease of the kidney, occasionally in acute nephritis, and rarely in chronic nephritis. (5) In chronic nephritis changes may take place in the renal vessels of an almost exactly similar nature to those in diffuse hyperplastic sclerosis, leading to hypertension and similar cardiovascular accidents. But there the vascular changes are secondary to the nephritis.

Hypertension is not by any means only the result of kidney disease. There are many other causes. Indeed, it may even be asked whether in some cases of hypertension and diffuse hyperplastic sclerosis, and chronic nephritis, the former may have been the cause of the latter? Or, again, may both be the result of a common cause?

While the great majority of cases of hypertension are of the nature of hyperpiesia, the condition may also be due to diffuse hyperplastic sclerosis (see page 1035), chronic and acute nephritis, polycystic disease of the kidney, increased intracranial pressure, *e.g.* intracranial hæmorrhage, and polycythæmia vera. With regard to the first, however, the condition is rather the result than the cause.

Symptoms.—In hyperpiesia the patient is often well nourished and may be plethoric. There may be a complete absence of subjective symptoms for some years, and the condition may be discovered accidentally, *e.g.* during an examination for the purpose of life assurance.

Usually the onset of symptoms is insidious. The most common early symptoms are a feeling of fullness and it may be of throbbing in the head, transient giddiness, tinnitus aurium, flushing, insomnia, palpitation, languor and early fatigue, the latter especially on mental effort, headache, of a dull aching or it may be of a throbbing character, particularly in the occipital region and especially in the morning, impairment of memory, mental irritability and diminished emotional control. The blood pressure readings are above those mentioned in the first paragraph.

The condition may subside, either spontaneously or the result of therapeutic measures. If not, the blood pressure readings usually increase, and the systolic may reach 240 mm. or even more. Consciousness of the action of the heart may become more prominent. Sooner or later, there is evidence of hypertrophy of the heart, especially of the left ventricle, and of diffuse hyperplastic sclerosis (see page 994). The walls of the radial, the brachial and temporal arteries may be felt to be uniformly thickened—the so-called “whip-cord” artery. The degree of hardening of the arteries is found on palpation to vary at different times. The artery feels hardest when the vessel is most contracted and consequently smallest. The character of the pulse is that of high-tension. The retinal arteries are often thickened, and on ophthalmoscopic examination may show a glistening light along their

course—the so-called “silver wire” arteries, due to reflection of light from the thickened vessel. Even more important is a visible irregularity in calibre of the arteries best seen near the disc. Further, at the arterio-venous crossings there may be seen an obstruction to the flow of blood through it, leading to distension of the peripheral part of the vein and also to deviation of the vein where it crosses the artery.

The subsequent clinical picture and course exhibit considerable diversity.

The cardiac symptoms are by far the most common. The most frequent are shortness of breath, palpitation and precordial discomfort or pain, referred to exertion. Later, cardiac failure, usually left-sided but it may be involving both sides, may supervene (see page 857). Acute left-sided failure may occur (see page 857). Angina pectoris is not very infrequent. There is occasionally coronary occlusion. For further particulars, the reader is referred to page 857.

Various gastro-intestinal symptoms are often present. There may be hæmorrhages from the nose, the stomach, the intestines and other mucous membranes, the kidneys, the conjunctiva, and occasionally the retina, which are sometimes flame-shaped. There may be tingling, numbness and cramp of the limbs, and occasionally intermittent claudication. Hypertensive encephalopathy (see page 1607) may occur. Cerebral hæmorrhage may be a terminal event.

Not infrequently there is albuminuria, generally slight and not constant, and there may be polyuria and nocturnal micturition, and hyaline and granular casts. There is usually no or little impairment of renal function. Uræmia is rarely a terminal event.

There is a small group of cases of hypertension in which, instead of the symptoms being mild or moderate, the course slow, and little or no renal insufficiency, the symptoms are severe, the course is progressively rapid, and the patients die from renal failure. The former, which constitutes the great majority, is called benign and the latter “malignant” hypertension. There are also intermediate forms between the two groups. The renal changes in malignant hypertension are described as malignant nephrosclerosis, and those in benign hypertension may be termed benign nephrosclerosis. It may be remarked, however, that the term malignant is not altogether an appropriate one.

MALIGNANT HYPERTENSION.—There appear to be two forms of this, namely: (1) In a case of benign hypertension of a varying number of years' duration, the symptoms suddenly become severe and the malady pursues a rapid course. (2) In an individual who has not been aware that he had hypertension, from the outset there are severe symptoms in association with hypertension and the course is rapid. The second usually occurs at an earlier age than the first.

There are the same changes in the kidneys as in the benign form, and, in addition, those which are believed to be characteristic, *i.e.* acute necrosis of the walls of the arterioles and also often of the root of the glomeruli, and it may be resultant thrombosis of the glomeruli. While acute necrosis is most marked in the vessels of the kidneys it is also found in those of other organs. There has been much discussion among pathologists regarding the interpretation of the distinctive changes in the kidneys. In the opinion of some, they are an intensified result of the ætiological factor of the changes

in the walls of the arteries in the benign form ; while others think they are due to a special toxin. Again, some are of opinion that malignant hypertension is merely a severe or extreme phase and a terminal result of the benign variety, whereas others think that it is a separate entity.

In malignant hypertension, the blood pressure, especially the diastolic, is usually materially higher than in the case of the benign form, being generally over 200-120, and may be very high and may become extreme. There is lassitude, anorexia, frequently loss of weight, severe headache and anæmia. Changes in the retina occur earlier and are more marked, hæmorrhages being more evident and retinopathy is frequent if not usual. There is polyuria and nocturnal micturition. The urine is of low specific gravity, and contains hyaline and granular casts, a variable amount of albumin and often red blood cells. As the disease progresses, there is evidence of renal impairment. Death occurs from uræmia, cardiac causes or cerebral hæmorrhage.

In hyperpiesia it is probable that more than half the patients die from cardiac causes—in the great majority from congestive failure, not infrequently from angina pectoris, and occasionally from coronary occlusion ; in a fair proportion of cases, cerebral hæmorrhage is a terminal event ; in less than 10 per cent. uræmia ; while in the remainder the cause of death is some intercurrent disease.

Diagnosis.—Hypertension is recognised by persistent blood pressure readings above those mentioned in the first paragraph of this article.

The diagnosis between hyperpiesia and malignant hypertension may readily be made from a consideration of the respective clinical features described above. That between malignant hypertension and chronic glomerular nephritis may be difficult.

Prognosis.—Hyperpiesia usually pursues a slow course. It is probable that the duration of life after the beginning of symptoms is ten to fifteen years, or even more. The outlook is chiefly affected by the blood pressure readings, particularly the diastolic, the state of the heart and of the arteries, and the patient's manner of life. See also page 996. The duration of life in malignant hypertension is usually at the most two years.

Treatment.—As regards the treatment of hyperpiesia, the first indication is a thorough review of the ætiology. Then, the question as to whether a preliminary rest and, if so, how much is advisable should be considered (see page 862).

Hyperpiesia is an excellent example of the value of treating not only the disease but the patient. He should be reassured and encouraged. It is inadvisable to let him know the exact blood pressure readings at any time.

In no cardiovascular malady, excepting perhaps in angina pectoris, is the manner of life of so great consequence.

The cardinal indications are that, while the patient's activities should not be unnecessarily curtailed, they should come within the limits of his strength and even keep something in reserve ; there should be a sufficiency of rest in his life ; and all causes of mental and emotional stress should be carefully avoided. With this object in view, inquiry should be made regarding the patient's environment, the nature and hours of his work, his habits, the question of sleep and other relevant matters, while his temperament should be taken into consideration.

Each patient should be in bed for at least ten hours each night, rest physically and mentally for at least half an hour after the midday meal and have a quiet day each week. If the blood pressure is more pronounced, he should be in bed for at least twelve hours, rest for at least an hour after lunch, and stay in bed on one day a week with, it may be, a diet limited to vegetables, fruit and milk. In addition to an annual holiday of good length, several shorter ones during the year are advisable, in each case with a sufficient element of rest. As indicated, physical exertion and mental effort should be within the limits of the patient's strength (see page 863), with something in reserve. With this proviso, walking in the open air, riding a non-pulling horse, golf and mild bicycling are suitable forms of the former. The patient should be encouraged not to worry, or be over-anxious, or to take things too seriously; to avoid all excitement and other forms of emotional stress; and cultivate the art of "relaxing," physically and mentally.

If the patient tends to worry, or be over-anxious, or to take things too seriously, sedatives (see page 863) are often of special value. The matter of sleep is also of particular importance, and if there is insomnia, it should be treated on the lines laid down on page 863. Again, any obesity should, without fail, be corrected.

The amount of fluid with meals should be diminished, while an ample quantity should be taken between meals. Some prefer the mildly alkaline waters. The benefit of a regulated diet of moderate latitude may be considerable. Its amount should preferably be rather less than is needed. Those articles of food which especially stimulate the cardiovascular system should be reduced and proportionately to the degree of hypertension. The quantity of beef and mutton should be diminished, while twice-cooked meat, salted and preserved meat, liver, kidney, brains, sweetbreads, meat soup, and gravies, and meat extractives are better avoided altogether. Fish, poultry, game (not high), rabbit, vegetables, salads and fresh fruit are suitable. Ruthmol may be used as a substitute for table salt with meals. Moderation in the use of tobacco, tea and especially of coffee should be enjoined. Alcohol is better avoided altogether.

Strict attention to the condition of the bowels, preferably by a saline each morning, and a mercurial preparation at bedtime once or twice weekly are indicated.

Cold and hot baths are contra-indicated; but a warm bath, the temperature of which is gradually lowered, may be taken daily. A Turkish bath once or twice a week may be helpful. Patients often derive much benefit from a course of treatment at a Spa where different kinds of baths are used, on account of the rest, the change of air, the regular mode of life and exercise, the careful dieting and other factors, and in such cases periodic visits are to be recommended.

Iodine, in some form, in small doses appears to be occasionally of help. The same perhaps applies to the nitrites, such as a combination of sodium nitrite, erythrityl tetranitrate and mannityl hexanitrate, when symptoms are present. I have been disappointed with the results of the administration of mistletoe, liver extract, and potassium and sodium thiocyanate.

Diathermy, high frequency currents and other forms of physiotherapy are employed by some.

When the blood pressure is very much raised, particularly if there are

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any head symptoms, venesection may be of great benefit as a temporary measure, especially in plethoric patients. At least a pint of blood should be withdrawn. If required, this method of treatment may be repeated at intervals. It is also indicated when there are manifestations of great distension of the right side of the heart.

If the foregoing measures fail, a prolonged holiday, or complete rest in bed and a diet restricted to vegetables, fruit and milk for 2 to 4 weeks should be tried.

During the last few years a variety of surgical measures have been introduced in the treatment of hyperpiesia with the object of denervation of the kidneys, the suprarenal glands and the splanchnic area. It would appear that (a) the only procedures which afford promise of some success are bilateral section of the splanchnic nerves and decapsulation; (b) a very careful selection of cases is required; and (c) only those which are severe and unaccompanied by cardiac and renal failure should be considered. Surgical treatment of hyperpiesia is in its experimental stage but deserves further investigation.

For headache in hyperpiesia, cold compresses, or the application of menthol, or a cup of tea, or phenacetin and citrate of caffeine, failing which a tablet of trinitrin or one of the other nitrites may be used.

For the treatment of cardiac failure in hyperpiesia, see page 996. That of hypertensive encephalopathy is described on page 1608. For the treatment of renal failure, see pages 1327, 1328.

In conclusion, I would repeat that in the treatment of a case of hyperpiesia the patient's manner of life is of the greatest consequence and its importance can scarcely be exaggerated.

HYPOTENSION

In hypotension the systolic blood pressure is persistently—not necessarily permanently—below 110 mm. in adult males and 105 mm. in adult females, from whatever cause. Usually the diastolic pressure is proportionately less affected.

Ætiology and Pathology.—Hypotension may be physiological, that is, in some healthy individuals the blood pressure is persistently below the level mentioned.

The pathological form of hypotension may occur in a variety of conditions, such as: endocrine glandular disturbances, for example, Addison's disease; acute infective diseases, especially diphtheria; in pulmonary tuberculosis; in shock and collapse; in focal sepsis; in most cases of fatty degeneration and fatty infiltration, and in some of fibrosis of the myocardium; in some cases of chronic valvular disease; in coronary occlusion; in anæmia; and in cachexia and malnutrition.

The chief factors in the causation of hypotension are: diminished contractile force of the left ventricle; and, more frequently, vaso-dilatation, especially of the splanchnic area.

Symptoms.—Among these may be languor; early fatigue on physical and mental effort, and on maintaining the upright position; giddiness and faintness, especially on change of posture; a tendency to mental depression

and headache ; coldness and pallor or lividity of the extremities ; and an abnormal response of the cardiac rate with the assumption of the upright position after lying down. In splanchnic stasis, pressure on the abdomen by the hand while the patient is lying down is sometimes accompanied by distension of the jugular veins.

Treatment.—The ætiology should be reviewed. In acute cases, rest in bed for a time is advisable. In others, care should be taken to avoid over-exertion, physical or mental ; and it is an advantage to rest after effort, and before and after meals. A tepid or cold sponge bath in the morning, followed by simple exercises, may be helpful. An ample amount of nutritious and easily assimilable food, with an increase of those articles which stimulate the cardiovascular system, such as meat, and the periodic administration of general tonics are indicated. Physio-therapy, including massage, is useful in certain cases. Strychnine, citrate of caffeine, nikethamide (coramine), ephedrine, and pituitary (whole gland) may be tried. For Addison's disease, see page 507. Digitalis is of no avail. The wearing of an abdominal belt is very helpful in some cases.

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SECTION XIV

VASOMOTOR NEUROSES (ANGIO-NEUROSES)

INTRODUCTION

UNDER this heading are described several diseases in which vasomotor disturbance is the prominent symptom. Sensory, secretory and trophic disturbances may also be present. These diseases differ from each other both in regard to the nature and location of the vasomotor changes. Thus in Raynaud's disease there is a spasm of peripheral arteries. In erythromelalgia there is vaso-constrictor paralysis or excitation of the vaso-dilator nerves. In angio-neurotic œdema there is disturbance of capillary permeability, and perhaps of capillary tone. These diseases are described as vasomotor neuroses because a lesion of structure is not an essential part of their pathology, and because a considerable functional element is generally present. Thus they are common in persons who have an unstable nervous temperament, and emotional disturbance and fatigue play a not unimportant part in their ætiology. It has been thought in the past that these diseases were primarily due to disorder of the involuntary or vegetative nervous system. Lewis, however, has shown in the case of Raynaud's disease that a local fault of the vessels rather than a disordered vasomotor impulse determines the spasm of the digital vessels, and he finds the explanation of Raynaud's disease in terms of vasomotor dysfunction unconvincing. Too little is known of the ætiology of acroparæsthesia, erythromelalgia and Milroy's disease to throw light on this aspect but whatever the basic pathogeny of these conditions may be, vaso-dilatation is a prominent feature of erythromelalgia.

It is important that as far as possible a distinction should be drawn between these diseases occurring as neuroses and similar syndromes complicating recognised pathological states, such as lesions of the spinal cord or brain (tabes dorsalis or hemiplegia), lesions of peripheral nerves (peripheral neuritis), and lesions of vascular channels, or local pressure effects, such as may result from a cervical rib. But they are not separated by a rigid line from slighter manifestations of vasomotor instability, such as are frequent in women at the climacteric, and in clinical disorders resembling exophthalmic goitre. They are undoubtedly akin to such common symptoms as flushings, cerebral hyperæmia, facial congestion, angio-spasm in all its varieties, tachycardia (in some of its forms), anginal attacks, migraine, vertigo, tinnitus aurium, universal or circumscribed hyperidrosis, and gastric disorders of certain forms of functional gastric dyspepsia.

Lewis's studies on the local vascular reaction to irritation of the human skin have thrown much light on these diseases. He showed that there are three components in the reaction: (1) a primary dilatation of capillaries—the *red line*; (2) an increased permeability of these capillaries, producing the *wheel*,

which is independent of the nerve supply ; and (3) the *flare*, which depends on the integrity of the sensory nerve fibres in the neighbourhood. All these phenomena can be produced by an intradermal injection of histamine, and he attributed them to the liberation of this or some similar chemical substance to which he gave the name of "H-substance." It has been suggested that local liberation of histamine may play a part in producing the vesicles in herpes and the rash in erythema nodosum. It is of special interest that these reactions are partially dependent upon and partially independent of the nervous system. Some of the angio-neuroses are therefore so closely related to allergy that a general description of that condition is called for here.

ALLERGY

Idiosyncrasy has been defined as an unusual physiological personal equation, and allergy is a chemical idiosyncrasy, which expresses itself as an urgent attempt on the part of the cell to conserve its chemical identity. The tendency to allergy is inborn, whereas anaphylaxis is an acquired sensitivity. All the phenomena of allergy can be reproduced by histamine. The question is, how does this substance, which is toxic to everyone, become liberated in the tissues of certain people in answer to stimuli which are quite harmless to everyone else ? Normally there are two antagonists to histamine, adrenalin and the special ferment histaminase. It has therefore been suggested that the chemical basis of allergy is a congenital lack of histaminase, aggravated by an intermittent deficiency in adrenalin. The reaction is usually excited by foreign proteins, but a similar idiosyncrasy may be shown to various drugs.

The manifestations of allergy express themselves chiefly (a) in the *respiratory system*, by asthma, hay fever, paroxysmal rhinorrhœa, recurrent catarrhs ; (b) in the *skin*, as urticaria, purpura and eczema, particularly of the infantile type ; (c) in the *alimentary canal*, by diarrhœa and vomiting, or spastic colon ; and (d) by *effusion into joints*. There are many other conditions which with more or less show of reason might be added to this list. Certainly some cases of migraine seem to be of this order. It will be noted that several of these manifestations could be interpreted as a violent attempt to expel the invader, at any rate from the vital organs.

Most allergic manifestations are worse at night, because of the prominence of vagus control then, so that there is a relative insufficiency of the sympathetic nervous system, and therefore in the supply of adrenalin. The principal conditions produced by allergy are described under their appropriate headings.

ANGIO-NEUROTIC OEDEMA

Synonym.—Quincke's Disease.

Definition.—A paroxysmal affection, characterised by the appearance of circumscribed oedematous swellings of the skin and subcutaneous tissues of transient duration. The mucous membranes are often affected.

Ætiology.—Heredity is an important factor. Osler reported the case of a family in which five generations had been affected, involving 22

members. The condition is more common in men than in women, and generally affects the young. Those attacked are usually of a nervous disposition. Garrod reported a case in which each recurrence of periodic hydrarthrosis was attended by circumscribed œdema, either of the lips or eyelids. The attacks may coincide with menstruation. The exciting cause is generally difficult to determine. It may be emotional strain, exposure to cold, or trauma. Local trauma sometimes determines not only the onset but the site of an attack, as in a case recently reported in which riding provoked an attack on the inner aspect of the thighs and knees.

Pathology.—In the absence of a known pathology various theories have been advanced to explain the condition. Local venous spasm, a direct nervous influence on capillary walls, as a result of which the permeability of the vessels is increased, and, more recently, the local action of a circulating toxin on the capillary walls, are theories which have obtained support. With regard to the last named, Garrod drew attention to the joint swellings that frequently accompany erythematous and urticarial rashes resulting from known toxic causes. Such conditions form a part of the clinical picture of serum sickness, or may occur after taking certain articles of diet, or as the result of stings of insects or nettles. Lewis has shown that a modification of the same toxin may produce a dermolysin or a hæmolysin. In the former instance œdema; in the latter purpura results. Thus, *B. welchii* may produce either condition, depending on the intensity of the infection. This thesis illustrates the present view of angio-neurotic œdema as being a local expression of the presence of a circulating toxin, prone to occur in persons of nervous temperament, rather than a disease *sui generis*. The patients often show other signs of allergy, especially in their sensitiveness to foreign proteins.

Symptoms.—The complaint takes the form of acute circumscribed swellings of the skin and subcutaneous tissues, 1 to 4 inches in diameter. The swellings are rounded, painless, rarely itch, and are generally pale or sometimes redder than the surrounding skin, from which they stand out prominently. They may develop simultaneously in different parts of the body, and disappear in a short time. They may recur repeatedly, or only after a period of years; the recurrence is occasionally periodic. They occur most commonly in the eyelids, lips, cheeks and backs of the hands, and are asymmetrical. The whole side of the face, one side of the scrotum, the penis, a whole limb, or in fact any part of the skin, may be involved. The pharynx, tongue and conjunctivæ may be implicated. Œdema of the glottis is rare, and has proved fatal. Swelling of mucous membranes may lead to symptoms of gastro-intestinal disturbance, such as nausea, vomiting and colic. Cases in which hæmorrhage from mucous membranes, stomach, bronchi, bladder, etc., occurred have been reported. Hæmoglobinuria has been observed; in such a case a Wassermann test is indicated. The attacks are generally afebrile, and there is no constitutional disturbance, unless the stomach or intestine is involved.

Course.—This is variable. Recurrence is frequent, often at intervals of 3 to 4 weeks, but sometimes after long intervals. It is rarely periodic.

Diagnosis.—The complaint is so characteristic, in the sudden onset and rapid subsidence of asymmetrical rounded swellings, that it is hardly likely to be confused with other affections. The condition is nearly allied to

urticaria, from which it is distinguished by the circumscribed and deep-seated nature of the swellings and the absence of itching. No distinction is made between angio-neurotic œdema and giant urticaria.

Treatment.—The general health must receive first attention, and a saline purge is indicated. Both arsenic and quinine have been advocated. It is advisable to avoid any particular protein in the food which is found to excite attacks. When the attacks occur after a particular meal of the day, a capsule containing 1 to 2 grains of peptone half an hour before that meal appears to have an effect in temporarily desensitising the body against foreign protein. This, combined with 5 to 7½ minims of tincture of belladonna and 10 to 15 grains of calcium lactate after meals, has prevented recurrences in several cases. The former drug diminishes the vagal hypersensitiveness, and the latter increases the viscosity of the blood. One of the most useful forms of treatment for the relief of the paroxysm is a subcutaneous injection of 3 to 7 minims of liquor adrenalinæ hydrochloridi. This excites the antagonistic action of the sympathetic. For the same reason half a grain of ephedrine orally administered may be tried. When the tongue is involved the patient should be given one or two of Armour's suprarenal tablets to suck. Pituitary (posterior lobe) extract injections have also been recommended. Bromide is often helpful as an additional measure when the symptoms are marked. In severe cases the intravenous injection of small doses of peptone might be considered. Autohæmotherapy has proved decidedly useful in some cases. Vitamin K is also worthy of a trial.

Intermittent Hydrarthrosis, which is described under "Diseases of the Joints" (p. 1367), presents some interesting affinities with the vasomotor neuroses, particularly in its association with angio-neurotic œdema.

RAYNAUD'S DISEASE

Definition.—"Intermittent pallor or cyanosis of the extremities brought on by cold, with the skin a normal colour between attacks" constitutes Raynaud's phenomenon, which may occur however in conditions other than Raynaud's disease.

Ætiology.—The cause of this malady is unknown; it almost exclusively affects young women, and symptoms may begin any time between adolescence and middle age. The diagnosis of Raynaud's disease in a man is nearly always wrong. Jonathan Hutchinson preferred to speak of Raynaud's phenomenon, regarding it not as an entity but as a syndrome occurring in many different conditions. As he first suggested, a few cases are due to syphilis, congenital or acquired. It has occasionally been observed after acute infections.

Pathology.—Lewis has shown that there is an abnormality of the digital arteries, which expresses itself in a hypersensitiveness of these vessels to relatively low temperatures. It seems therefore that the fault lies primarily in the vessel wall rather than in the nerve supply to the muscle fibres. In advanced cases there is endarteritis, with partial occlusion of the lumen of the artery.

Symptoms.—The patients complain of attacks of pallor, blueness or numbness of the fingers, brought on by contact of the hands with anything

cold. Keeping the palms of the hands in cold water (15° C.) for 15 minutes in a cool room (18° C.) is usually sufficient to induce an attack. The body temperature is just as important, if not more so, than that of the hands in determining the onset of cyanosis. When such patients feel chilly in themselves their hands go blue on the slightest provocation, *e.g.* when sitting in a draught; when walking, cycling or driving a car in cold weather; or when swimming. When the body is really warm it is impossible to induce an attack no matter how cold the hands are. Emotion also may bring on an attack. In this connection it is interesting that an injection of adrenaline may have a similar effect. The attacks vary from slight pallor of one finger-tip to cyanosis and numbness of all the fingers of both hands. The cyanosis begins as a light-bluish tint, and later becomes a deeper blue. It always starts at the finger-tips, and spreads proximally to the base of the fingers and perhaps to the palm; rarely, if ever, does it reach above the wrist. If the attack persists for long, a secondary waxy pallor replaces the cyanosis. The hands remain blue or pale until they are warmed. They feel cold to the observer's touch. When the hands are warmed (40° C. for 3 minutes), or when the body temperature rises, the blueness begins to pass off, and irregular red blotches appear in its midst "like the spots on a plaiice." Some of these appear and fade away, but in the end they coalesce until the dorsum of the hand and the palm are fiery-red or scarlet. Gradually this redness spreads up each finger from base to tip.

Throughout the period of cyanosis the patients complain of a "tingling" or of a "feeling of numbness" in the fingers; some of an "uncomfortable sensation"; some of a "slight pain"; severe pain is unusual. When warmth is applied in any form—hot air in front of a fire, or by friction—the fingers quickly recover their normal colour. During this stage there are paræsthesiæ, "pins and needles," etc. In only a few patients is sweating of the hands a marked feature during attacks. Swelling of the fingers is rare. In a severe attack local pressure on a finger leaves an indentation which takes longer to disappear than when the circulation is normal. While the hands remain cold the radial pulses are of smaller volume and the veins less prominent than when warm. If a finger is accidentally cut when cold it does not bleed; "only a little dark blood oozes out." If symptoms occur in the feet they are usually less severe than in the hands. Nutritional changes at the extremities are rare in true Raynaud's disease. But in the later stages, when secondary arterial changes in the arterial walls have occurred, small areas of superficial necrosis at the finger-tips may be found, which leave small depressed tender scars.

Diagnosis.—This has been considerably clarified by John Hunt. The first question to be answered is: Is the complaint "Raynaud's phenomenon"? *i.e.* Is there *intermittent* cyanosis of the extremities, brought on by cold, with the skin a normal colour between attacks? The following conditions with their vascular phenomena are eliminated by this simple definition, many of them because their symptoms are continuous and not intermittent: chilblains; frost-bite; acrocyanosis; erythrocyanosis; clubbed fingers and cyanosis of the fingers and toes due to lesions of the heart and lungs; enterogenous cyanosis; incipient gangrene from arterial thrombosis (in advanced arteriosclerosis, thrombo-angitis obliterans, or ergotism); arterial embolism; diseases of the nervous system

(neuritis, poliomyelitis, syringomyelia, pyramidal lesions, and hysterical paralysis), cervical rib. If true Raynaud's phenomenon is present then the other conditions in which this phenomenon occurs must be considered :

(1) *Hereditary cold fingers*.—Many healthy young people find that their fingers go white and numb on exposure to cold. This is the commonest cause of Raynaud's phenomenon, and is local syncope in its simplest form. The onset is usually during childhood up to about the eighth year. Both sexes are affected, as are often members of the same family. These three points clearly differentiate the condition from Raynaud's disease.

(2) *After local injury or infection of the hands or feet, and in workmen using vibrating instruments*—pneumatic chisels, hammers, riveters, road drills, etc.

(3) *Scleroderma*.—This is diagnosed from Raynaud's disease by the following points : The fingers soon become stiff, and the stretched shiny skin cannot be picked up from the underlying tissues. Nutritional changes in the finger-tips are frequent. It is not confined to the extremities, and is much more rapidly progressive, painful and depressing than Raynaud's disease.

(4) *Syphilitic Arteritis*.—This may be diagnosed when Raynaud's phenomenon is associated with severe necrosis of the nose and ears, and when hæmoglobinuria is present. A blood Wassermann may be positive.

(5) *Other rare causes of Raynaud's phenomenon*.—Erythræmia is one.

Treatment.—Cold in any form should be avoided. The temperature of the body as a whole is almost more important than that of the hands and feet, and warm clothing is essential. The temperature of a living-room should be about 20° C. (68° F.). Cold water should be banned, and gardening in cold weather left to others. Gloves should be loose and long, overlapping the coat-sleeves. For people who work with their fingers, mittens are invaluable. A muff, a small hot-water bottle, or an electric heater in a handbag or pocket may be recommended. Cracks at the ends of the fingers may be covered with a collodion preparation, or with narrow strips of elastic adhesive plaster. The skin of the fingers may be kept soft by applying liquid paraffin. Care should be taken to avoid minor injuries to the fingers, and when these occur they should be treated at once. Boots or shoes should be of stout construction, allowing plenty of room for the toes, and stockings or socks should be thick and soft ; two thin socks often keep a foot warmer than one thick one. Tight suspenders should be avoided. Bedsocks and hot-water bottles are helpful at night.

The number of drugs that have been recommended to relieve symptoms indicates how few are really valuable. Thyroid, calcium lactate or gluconate, potassium iodide, and belladonna are perhaps the favourites. In some patients physical therapy is needed to give relief : postural exercises ; radiant heat, taking special care to avoid burns ; contrast baths ; intermittent venous occlusion by alternating positive and negative pressures ; and galvanic baths. Sympathectomy is indicated when the attacks of cyanosis are causing definite distress or recur so frequently as to interfere with work, and when temperature tests suggest that there is a considerable degree of vasomotor tone. The immediate results of the operation are excellent ; but after some months, perhaps two years or more, slight symptoms may return. In spite of this disappointment the great majority

of patients insist, even several years after the operation, that their hands are better than they were before it. The relief of major symptoms is due to removing from the vessels the burden of their normal vasomotor tone. The local fault in the vessels remains untouched, for on this the operation has no effect.

ACROPARÆSTHESIA

Definition.—A vasomotor neurosis, characterised by paræsthesiæ of the hands, especially affecting the finger-tips.

Ætiology.—The condition is usually observed in women, especially at the climacteric. It rarely occurs before the age of 30. It is frequently associated with a neuropathic diathesis and a lowered vitality due to any cause. General causes include inanition, anæmia and pregnancy. Local causes are exposure to cold, particularly cold water, or to alternate hot and cold water as experienced by washerwomen.

Symptoms.—The onset is insidious and the symptoms are almost entirely subjective. The affection is often limited to one hand or certain fingers, the toes rarely being affected. The patient complains of numbness, tingling, formication of the fingers or tenderness of the finger-tips. There may be slight loss of sensibility in the finger-tips and occasionally evident pallor.

Diagnosis.—The condition is readily distinguished from Raynaud's disease by the absence of local asphyxia. It is important to exclude any affections of the spinal cord, such as tabes dorsalis.

Prognosis.—The complaint tends to be continuous and persistent. The outlook regarding recovery is not good, unless the condition is due to a recognisable and removable cause. There are, however, no complications.

Treatment.—This is directed to the removal of the cause, and improvement of the general health and of the local circulation. Sodium salicylate and bromides are often helpful, and radiant heat and massage are of value.

ERYTHROMELALGIA

Definition.—A rare condition characterised by pain, redness and swelling of the toes and feet, and less often of the hands.

Ætiology.—Little is known of the ætiology of the disease. Men are more often affected than women. The condition may occur in the course of a disease of the central nervous system, such as hemiplegia, disease of the cauda equina, and disseminate sclerosis. The swelling and pain are aggravated by standing and by warmth.

Pathology.—Disease of the peripheral arteries—a chronic endarteritis—has been described in three cases by Batty Shaw. Changes in the peripheral nerves have been held responsible, and Weir Mitchell found marked degeneration of the fine nerve branches in one case. Others regard the malady as an angio-neurosis, allied to acroparæsthesia and Raynaud's disease. Lewis prefers to regard erythromelalgia as a special instance of a condition he designates as *erythræmia*, which he maintains has nothing to do with abnormality of the vasomotor system, but always results from a local process.

There is a release into the skin of an unknown substance not histamine, which lowers the threshold of the pain nerve endings.

Symptoms.—The first case was described by Weir Mitchell, and was that of a sailor, aged 40, whose first complaint, following an African fever, was of "dull, heavy pains at first in the left, and soon after in the right foot. There was no swelling at first. When at rest he was comfortable, and the feet were not painful; after walking the feet were swollen. They scarcely pitted on pressure, but were purple with congestion; the veins were everywhere singularly enlarged, and the arteries were throbbing visibly. The whole foot was said to be aching and burning, but above the ankle there was neither swelling, pain . . . nor flushing."

Pain is generally the first symptom, soon followed by redness and swelling, most marked in the terminal phalanges of the toes or fingers. The pain is generally severe; at first it occurs only in the evening, but later it becomes chronic or remittent and may be agonising. The redness may increase to cyanosis. The swelling is more marked in the latter part of the day, and is aggravated by standing, walking, dependence of the limb and by heat. These symptoms are relieved by cold and recumbency. Hyperidrosis of the affected part is not uncommon. The condition may be complicated by general weakness, vertigo, headache, palpitations and tachycardia. Its complication with erythraemia has been described. Pellagra has been mistaken for erythromelalgia.

Prognosis.—The complaint is intractable, and tends to persist, with exacerbations and remissions, for many years.

Treatment.—The affected part should be elevated and immobilised. Faradism and cold have been recommended. Analgesics are required for the relief of pain, which may even necessitate amputation (Shaw). Sympathectomy is contra-indicated and should never be performed for this condition.

ERYTHROCYANOSIS

This condition occurs, as its full name, Erythrocyanosis Crurum Puellarum Frigida, implies, chiefly in the legs of young women. The feet and legs are cold, and the back of the leg and ankle is swollen and blue, especially at the insertion of the tendo Achillis. Chilblains are often present, and ulceration may occur where the swelling is most severe. The condition is due to vascular spasm, in which the modern fashion of inadequately protected extremities is presumably a factor. Treatment includes warmer stockings, with silk ones outside to gratify the usual desire for appearance, and the loosening of all tight bands above and below the knee. Exercises, such as walking and skipping, with local massage should be ordered, and elevation of the heel may give some relief. Calcium gluconate, vitamin D and small doses of thyroid have been advocated, and daily subcutaneous injections of acetyl choline bromide, beginning with $\frac{1}{4}$ c.c. and gradually increased to 1 c.c., have given good results. After a fortnight the injections may be administered less frequently, and at the end of six weeks discontinued. Bilateral lumbar ganglionectomy has been recommended, but should not be considered unless thorough medical treatment fails. It may improve the colour of the skin and promote healing of any ulcers, but it does not diminish the swelling.

CAUSALGIA

This is a form of neuralgia usually following injuries in the neighbourhood of certain nerves, particularly the median and sciatic. It is probably due to irritation of the peri-arterial sympathetic fibres. On account of the burning character of the pain, Stopford has suggested the name *thermalgia*. The malady is fully described under lesions of the median nerve.

MILROY'S DISEASE

Synonym.—Hereditary Œdema.

Definition.—In 1892, Milroy described a persistent œdema of the legs, occurring in the absence of any of the known causes of œdema, affecting members of the same family in successive generations.

Ætiology.—The disease has occurred in six generations of the same family, but the percentage of incidence in the families has varied greatly. It is apt to appear in neurotic families. Both sexes are affected about equally, and the œdema may either appear soon after birth, or its onset may be delayed till puberty or even till adult life. Thirty-five years after his original description, Milroy found that the disease was tending to die out in the family in which he first observed it.

Pathology.—Nothing is known of the pathology of the condition. There is no evidence of venous or lymphatic obstruction.

Symptoms.—Only the legs are affected, and these to a variable extent. Thus the swelling may be limited to the ankles; it usually does not extend beyond the knees, but may reach the thighs in long-standing cases. It never extends above Poupart's ligament. Gradually the affected part becomes hard and brawny. The swelling increases in the standing posture, and, once established, it is permanent. There is no pain or redness, the veins are not enlarged, and the general health is not affected.

In some cases there are acute attacks accompanied by fever and pain. During this phase the condition resembles erythromelalgia.

Diagnosis.—This is made on the familial incidence, and the absence of all other recognised causes of œdema. A group of cases in which there is swelling of the feet, ankles and legs without albuminuria or discoverable organic disease to account for the swelling, has recently been recognised by Osman. The patients the writers have seen have been women in the third decade. The swelling is pale, brawny, and does not pit on pressure. This type may respond to rest in bed and intensive alkali therapy.

Prognosis.—The affection does not tend to shorten life.

Treatment.—The affected parts should always be kept bandaged with crepe, as by this means the swelling can be kept under control, and the patient remains able to lead an active life; but if such measures be not employed the œdema gradually extends. Acute attacks may require opium internally, and evaporating lotions locally.

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SECTION XV

DISEASES OF THE RESPIRATORY SYSTEM

THE PHYSICAL SIGNS IN THE CHEST IN HEALTH AND DISEASE

ACCURATE diagnosis in diseases of the air-passages and lungs depends largely upon careful observation and record of physical signs, especially in their relation to subjective symptoms. Unfortunately, there is no strict uniformity in regard to the nomenclature of physical signs. It is, therefore, desirable to define explicitly the sense in which the various technical terms used in this section are employed. At the outset, it is well to emphasise the importance of a careful and methodical examination in every case. The magistral sequence of inspection, palpation, percussion, auscultation and mensuration has more than the sanction of tradition to commend it. Unless confined to bed, the patient should be examined both in the erect and recumbent positions in all cases of difficulty.

INSPECTION.—The patient being placed in a good light, the configuration of the chest, the range and character of the respiratory movements and the position of the cardiac pulsations should be carefully noted. Most of the terms used in this connection, such as flattening, retraction, recession of intercostal spaces and diminished movement are self-explanatory.

The respiration may be unduly slow (*bradypnoea*), rapid (*tachypnoea*) or distressed (*dyspnoea*). *Dyspnoea* may be inspiratory, expiratory or spasmodic. A peculiar periodic disturbance of the respiratory rhythm is that referred to as *Cheyne-Stokes breathing*. In this condition, the respiratory movements wax and wane in short periods of *dyspnoea*, each followed by an interval of *apnoea* or cessation of respiration lasting up to 30 or 40 seconds. It is due to deficient aeration of the blood and is met with in respiratory, cardiac and renal disease, and also in cerebral lesions and after some poisons.

A variety of grouped breathing is *Biot's breathing*, sometimes seen in tuberculous meningitis. The hyperpnoeic period consists of a few breaths, deep or of increasing depth, followed by *apnoea* without the waning.

Certain abnormal forms of chest configuration are described: The *alar*, *phthinoid* or *ptyergoid* type of chest is long, narrow and flat, with winging of the scapulæ; the subcostal angle is narrow and the upper interspaces are wide. The *emphysematous* or barrel-shaped chest is broad and rounded, the angle of Louis is prominent, the subcostal angle is wide, and the movements are restricted. The *pigeon breast* is characterised by prominence of the sternum, with sloping anterior thoracic walls. The *funnel breast* is

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the converse of this, with depression of the lower end of the sternum and of the cartilages attached to it.

PALPATION.—Vocal fremitus or tactile fremitus is the vibration felt over the lung when the hand is placed flat upon the chest-wall without pressure and the patient says “*ninety-nine*” or some other resonant syllables. Vocal fremitus may be increased, decreased or absent in disease.

Tussive fremitus.—The similar vibration felt during cough.

Rhonchal fremitus.—The vibrations communicated to the chest-wall by sonorous rhonchi in bronchitis, and felt by the hand.

Friction fremitus or pleural fremitus.—A rubbing sensation communicated to the palpating hand in certain cases of dry pleurisy. A similar fremitus is occasionally felt in pericarditis.

PERCUSSION.—This consists in tapping the chest-wall over the lung and observing the note produced and the sense of resistance felt. Percussion may be *direct* on to the chest-wall or *mediate*, when the tap is made on to a finger or an instrument placed on the chest. The applied finger or instrument is called the pleximeter, the tapping finger or instrument, the plessor. Percussion should always be light, except over very muscular parts of the chest.

Normal resonance is the note obtained over healthy lung tissue.

Hyper-resonance is an increased resonance, with diminished sense of resistance obtained over emphysematous lung tissue.

Dullness is diminution or loss of resonance, with increase in the sense of resistance. Various degrees of dullness are described, such as impaired percussion, slight dullness, flat, wooden or stony note.

Tympanitic resonance—a hollow drum-like note.

Skodaic resonance—a clear, high-pitched note intermediate in character between the hyper-resonant and tympanitic notes.

Cracked-pot sound—or *bruit de pot fêlé*—a hollow note with a slight jingle added to it, obtained by smart percussion over a fair-sized cavity. It is also heard on percussion of a crying baby.

AUSCULTATION.—The breath sounds should be listened to first, then the adventitious or added sounds, and lastly the vocal resonance.

(a) **BREATH SOUNDS.**—The following varieties of breath sounds may be differentiated :

Vesicular breathing.—The normal respiratory murmur or faint rustling sound audible during inspiration and expiration, the former phase being two or three times as long as the latter. The pause between inspiration and expiration is short.

Cog-wheel, jerky or interrupted breathing is a form of vesicular breathing in which inspiration waxes and wanes, or is divided into two or more parts.

Harsh, exaggerated or puerile breathing.—An intense form of vesicular breathing heard in children and in some forms of emphysema.

Vesicular breathing with prolonged expiration.—There is no alteration in the intensity or pitch of inspiration, but expiration is more prolonged and often harsher.

Absent, diminished, weak and suppressed breathing are self-explanatory.

Bronchial breathing.—The pitch of both inspiration and expiration is raised. Expiration is as long as inspiration and is separated from it by a distinct pause.

Broncho-vesicular and vesiculo-bronchial breathing are incomplete forms of bronchial breathing in which inspiration or expiration respectively assume the bronchial type.

Tubular breathing is a peculiar form of high-pitched bronchial breathing of whiffing character, sounding as if produced close under the stethoscope. This term is often used as if synonymous with bronchial breathing, but should be restricted to breathing of the type just described, which is only heard in consolidation from lobar pneumonia and broncho-pneumonia and in collapse of the lung.

Cavernous breathing is bronchial in type, but both inspiration and expiration have a peculiar hollow character. Expiration is more hollow and more prolonged than inspiration.

Broncho-cavernous breathing is incomplete cavernous breathing, inspiration being bronchial, while expiration is cavernous.

Amphoric breathing.—An intense form of cavernous breathing, often having a very hollow metallic sound.

(b) **ADVENTITIOUS SOUNDS.**—These were formerly divided into dry and moist. The former are now called rhonchi, the latter râles.

Rhonchi are musical sounds produced by the passage of air over mucus or muco-pus in the bronchi. Those arising in the larger tubes are called sonorous rhonchi, those in the smaller tubes sibilant or whistling rhonchi.

Râles are bubbling or crackling sounds produced in the bronchi or alveoli by the passage of air through fluid exudate or secretion. They are usually divided into bubbling and crackling râles. Bubbling râles are heard when the lung tissue is still spongy. Crackling or crepitant râles are produced in consolidated or softening areas of lung. Both varieties are arbitrarily subdivided into fine, medium and coarse râles. Crepitant râles are sometimes referred to as "creps"; this practice may lead to confusion with crepitation and is better avoided. Crepitant râles were formerly called consonating, bubbling râles non-consonating. Gurgling râles are coarse, low-pitched râles, usually heard over a cavity, especially after a cough.

Crepitations are fine "hair-like" crackling sounds. They may be produced either in the pleura or in the lung. In the latter they occur only in pneumonia, broncho-pneumonia, collapse and œdema. They are heard chiefly with inspiration and may be increased in number and intensity by coughing. A coarse variety heard in resolving pneumonic lung is called *redux crepitation*.

Pleural crepitations are fine sounds of similar character occurring in the early or dry stage of pleurisy. They are heard rather towards the end of inspiration and are usually unaffected by cough.

Friction is a coarse rubbing, creaking or grating sound heard in pleurisy when there is rough exudate on the pleural surfaces. It may occur with either inspiration or expiration or with both.

Stridor is a loud, coarse sound, heard chiefly during inspiration in cases of obstruction of the larynx, trachea or main bronchi. It is louder and lower pitched than a rhonchus.

Post-tussive suction is a hissing sound, audible directly after cough. It is heard only over a cavity, and is caused by the influx of air to replace that expelled by cough.

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Veiled puff of Skoda is a puffing sound heard towards the end of inspiration. It is said to indicate small and sacculated bronchiectatic dilatations.

Metallic tinkling and amphoric echo are terms used to describe the quality of certain sounds produced near a very large cavity or a pneumothorax, in breathing or coughing, or by the heart's action.

Succussion splash is a splashing sound produced in a hydro- or pyopneumothorax by shaking the patient, or getting him to shake his thorax. If a gastric splash can be excluded, it is pathognomonic of a pleural or subphrenic hydro- or pyopneumo-thorax.

Bell sound or *bruit d'airain*.—A ringing sound heard on auscultation over a pneumothorax or any large cavity when a coin placed flat on the chest-wall over the air-containing space is tapped by a second coin. A similar sound is often audible on flicking with the finger and thumb over the chest-wall under similar conditions.

VOICE SOUNDS or Voice Conduction.

Vocal resonance is the muffled sound on listening over normal lung when the patient articulates " ninety-nine " or some other resonant syllables.

Bronchophony is an increase in the intensity of the normal vocal resonance.

Pectoriloquy is conduction of the articulate voice sounds which are clearly heard as if spoken into the stethoscope. It is best appreciated by auscultating the whispered voice, and is then called whispering pectoriloquy.

Ægophony denotes a peculiar bleating or nasal modification of the voice sounds, sometimes heard on listening to them through fluid in the pleural cavity.

Physical examination of the chest includes mensuration, estimation of vital capacity and examination by the X-Rays when these are necessary.

Vital capacity is determined by a spirometer, which measures the amount of air which can be expired by a full expiration after the deepest possible inspiration. The average for an adult man is about 3600 c.c. The vital capacity is diminished in many diseases of the respiratory system, notably in acute pneumonia, pulmonary tuberculosis and in attacks of asthma.

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DISEASES OF THE NOSE

ACUTE CATARRHAL RHINITIS

See The Common Cold, p. 133

CHRONIC CATARRHAL RHINITIS

Ætiology.—Simple chronic rhinitis appears to result from frequently occurring attacks of coryza, or even from the persistence of a single acute attack. Predisposing causes of this persistence are deficient resistance, local irritation, auto-intoxication from the gastro-intestinal tract, and reflex vasomotor disturbance. Thus we find chronic catarrh associated with

anæmia, stuffy and over-heated rooms, occupations involving inhalation of irritating dust or vapour, excessive smoking, snuff-taking (a commoner habit than is generally realised among shop-assistants and clerks who are prevented from smoking during working hours), dyspepsia and constipation, alcoholism, sexual excess and masturbation. Nasal obstruction is an important factor in keeping up chronic catarrh by preventing ventilation of the passages, allowing mucus to collect and encouraging the growth of micro-organisms. In children the presence of adenoids is the commonest cause of catarrh.

Symptoms.—The symptoms are nasal obstruction and excessive secretion, which may be watery or muco-purulent, and may come forwards to the nostrils or pass backwards into the throat ("post-nasal catarrh"). Secondary results, from the extension of the inflammation, include catarrhal and suppurative otitis media, pharyngitis, laryngitis and bronchitis.

The nasal mucosa may be reddened, but often has a pale, sodden appearance. The turbinals are swollen and are at first quite soft, but later, when definite thickening has occurred, they feel firmer, and no longer shrink after the application of cocaine or adrenaline. At this stage the condition may be called "hypertrophic rhinitis," and the mucosal thickening, most marked over the two ends of the inferior turbinals and over the lower margin of the middle turbinal, may form large lobulated masses.

Diagnosis.—The diagnosis, in cases of hypersecretion, can only be made after excluding by rhinoscopic examination all other causes, such as a foreign body, mucous polypus, syphilitic, tuberculous and lupoid ulceration, and, more especially, suppuration in any of the accessory sinuses; in the latter, the discharge is not scattered over the nasal passages, but emerges in a localised stream from one or other of the ostia and reappears in the same situation after being removed. The discharge of chronic rhinitis is bilateral, and, though often muco-purulent, is never true pus. The diagnosis from vasomotor rhinorrhœa is often difficult; the symptoms of the latter come and go with great suddenness, often as a result of definite causes such as going into a hot room, and there is complete absence of symptoms between the attacks. A bacteriological examination is often helpful.

Treatment.—General treatment, directed to the predisposing causes mentioned above, is necessary if a good result is to be obtained. Next, nasal obstruction must be removed, and operative treatment is called for if the cause be adenoids, deviation of the septum, or great hypertrophy of the extremities of the inferior turbinal bodies. For details, the reader is referred to surgical works; but it must be emphasised that the valuable secreting surface of the nasal mucosa must not be recklessly sacrificed, and that large portions of inferior turbinal must on no account be removed, nor should every slight deviation of the septum be submitted to operation, for it is rarely quite straight. When the turbinal enlargement is soft and shrinks after the applications of cocaine, the galvano-cautery should be used under local anæsthesia to draw one or two lines along the length of the inferior turbinal, the result of which is to produce a scar binding the mucosa to the bone. When the case has not gone on to definite hypertrophy, the most valuable form of local treatment is cleansing of the nasal passages. The lotion must be warm, about 90° F., and quite unirritating; for this reason it should have approximately the same specific gravity as serum, and

normal saline solution does very well. A mildly antiseptic and alkaline lotion is usually preferred, of which Dobell's solution is the type, such as sodium bicarbonate, grs. 3; sodium chloride, grs. 3; phenol, gr. 1; glycerin, min. 45; water, fl. oz. 1. A convenient instrument for the purpose is a small rubber ball of a capacity of 2 oz. with a blunt nozzle moulded in one piece, which is slowly emptied into the nostril, while the patient breathes deeply through the mouth and inclines the head forward over a basin; in this way the palate is raised and the lotion passes through the naso-pharynx and out by the other nostril. The nose must not be violently blown afterwards, nor must any force be used during syringing, or fluid may be injected into the Eustachian tubes. Occasionally syringing causes headache, in which case the lotion may be used in a coarse spray-producer. Chronic nasal catarrh is, however, in certain cases notoriously resistant to treatment, especially under the conditions of civilised town life; indeed, many sufferers found themselves better in the wet and exposed conditions of life in the trenches during the War of 1914-1918. Vaccine therapy is uncertain in its effect, but gives good results in a proportion of cases; an autogenous vaccine should be prepared from the patient's nasal secretion.

ATROPHIC RHINITIS

Synonym.—Ozoena.

Ætiology.—Cases usually first come under treatment between the ages of 15 and 18, but its insidious beginnings date from an earlier age, and a history of nasal discharge through childhood is often obtainable. It affects females at least three times as often as males. A peculiar physiognomy is to be noticed in nearly half the cases; the skull is brachycephalic, the nose wide and flat, and the nostrils broad and so directed forwards as to be more than usually conspicuous. The affection is sometimes unilateral, in which case the septum is deflected and the disease occurs on the wider side. Occasionally it is found among several members of a family, which might be the result of contagion, but it is also inherited in circumstances where contagion cannot apply, and this may be due to inheritance of the disease or merely of the predisposing physiognomy. It is more often seen in the poorer than in the more well-to-do classes and, in England, it has become much rarer during the last twenty or thirty years. Of the many bacteria found in association with the affection, the most important are the Klebs-Loeffler bacillus, the *Cocco-bacillus fatidus* of Perez, and the *Cocco-bacillus mucosus* of Beeritz, but the consensus of opinion is that they are secondary, and, though helping to produce the fœtor, are not the primary cause of the disease. The condition occurs at too early an age to be the final stage of hypertrophic rhinitis, nor is it usually due to accessory-sinus disease, which can be excluded in the majority of cases. It is probably the sequel of prolonged purulent rhinitis in childhood, which results in the replacement of the ciliated by squamous epithelium, and thus destroys the principal agent for the removal of secretion; the undue width of the nasal passages promotes this retention by diminishing the force of the expulsive current of air, by drying the secretions and by unduly admitting dust and micro-organisms. In this way crusts of dried mucus are formed and decompose, and the resulting inflammation prevents the

development of the turbinals and thus further increases the width of the nasal fossæ.

Pathology.—There is a chronic inflammation resulting in sclerosis and atrophy of the mucosa; the ciliated epithelium is replaced by squamous, the mucous glands are degenerated and the venous sinuses have disappeared. These changes are most marked over the inferior turbinals; the middle turbinals are frequently large and cedematous. The discharge is not pus, but mucus precipitated by evaporation, mixed with shed epithelial cells and teeming with micro-organisms. This collects and dries into large greenish-black crusts which give rise to the peculiar sweetish and horribly offensive odour. There is never true ulceration nor necrosis of bone.

Symptoms.—The symptoms are chiefly those of the nasal discharge and the offensive stench; the latter is rarely perceptible to the patient, who usually, in established cases, has complete anosmia. There are also obstruction from the crusts, dryness of the throat and cough, and often some degree of ill-health from toxic absorption.

The inside of the nose is full of crusts; the inferior turbinals are reduced to mere ridges, the mucosa is pale and thin, and through the widened nasal passages the body of the sphenoid and the wall of the pharynx are plainly visible.

Complications.—Infection of an accessory sinus may result by extension from the septic nasal cavities, but is not very common; conversely suppuration of the sphenoidal or a posterior ethmoidal cell is a possible, and has been considered by some authorities as the usual, cause of the affection. The lymphoid tissue of the throat is conspicuously absent; there is often a dry pharyngitis and laryngitis, and sometimes the crusting extends to these parts, or even down the trachea. Catarrhal and suppurative otitis are common, and the disease is thought by many to predispose to pulmonary phthisis.

Prognosis.—As the ciliated epithelium can never be replaced, the affection is not truly curable, though regular treatment can keep it in an inoffensive condition, and it is common to see a suggestive degree of atrophy of the turbinals in young people with rhinitis completely disappear under treatment. Also, the crusting tends to become less troublesome as time goes on and ultimately to cease, a state of things which is difficult of explanation.

Treatment.—The nose must be kept clean by regular syringing with a mild alkaline antiseptic lotion, of which a large quantity should be used with a Higginson syringe provided with a fine nozzle which cannot block the nares. An oily, stimulating or emollient application, such as *oleum eucalypti* 15 minims to 1 ounce of *paraffinum liquidum* may be applied as a paint or spray, or a 25 per cent. solution of dextrose in glycerin as a paint. The crusting can be prevented by excluding the air and, when syringing is insufficient, this should be done by introducing a plug of gauze or cotton wool loosely into the anterior nares, which should be changed twice a day by the patient. After some weeks of treatment the packing may be omitted, but resumed in the event of a relapse. When the surgeon removes the plug the discharge is seen to be a clear mucus; if pus be found, it must be traced to its source in an accessory sinus. Paraffin wax may be injected under the mucosa, to narrow the nasal passages, but it is liable to slough out, and a piece of costal cartilage has been implanted with the same object. A plastic operation

has been devised to shift the antro-nasal wall inwards and has given encouraging results. The treatment of anæmia is important; good food and an open-air life, especially at the seaside, are beneficial.

EPISTAXIS

Ætiology.—The causes of epistaxis may be classified as follows:

Local causes.—Traumatism, including blows on the nose, fracture of the base of the skull, surgical operations and foreign bodies; the small septal erosion of rhinitis sicca, which is the commonest of all causes; malignant disease; angio-fibroma, or “bleeding polypus,” of the septum; multiple telangiectasis, a curious hereditary affection characterised by numerous minute dilations of the capillaries on the face and mucous membranes of the nose, mouth and throat; the general congestion caused by adenoids; and syphilis, lupus and the rarer granulomata, though in these the bleeding is usually an insignificant symptom.

General causes.—High blood pressure, as in arterial disease, chronic nephritis, cirrhosis of the liver, violent exertion, extremes of heat and cold, congestion at the menstrual period or “vicarious menstruation”; venous congestion, as in mitral stenosis, tumours in the thorax or root of the neck, emphysema, bronchitis and whooping-cough; toxic blood conditions, as pernicious anæmia, leukæmia, purpura, scurvy, and all the acute infectious fevers, especially in the prodromal stage. To these may be added rarefaction of the air, as in aeroplane ascents and mountaineering, and poisoning by some drugs, especially salicylates and quinine.

The source of the bleeding is, in the large majority of cases, in a region called Little’s or Kiesselbach’s area, situated on the front and lower part of the septum just beyond the vestibule.

Treatment.—Epistaxis, of sufficient severity to call for the attention of the doctor, should always be treated, though it is of comparatively little importance in healthy young people; in older patients with high blood pressure the loss of blood may be beneficial, but the occurrence is so distressing and alarming to the patient that other means to lower the pressure should be adopted.

The source of the bleeding is usually so far forward that a pledget of wool introduced for less than an inch into the naris, and held by compressing the nostril, will generally control it temporarily. To arrest it and prevent recurrence the bleeding spot must be found, started if necessary with a probe, controlled by application of cocaine and adrenaline on a plug of wool, and sealed by the galvano-cautery at dull-red heat. The use of an emollient ointment during the separation of the scab is advisable. In obstinate cases the bleeding may recur from another spot or from the opposite naris, when the treatment must be repeated. As in other forms of hæmorrhage, a rapid excited heart’s action, associated with restlessness and fright, is often present, and an injection of morphine is of great value. Calcium lactate is often recommended and may be given in 20-grain doses three times a day for 2 or 3 days; or colloidal calcium may be injected subcutaneously. If the bleeding is from the usual situation, formal plugging of the nose is seldom called for; but sometimes the bleeding proceeds from farther back in the nose, or is so

profuse that its situation cannot at first be determined. In such cases the naris should be evenly packed with ribbon-gauze introduced on forceps under inspection. The older method of plugging the posterior nares is seldom required, and carries the risk of causing otitis media. Nasal plugs quickly become septic, and should ordinarily be removed in 24 hours; but they may be kept sweet for several days, should it be necessary to retain them, by moistening them frequently with peroxide of hydrogen.

MUCOUS POLYPUS

Ætiology and Pathology.—Nasal polypi are rare before puberty and are somewhat commoner in men than in women. They never grow from the septum, inferior meatus or inferior turbinal, but only from the ethmoidal region and interior of the accessory sinuses. They are not neoplasms, but are essentially due to a local œdema of the mucous membrane; the swelling thus produced is acted upon by the expulsive forces of the nose, and, being so pulled down and elongated, the return flow of its blood vessels and lymphatics is further impeded and a greater degree of œdema results. All stages of polypus formation may be found, ranging from an œdematous fringe along the border of the middle turbinal to enormous pedunculated masses which block the nose and expand its bony walls. In the majority of cases the œdema is due to inflammatory infiltration of the muco-periosteum of the ethmoidal labyrinth and is often associated with inflammation in the ethmoidal cells; sometimes, however, the cause is vasomotor disturbance, for polypi are found in cases of hay fever and paroxysmal rhinorrhœa in the absence of true inflammation. There is also another form, the so-called "choanal polypus," in which a large single polypus hangs into the nasopharynx from a long pedicle attached within the antrum and passing through the ostium into the nose.

Symptoms.—The cardinal symptoms are nasal obstruction and discharge, which is profuse and watery. The symptoms are worse in damp weather. Cough, headache and asthma are not infrequent, and a loss of the power of mental concentration often occurs.

Polypi are smooth, shiny, white, translucent bodies, pedunculated and extremely soft and movable to the probe; their appearance is so characteristic that they cannot properly be mistaken for anything else. If they project into the nostril they become pinker and more opaque.

Treatment.—The best method of removal is in most cases with a wire snare, a process which can be rendered quite painless with skilful manipulation and the application of cocaine. Any polypoid mucosa in the neighbourhood should be removed with punch-forceps, but the application of caustics or the cautery only does harm. Recurrence is common, but becomes less rapid if the new polypi are removed at regular intervals before they have grown large. Inflammatory disease in the ethmoidal cells and other sinuses must, of course, receive treatment. In the worst cases, the polypi are so numerous and return so rapidly that the snare cannot deal with them adequately; in such they should be removed with a ring-knife or suitable forceps under general anæsthesia, together with the softened ethmoidal tissue, and any suppurating sinuses be opened at the same time.

PAROXYSMAL OR VASOMOTOR RHINORRHOEA

Synonym.—Allergic Rhinitis.

In this condition fits of sneezing are associated with a profuse watery nasal discharge, irritation of the nasal and conjunctival mucosa, nasal obstruction, and often marked depression and prostration. The discomfort is usually worse in the morning, in overheated rooms, or on going out into the cold. The rapidity with which the attacks come and go is sufficient to distinguish them from an ordinary coryza. The affection usually shows itself in early adolescence and tends to improve with advancing age; it is distinctly hereditary and is often associated, either in the patient or in his relations, with other symptoms of allergy, such as asthma, urticaria or chilblains. Males and females are equally affected, and it is most frequent among the cultivated classes; a mental shock is sometimes the starting-point of the attacks.

Of these cases, *hay fever* is the best known and most marked variety, and is due to specific susceptibility to a proteid substance contained in the pollen of certain grasses; in this country, attacks begin about the end of May and terminate in August. Hay-fever subjects are affected by very minute quantities of this toxin, whereas ordinary people are completely immune. Other individuals are susceptible to the pollen, seedlets or scent of other plants and flowers, and others again to the emanations from horses,* cats, dogs and other animals; and researches show that asthma, urticaria, eczema or rhinorrhœa may be variously produced by many kinds of proteid substances, including common and uncommon articles of diet, such as eggs or lobsters, in certain people who are specifically susceptible to these substances.

Treatment.—The determining factors, which should receive attention, are heightened irritability of the nervous system, occasionally some intranasal abnormality which increases the sensitiveness, and the specific irritant. Nervine tonics, strychnine, arsenic and valerian, are indicated, and attention to the general health. Hay-fever patients are better in a locality as free as possible from pollen; some remain comparatively well at the seaside others only on board ship, while some have to spend the best days of the year in a darkened room. Occasionally great benefit results from the removal of some nasal abnormality, a polypus or a sharp spur impinging on the turbinal, but the result of operative treatment is uncertain. In most cases the nares are normal, and in many of these a light cauterisation of the most sensitive areas is very helpful; the sites usually chosen are on the upper anterior part of the septum, and on the anterior part of the inferior turbinal. Ionisation of the nasal mucosa with sulphate of zinc is also employed. True hay-fever patients may have their susceptibility to pollen lessened by inoculation with dilute extract of pollen; the use of these extracts gives excellent results in a proportion of cases, and, more recently, the attempt has been made to test susceptibility to, and to immunise against, other proteid poisons.

ACCESSORY-SINUS SUPPURATION

Ætiology.—In the large majority of cases infection reaches the accessory sinuses from the nasal cavity, and may result from a simple coryza or from

one of the acute infectious fevers. Influenza is especially liable to produce disease of the sinuses, which may also be caused by measles, scarlet fever, erysipelas, enteric, pneumonia or small-pox. In addition, antral suppuration is caused by infection from the teeth, particularly the second bicuspid and first two molars, whose sockets are in closest proximity to the antral floor. The discharge from one sinus readily enters and infects another, so that disease of several cavities often coexists.

Symptoms and Diagnosis.—If the ostium of a suppurating sinus be occluded the pus is secreted under pressure, and the local symptoms are severe, whereas if the secretion can escape freely there may be no symptoms except discharge. The former class of case has been called "closed" and the latter "open" empyema. The difference between the two is, however, only relative, and many cases are alternating, the severe symptoms being relieved by periodical discharge. As the pressure of the pus in the cavity depends on the rapidity of its secretion, and the degree of occlusion of the ostium by inflammatory swelling, it follows that the closed and open cases correspond generally to acute and chronic suppuration; acute suppuration is usually fairly obvious, but some chronic cases with scanty secretion are only to be detected after very careful examination and may be for long the undiscovered cause of post-nasal catarrh, pharyngitis or chronic toxæmia.

The symptoms, then, are swelling, pain, tenderness and discharge, together with the secondary effects of the suppuration. Swelling is rare; the bony walls are not bulged by an empyema, and this is a point of distinction between it and a tumour or cyst, but occasionally spread of the inflammation causes periostitis, or a fistula in the bone is formed leading to an abscess outside the sinus. Thus, in frontal sinusitis a swelling may appear at the junction of the inner and upper walls of the orbit, displacing the eye downwards and outwards, or an abscess may form here and, after opening, leave a fistula. Similarly, ethmoidal disease may produce a swelling farther back on the inner wall of the orbit, displacing the eyeball outwards. In antral empyema, a little cedema of the cheek, or slight swelling in the canine fossa, may be found, but a swollen cheek is more likely to be due to dental periostitis, while any definite bulging of its bony walls is an indication of a tumour. Pain is often severe in acute cases, and in chronic suppuration there may be considerable neuralgic pain. Pain of an intermittent character, relieved by a sudden gush of discharge from the nose, is highly characteristic of sinus disease, as also is a peculiar periodicity, for it tends to begin regularly at the same time every morning and to get better during the afternoon. The pain may be of a local inflammatory character, or may be of a neuralgic type and referred to various parts. In antral suppuration it is over the cheek, or may be referred to the teeth or frequently to the supra-orbital region. The pain of frontal sinusitis is over the cavity or along the supra-orbital nerve; that of ethmoidal disease is over the nasal bridge, behind the eye or in the temple, and in sphenoidal suppuration, in the middle of the head, behind the eye, on the vertex or in the occipital region. Tenderness can usually be elicited in frontal empyema by percussion over the anterior wall, and especially by pressing upwards against the floor of the cavity; it is less marked in antral disease, in the canine fossa. Discharge into the nose is the most important, and often the only, symptom. A localised stream of pus in the nose, which reappears after removal, is, in the absence of a foreign body, conclusive

evidence of suppuration in an accessory sinus. The differentiation of the affected sinus is made by following the pus to its source with a probe and, in the case of the antrum, by tapping with a trocar and cannula. The antrum, frontal and anterior ethmoidal cells open into the middle meatus, and the posterior ethmoidal and sphenoidal into the superior meatus. Further assistance is afforded by transillumination and skiagraphy. Fœtor, both subjective and objective, is often present, and a serious degree of anæmia and ill-health frequently results.

Complications.—These include pharyngitis, laryngitis, bronchitis, and otitis media; the swallowed pus causes various forms of gastric and intestinal disorder, including appendicitis. Acute septicæmia, and pyæmia are rare, but symptoms of chronic poisoning are common, and include anæmia, arthritis, fibrositis, and even mental aberrations. A very important series of complications results from extension of the inflammation to surrounding parts: orbital abscess or cellulitis, osteo-myelitis of the frontal bone, cerebral abscess, thrombosis of the cavernous sinus, paralysis of the oculo-motor nerves and, from the sphenoidal sinus, papillœdema and optic atrophy.

Treatment.—This, in acute cases, consists in rest in bed, hot fomentations to the affected part, aperients, a light diet, and a few doses of aspirin. Inhalations of mentholised steam at frequent intervals are of value, and may be prepared by adding 10 drops of 25 per cent. solution of menthol in spirit to a pint of steaming water in an inhaler. In recent cases of antral suppuration, the cavity should be tapped with trocar and cannula and washed out with a warm saline lotion; this should be repeated daily or every two or three days, according to the severity of the disease, and will effect a cure in a large proportion of cases in an early stage. Frontal sinusitis has a greater tendency to spontaneous cure; the anterior end of the middle turbinal may be amputated and occasionally a cannula can be passed and the cavity washed out. Cases which fail to recover under such treatment, and those of chronic suppuration, must be submitted to operation.

SYPHILIS

CONGENITAL SYPHILIS

The *early form* appears at any time within 3 months after birth, usually within the first few weeks. The symptoms, frequently called "the snuffles," are those of nasal discharge and obstruction; the former may be thin and ichorous, or purulent and bloodstained, and is often associated with cracks and excoriations about the nostrils, upper lip and angles of the mouth; the obstruction may cause attacks of choking and frequently prevents the baby from taking the breast, and so produces wasting and malnutrition. These symptoms are not pathognomonic of syphilis, but may also be caused by catarrhal and purulent rhinitis, therefore the diagnosis must be established by the concomitant lesions.

The *late form* appears usually about the period of puberty, but may occur at any time after the age of about 5 years. It is characterised by a slow destructive gummatous infiltration and ulceration, and the symptoms are those of nasal catarrh and obstruction, frequently with fœtor and crusting;

this chronic rhinitis destroys the ciliated epithelium, and may thus cause a true atrophic rhinitis which persists after the syphilis has become quiescent or cured. Congenital syphilis is apt to produce a very characteristic "saddle-back" flattening of the bridge of the nose.

ACQUIRED SYPHILIS

Primary chancre is very rarely seen on the ala of the nose, and is accompanied by bubo of the submaxillary and pre-auricular glands, and by much induration and swelling.

Secondary syphilis does not produce noticeable symptoms in the nose; there may be rhinorrhœa and obstruction associated with hyperæmia of the mucosa.

Tertiary syphilis occurs usually in the form of a diffuse gummatous infiltration and ulceration, which may proceed to necrosis of any of the bony or cartilaginous walls of the nose; there is profuse purulent discharge, often bloodstained, which tends to dry into greenish-black crusts, the odour of which is extremely offensive. A localised gumma may occur on the septum, where it forms a smooth round swelling projecting into both nostrils which, by its contraction after healing, produces a steep depression of the bridge just below the nasal bones. Syphilitic ulceration sometimes attacks the external parts of the nose, causing perforation of the ala, or destruction of the columella with a characteristic depression of the nasal tip.

Diagnosis.—This seldom presents much difficulty; the form with crusting and ozæna imitates atrophic rhinitis, but in the latter there is never necrosis or decided ulceration—indeed intranasal necrosis may be considered pathognomonic of syphilis. A septal gumma has an appearance identical with that of a hæmatoma, but without the sudden onset and history of traumatism. Syphilitic perforations nearly always involve the bone, whereas those due to rhinitis sicca or lupus never do. Some cases of diffuse infiltration resemble lupus; but in the latter there is no necrosis or offensive odour, the characteristic nodules are usually to be seen at the edges of the lesion, and other patches of lupus may be found on the skin or in the fauces. The chief difficulty of diagnosis lies between severe syphilitic infiltration and malignant disease, but it can usually be determined by the clinical appearance, especially by the characteristic edge of the syphilitic ulcer, by the examination of an excised portion, by the Wassermann reaction, and by the results of anti-syphilitic treatment.

Treatment.—General treatment must be very prompt and energetic to prevent irremediable deformity, and should ordinarily be begun with the injection of arsphenamine or of one of its congeners. Of local treatment, the lesions should be kept clean by frequent syringing with a saline lotion, to which lysol, sanitas or listerine may be added when the odour is offensive; any necrosed bone must be removed as soon as it is loose.

LUPUS AND TUBERCULOSIS

Ætiology.—With the exception of the rare occurrence of tuberculous ulceration as a terminal infection in advanced phthisis, the lesions produced

in the nose by lupus and by tuberculosis are indistinguishable; it appears that the tubercle bacillus finds in the nasal mucosa a medium unsuitable for its development, its virulence is diminished, and it can only produce the modified lesions known as lupus. It is possible, also, that this modification of the bacillus by sojourn in the nose is the ordinary cause of lupus; at any rate it is frequently primary in the nares, whence it spreads to the fauces and larynx and on to the face and hands. The disease begins most often between the ages of 15 and 30, is twice as common in females as in males, and is usually seen in badly nourished people of the poorer classes.

Symptoms.—The early lesions are found on the antero-inferior part of the septum, the nasal floor and the front end of the inferior turbinal, within reach of the finger-nail, which probably conveys the infection. The characteristic "apple-jelly" nodules are seen, with or without ulceration, the latter with rounded slightly raised margins, and tending to spread in some directions and cicatrize in others. The lesions are covered by small adherent scabs, and perforation of the septal cartilage is common. The alæ often become involved with destruction of the margin or with perforation, and the nostrils may be much narrowed and deformed by scarring, while the lachrymal duct is frequently involved. The progress of the disease is extremely slow and may continue over many years. The subjective symptoms are nasal obstruction with a slight sticky discharge.

Diagnosis.—The nares should be examined in all cases of cutaneous lupus, for, if the disease remain unhealed in the nose, relapses will continually occur. In the majority of cases of nasal lupus the diagnosis is cleared up by the presence of lesions or scars on the face, fauces or larynx. The difficulties of diagnosis are from rhinitis sicca with perforation, and from syphilis. The typical brownish nodules are pathognomonic and can always be found by careful examination when the lesions are progressing; they can be made more conspicuous by blanching the mucosa with adrenaline. The scabbing of rhinitis sicca quickly clears up under simple emollient treatment, while the lesions of syphilis are more rapidly progressive and tend to involve bone.

Treatment.—The affected areas are defined by the application of adrenaline and thoroughly and carefully scraped away with a sharp spoon, a general anæsthetic being employed if the lesions are extensive; small lesions and recurrences are destroyed with the galvano-cautery. Nascent iodine by Pfannenstiel's method may be employed; sodium iodide is given in 7-grain doses six times a day, while the nose is packed with gauze kept moist with peroxide of hydrogen, 10 volume strength, with 5 per cent. of acetic acid added; when a marked reaction has been obtained this solution should be diluted to half its strength, the iodide being continued as before; the treatment may have to be persisted in for several months. Radium is still under trial, and tuberculin has not established its value in this affection.

Of general treatment, arsenic in full doses, fresh air, cod-liver oil and fattening foods are of value. Lupus does not show the same tendency to spontaneous cure in the nose as in the larynx; it is easy to obtain improvement, but complete cure is difficult.

TUMOURS OF NOSE

Papilloma occurs on the skin lining the vestibule and differs in no respect from cutaneous warts elsewhere. On the nasal mucosa it is excessively rare, occurs usually on the septum, has a narrow pedicle, a rough red or greyish surface, and bleeds readily when touched; occasionally the growths are multiple and, when large, are difficult to diagnose from a malignant growth, but they do not erode bone; they tend to recur locally after removal.

Fibroma, similar to the naso-pharyngeal fibromata, occurs, though rarely, as a smooth pink growth attached to the posterior region of the nares. It bleeds readily and spontaneously and demands great caution in removal.

Angio-fibroma, or "bleeding polypus of the septum," is less uncommon. It varies from the size of a pin's head to that of a filbert, is red or purple, smooth or finely lobulated, sessile or pedunculated, and grows from the anterior part of the septum. The prominent symptom is epistaxis, for the tumour bleeds freely and spontaneously. When pedunculated, it may be removed with the snare and the base cauterised; when sessile, it should be stripped off the underlying cartilage with an elevator. Recurrence is common.

Osteoma and *enchondroma* are extremely rare, usually grow from the ethmoidal region and produce obstruction and deformity.

Cysts.—Mucous polypi are occasionally cystic; the so-called "ethmoidal cyst" is an enlarged cell in the anterior part of the middle turbinal pressing on the septum and causing obstruction and headache. True cysts are sometimes seen on the anterior part of the nasal floor, and arise from the roots of incisor teeth. When small, they may be treated by intranasal removal of part of the wall; when large, they should be dissected out from the gingival fold. *Mucocele*s are cystic dilatations of the antrum, frontal sinus, or an ethmoidal cell.

Malignant tumours occur in all varieties: carcinoma, sarcoma, and endothelioma. Though they sometimes appear on the septum or nasal floor, the usual site of origin is the ethmoidal region. They tend to expand the bones of the face, producing a characteristic frog-like deformity, and they frequently invade the antrum and expand its walls, constituting a common variety of tumour of the upper jaw. The facial, palatine and orbital walls of the antrum may each be bulged outwards, the latter with displacement of the eyeball, and egg-shell crackling can sometimes be elicited. Sanious discharge and free spontaneous hæmorrhage are prominent symptoms and important for diagnosis. Surgical excision produces permanent cure in only a small proportion of cases; better results, especially in endothelioma and sarcoma, are obtained by the implantation of radium. According to the situation of the growth, access is gained by opening the antrum through the canine fossa, by lateral rhinotomy, or for the lower part of the nose by an incision in the gingivo-labial fold from the molar teeth of one side to the other.

DISEASES OF THE NASO-PHARYNX

ADENOIDS

Ætiology.—By this term is implied a chronic enlargement of the lymphoid tissue of the naso-pharynx, the “pharyngeal tonsil.” This is normally present in childhood and disappears by the age of 20 or thereabouts, but if chronically enlarged may remain up to any age. The precise stage at which the enlargement becomes pathological can only be determined by the symptoms which it produces; these usually become manifest between the ages of 3 and 8, but occasionally show themselves at or soon after birth. The incidence of adenoids is universal, but they are most common in damp temperate climates, and chronic or recurrent nasal catarrh is the principal factor in the causation; the infectious fevers, particularly measles, scarlet fever, and diphtheria, are also a frequent cause of the hypertrophy.

Pathology.—The adenoid, as it should really be called, or enlarged pharyngeal tonsil, is a mass of lymphoid tissue of definite anatomical shape; it is thickest above and tapers away below, and presents a series of ridges which radiate from below upwards and slightly outwards. In older patients the mass is firmer and more fibrous, and the ridges are often adherent in places, leaving deep clefts and furrows in which secretion can collect and decompose.

Symptoms.—The symptoms of adenoids are many and various, and include those due to nasal obstruction, those caused by infection and by the extension of inflammation, and reflex processes attributable to irritation and lowered vitality. In infants the nasal obstruction interferes with sucking and a serious degree of malnutrition will result unless the baby be carefully spoon-fed. Older children snore at night, breathe heavily in the day, and either bolt their food or eat very slowly owing to the necessity of breathing through the mouth. Owing to lack of oxygen the patients sleep restlessly, wake unrefreshed and often suffer from a peculiar inability to concentrate the attention sometimes called “aproxexia.” Persistent nasal obstruction during the period of growth mechanically produces permanent deformities of the jaws and face which narrow the nasal passages, prevent the mouth from closing naturally and thus perpetuate mouth-breathing. When the mouth is habitually held open, the *alæ nasi* are pulled downwards with the cheeks, and become narrow and slit-like and fall in like valves with each inspiration; this “alar collapse” is an important cause of obstruction in neglected cases of adenoids. The palate is narrow and highly arched; the dental arch is narrow and V-shaped, so that the upper incisors, crowded and prominent, look outwards rather than forwards, and are not covered by the short upper lip; the lower jaw retains its infantile obtuse angle, and the lower incisors lie behind the upper; the chin is receding and, in the worst cases, when the molar teeth come into contact on biting, the incisors cannot meet. Only a proportion of cases of adenoids show these deformities, and there is, indeed, considerable uncertainty as to the importance of adenoids in their ætiology; undue softness of the bones, such as occurs in rickets, is doubtless an additional factor, and also in the causation of the malformations of the chest which result from the obstruction to the entry of air. The long

narrow unexpanded chest with acute costal angle and prominent scapulae is the commonest deformity. Harrison's sulcus, a transverse depression corresponding to the attachment of the diaphragm; pigeon-breast, a prominent sternum with depressed costal cartilages; and funnel-breast, a sharp depression at the lower end of the sternum, are also encountered.

Various infective processes result from the spread of inflammation and, if the naso-pharynx be large, are not necessarily associated with nasal obstruction. The terribly common catarrhal and suppurative affections of the ear in children are, in an overwhelming majority of cases, the result of adenoids. Blepharitis and phlyctenular conjunctivitis are also associated with adenoid vegetations. Feverish attacks, often with tender enlargement of the cervical glands, are caused by infection of the pharyngeal and faucial tonsils, and tuberculous disease of the glands is usually due to passage of the bacilli through these portals; in such cases the tonsils and adenoids may remain unaffected or may themselves show tubercles under the microscope. Chronic or recurrent bronchitis frequently results from the infection spreading to the lower air-passages. The mucus secreted by the adenoids is swallowed in large quantities, and produces derangements of stomach and intestines with failure of growth and general health. Finally, mouth-breathing predisposes to dental caries. The irritation of these vegetations, and their effect on respiration and the general health, account for numerous reflex and nervous disturbances. Among them may be enumerated laryngitis with spasm called "laryngitis stridula," spasm of the glottis without laryngitis or "laryngismus stridulus," stammering, reflex cough, asthma, night terrors and nocturnal enuresis; it should be stated that the latter disorder is by no means always to be cured by removal of the concomitant adenoids, and that in general too much stress must not be laid upon the presence of adenoids as the causative factor in all these reflex disturbances.

Diagnosis.—In the majority of tractable children a view of the naso-pharynx can with patience be obtained with a good light and a very small rhinoscopic mirror, when the upper part of the septum and the concavity above it are seen to be occupied by an irregular convex mass. Where this is impossible a rapid digital examination may be required; this is extremely unpleasant to the little patient, and may be postponed, in those cases where the tonsils are sufficiently large to call for removal, until the child is anaesthetised. Similarly, in very frightened, intractable children, if the symptoms point strongly to adenoids it is wiser to give an anaesthetic for examination, being prepared to remove the vegetations if present. In the mongolian type of idiocy the tongue is large and the mouth persistently open, and in microcephaly the extremely undeveloped naso-pharynx causes nasal obstruction; cases of both these types of maldevelopment are often brought to the doctor in the hope that removal of their adenoids will cure their "backwardness," and care should be taken not to fall into the error of performing a useless operation, though if a well-marked adenoid be present it should be removed under a guarded prognosis. On the other hand, adenoids can be present and produce serious secondary effects without causing nasal obstruction or any appearance of the typical "adenoid facies."

Treatment.—The normal naso-pharyngeal tonsil becomes swollen during a coryza, and such temporary swelling should not be diagnosed as "adenoids," by which term chronic hypertrophy is understood, and does not call for

removal provided that it subsides promptly, does not frequently recur, and is not associated with otitis media or other important complications. In such cases, and when the only symptom is a mild catarrh, the regular use of a simple warm saline lotion with a rubber ball-syringe (see p. 1086) will often effect a cure; in children below the age of 5 or 6 syringing is apt to be difficult and the lotion may be used in a spray, while in infants it is best to drop it into the nostrils from a small pipette like the filler of a fountain-pen. This treatment should be combined with open air—if possible a change to the seaside or a bracing country district—cod-liver oil, iodide of iron, or arsenic. Breathing exercises are of great value in these slight catarrhal cases, but only do harm where there is marked obstruction.

When the enlargement frequently recurs or has gone on to chronic hypertrophy, operative removal is the only treatment, and this is especially called for when any aural symptoms supervene, or when cervical adenitis is present. After operation the general treatment referred to above is valuable, but the nose should on no account be syringed until healing is complete, as this encourages aural complications. If the alae nasi are collapsing, or the chest narrow, breathing exercises are of use, but healthy open-air occupations are more useful still. In patients in their teens, or upwards, turbinal hypertrophy has not infrequently resulted; the surgeon should be prepared to snare off enlarged posterior ends of the inferior turbinates at the time of the operation, and intranasal cauterisation may be required later.

TUMOURS

Innocent tumours in the naso-pharynx are exceedingly rare; the so-called "choanal polypus" is a variety of nasal mucous polypus which hangs into the naso-pharynx from a long pedicle attached within the nares.

Fibroma of the naso-pharynx, or naso-pharyngeal polypus, occurs usually in males between the ages of 10 and 25, grows by a broad pedicle from the periosteum of any part of the walls of the naso-pharynx, usually from the basi-sphenoid, and forms a smooth, rounded, pink mass which fills the naso-pharynx and sends prolongations into the nasal cavities. The palate is pushed downwards, the bones of the face expanded, and the eyeballs separated and displaced, producing the "frog-face" deformity. The cardinal symptoms are nasal obstruction and discharge, with headache and severe epistaxis; aural complications may follow, or the eyes may be involved, with diplopia, exophthalmos and compression of the optic nerve; and, finally, death results from exhaustion, hæmorrhage, sepsis or cerebral invasion. Histologically the tumour is composed of fibrous tissue containing numerous thin-walled blood vessels and a variable admixture of round or fusiform cells, so that in some cases it might be described as a fibro-sarcoma; but it is not truly malignant, for it neither involves the glands nor becomes disseminated, though there is a tendency to local recurrence after removal.

Radium has proved of considerable value in reducing the size and vascularity of these growths, as a preliminary to operation.

Malignant tumours are not common in the naso-pharynx, but epithelioma, sarcoma and endothelioma all occur. The early symptoms are chiefly pains of a neuralgic character and those produced by Eustachian obstruction;

later, epistaxis, nasal obstruction, secondary involvement of glands and affections of the eye and cranial cavity may appear. Surgical removal is rarely feasible, but sarcomata and endotheliomata in this region sometimes yield remarkably to treatment by radium.

HAROLD S. BARWELL.

DISEASES OF THE LARYNX

ACUTE CATARRHAL LARYNGITIS

Ætiology.—The affection ordinarily occurs as part of a coryza, or cold, the inflammation spreading downwards from the nose or naso-pharynx. It is also caused by over-use of the voice, especially with faulty voice-production, and frequently a slight catarrh is made worse by using the voice during a cold. It occurs in many infectious fevers, *e.g.* influenza, measles, scarlet fever, typhoid and small-pox, and it is occasionally a result of traumatism, instrumentation, or the inhalation of irritating fumes in chemical works or of the gases used in warfar. Predisposing causes are chiefly those factors which favour attacks of coryza, such as nasal obstruction or discharge, sedentary occupations and overheated rooms; apart from local tuberculous lesions, consumptives are very subject to laryngeal catarrh.

Symptoms.—The symptoms consist of hoarseness or aphonia, local discomfort varying from dryness or tickling to a burning sensation or actual pain, and irritable cough. There is little expectoration, unless the trachea and bronchi are involved. At the onset there may be slight feverishness and malaise. The degree of hoarseness is by no means proportionate to the objective appearances; the voice may be quite good in cases of decided hyperæmia, and may be completely lost when little abnormal is to be seen. This depends largely on the neuro-muscular tone; a muscular man will retain a strong voice with a degree of inflammation which would render a weakly woman completely aphonic—indeed some women lose the voice with every slight cold, so that it becomes difficult to differentiate between laryngeal catarrh and “functional aphonia.” On the other hand, in some voice-users redness of the cords appears to be the normal condition and causes no interference with function. This variable effect on the voice is to be observed in all forms of laryngeal disease. In children, acute laryngitis is a serious affection. They show a far greater tendency to œdema and to spasm, and as the glottis is not only absolutely but relatively smaller than in adults, a dangerous dyspnoea may ensue with great rapidity. The larynx is reddened, and this is most obvious on the parts usually pale—the epiglottis and vocal cords, the vessels on the former being unduly prominent. The cords may be red, pink, yellowish, or merely have lost their bright pearly lustre. A small amount of mucous secretion is generally present, but no large accumulations or strings of mucus, such as are seen in chronic laryngitis. There is often a little swelling of the cords so that, on phonation, their edges come into contact at the centre; this explains how singers’ nodules are caused by use of the voice during a laryngitis.

Treatment.—People suffering from the slighter degrees of laryngitis

rarely apply for treatment, unless they are professional voice-users. The patient should remain in a warm (65° F.), well-ventilated room, preferably in bed, and must not attempt to use the voice. The coryza, if present, should be treated at the same time. Steam inhalations are of value and may be used from an inhaler or from a jug round the mouth of which a towel has been wrapped in the shape of a cone. The water should be at a temperature of 130° to 140° F., and one of the following medicaments may be added in the proportion of one drachm to the pint: Compound tincture of benzoin (Friar's balsam) fl. oz. 1, with or without menthol, grs. 10–15; or benzoic acid, grs. 3, kaolin, grs. 12, tincture of tolu, min. 18, and water to fl. oz. 1, these being sedative, while oleum pini sylvestris, min. 40, magnesii carb. levis, grs., 20, water to fl. oz. 1 is mildly stimulating. Steam inhalations should only be ordered when the patient can remain in a warm room; when he is not confined to the house, or in a later stage, an oily solution from an atomiser is preferable, such as menthol, grs. 7, camphor, grs. 3, chlorbutol, grs. 5, liquid paraffin, fl. oz. 1. Internally, expectorants are indicated; tinctura ipecacuanhæ, min. 10, or vinum antimoniale, min. 5, potassium iodide, grs. h2 or 3, ammonium carbonate, gr. 4—singly or in combination—or amonium chloride, grs. 5, or oil of cubebs, min. 5 in syrup, every 4 or 6 hours. If cough is severe it should be restrained; a lozenge of morphine and ipecacuanha is useful, or a linctus containing diamorphine hydrochloride, gr. $\frac{1}{4}$, or liquor morphinæ, min. 2 to 4 to each drachm.

The acute laryngitis of children calls for prompt treatment. One or two grains of calomel may be given every 3 hours until the bowels have acted freely, after which it may be continued in $\frac{1}{2}$ -gr. doses three times a day. Hot fomentations to the neck and a steam-kettle are advisable, and in acute febrile cases, 1 minim each of tincture of aconite and vinum antimoniale every 3 hours. If dyspnœa occurs, an emetic dose of ipecacuanha often gives prompt relief; 60 minims of the tincture followed by 30 minim doses every half-hour until vomiting occurs: in very young or weakly children, 15 minims every quarter-hour is to be preferred.

SPASMODIC LARYNGITIS

Synonym.—Laryngitis Stridula.

This is simply catarrhal laryngitis with spasm of the glottis as a marked feature. It is a disease of childhood and is predisposed to by general ill-health, rickets and adenoids. The onset is that of an ordinary cold, with slight feverishness, hoarseness and a frequent cough, and during the evening or night the respiration becomes embarrassed. There is inspiratory stridor, recession of the epigastrium and lower ribs and, in some cases, an alarming degree of asphyxia. The symptoms tend to subside towards morning and, though they may recur on the next few nights, it is usually with diminishing severity. The condition should be distinguished from laryngismus stridulus, in which there is no hoarseness or other symptoms between the attacks. The evanescence of the symptoms serves to distinguish it from cedematous laryngitis and from diphtheria, in which the attacks become increasingly severe. The general health requires attention, and adenoids, if present, must be removed after recovery has taken place.

ŒDEMATOUS LARYNGITIS

Synonym.—Œdema of the Larynx.

Ætiology.—Œdema of the larynx is not a disease but a pathological condition due to a variety of causes. Non-inflammatory œdema may be mentioned here for the sake of completeness; it occurs, though rarely, as part of the general anasarca of renal and cardiac disease. Angio-neurotic œdema sometimes occurs in the larynx, in which event it produces rapid and sometimes fatal dyspnoea (see p. 1074). The swelling which occasionally results from administration of potassium iodide in susceptible subjects may be placed in the same category.

Inflammatory œdema seldom results in adults from a simple catarrh, but it may do so in children; it more often occurs as part of an acute septic infection of the pharynx, trachea and bronchi, "acute fulminating laryngo-tracheo-bronchitis." Œdema may follow various forms of traumatism, the drinking of corrosive poisons, inhalation of irritating vapours, such as the poison gases of warfare, the lodgment of foreign bodies, or rough or unduly prolonged bronchoscopy. Scalding, from attempts to drink from a kettle-spout, is a common cause among children. In other cases it is a sequela of typhoid fever, pneumonia, scarlet fever or small-pox, and is a local complication of syphilitic, tuberculous, cancerous or traumatic ulceration.

Symptoms.—If part of a septic pharyngo-laryngitis, the general symptoms are severe. The chief local symptom is dyspnoea with inspiratory stridor and the associated symptoms of asphyxiation; there is hoarseness or aphonia, local discomfort and tenderness, and sometimes dysphagia. The aryteno-epiglottidean folds are enormously swollen, appearing as pale or purple translucent flask-shaped masses; if the epiglottis be œdematous it forms a sausage-shaped swelling of the same appearance. The mucosa of the vocal cords is too adherent to permit much swelling, and "œdema of the glottis" is therefore a misnomer. The subglottic region is lax and may become swollen; indeed, the œdema may be confined to this region and then appears as a red swelling below each vocal cord. In children œdema may be inferred from the steadily increasing dyspnoea without the rapid increase and decrease typical of spasmodic laryngitis.

Treatment.—In slight cases, the swelling may be reduced by sucking ice and by the application of an ice-bag to the neck; the latter is inadmissible in young children. A spray of adrenaline, 1 in 1000, may be used. Hypodermic injections of pilocarpine, gr. $\frac{1}{2}$, are recommended and, for the œdema produced by iodides, large doses of bicarbonate of soda. Free scarification of the œdematous tissues at the upper aperture should be practised without undue delay; in adults this should be done with a laryngeal lancet under guidance of the mirror, but in children it is best performed with a curved bistoury guarded to near the point with strapping and passed down along the left forefinger as a guide. If this does not give quick relief, or if the dyspnoea be severe, tracheotomy should be performed without delay. Intubation is not suitable for cases of œdema of the upper aperture of the larynx, though it may be employed for subglottic cases, provided that skilled attention be immediately available should the tube be coughed out. Angio-neurotic œdema should be treated by a spray of adrenaline, and the same

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drug, or a colloidal preparation of calcium, may be injected hypodermically ; tracheotomy may here also be required.

MEMBRANOUS LARYNGITIS

The formation of false membrane in the larynx is nearly always part of an attack of diphtheria, which is discussed elsewhere, but by the term "membranous laryngitis" is implied a formation of membrane of non-diphtheritic origin. Apart from traumatic cases, due to irritating chemicals and scalds, inflammation of the larynx accompanied by membrane may be caused by streptococcal infection. The affection occurs especially in children between the ages of 2 and 8 years. The first symptom is hoarseness, soon followed by a brassy cough and the signs of dyspnoea ; the patient is restless and the temperature rises rapidly to 103° or 104°. In some cases, however, the disease takes a subacute form, the attacks of dyspnoea being worse at night and abating towards morning. The diagnosis from diphtheria is only possible by bacteriological examination, and pending the report the case should be treated with antitoxin ; but it may be noted that the pharynx is nearly always involved in diphtheria, whereas in membranous laryngitis the disease is often primary in the larynx. The prognosis is grave, worse than that of diphtheria since the introduction of antitoxin. An emetic dose of ipecacuanha should be given, and a steam-tent and hot fomentations to the neck are advisable. Calomel treatment is recommended ; 1 or 2 grains every 3 hours until the bowels have acted freely, and subsequently 1 grain three or four times a day ; the sulphanilamide group of drugs will doubtless be of value in this, as in other streptococcal infections. Tracheotomy or intubation must not be delayed when there is serious dyspnoea.

CROUP

Croup is an expression which dates from a period when the diagnosis of diseases of the throat was less exact than now but, as it is still in occasional use, it may well receive a definition in this place. The term can be used to cover any respiratory obstruction or obstructive dyspnoea, especially in children, but is usually limited to acute affections and therefore does not ordinarily include such conditions as multiple papillomata, congenital web or cicatricial stenosis. Croup may thus be caused by various inflammatory affections such as cedematous laryngitis, membranous laryngitis (both diphtheritic and non-diphtheritic), spasmodic laryngitis and retropharyngeal abscess, or by such reflex disturbances as laryngismus stridulus and spasm of the glottis, and is in fact descriptive of a symptom rather than of a disease.

CHRONIC LARYNGITIS

Ætiology.—The causation is similar to that of acute catarrhal laryngitis ; indeed, chronic laryngitis is often the result of recurrent or persistent acute catarrh. The principal factors which predispose to chronicity are nasal

obstructions and discharges, dusty occupations and lack of fresh air, over-use of the voice and faulty voice-production, and the abuse of alcohol or tobacco ; consumptives are particularly liable to non-specific catarrhal laryngitis, and oral sepsis must not be omitted. Almost any cause of general ill-health may be included among the predisposing causes, such as gout, rheumatism, anæmia, and gastro-intestinal, cardiac and hepatic disorders.

Symptoms.—The only constant symptom is impairment of the voice, which is hoarse, easily tired or even, though rarely, completely aphonic ; it is sometimes weakest when tired in the evening, but is often at its worst on rising in the morning or after a rest. There is frequently a sensation of aching, dryness, tickling or of a lump in the throat, and there is usually some cough, but little expectoration unless the trachea and bronchi are involved.

The objective appearances vary with the severity of the affection. The larynx generally is of a deeper red than usual, and the vocal cords have lost their normal pearly lustre and are pink or grey ; they are usually somewhat thickened at the edges, and enlarged vessels may be visible on their surface ; the vocal processes are often prominent and may be reddened or show up white against the hyperæmic cord. Strings of sticky secretion may stretch between the cords, or a little globule of mucus may form on the centre of the cord during phonation ; adduction is frequently imperfect. When the epiglottis is reddened, its yellow edge stands out clearly and enlarged vessels are visible ; the ventricular bands are often swollen so as to hide the outer part of the cords. The mucous membrane in the inter-arytenoid space, thickened and relaxed, is seen to be thrown into folds on adduction of the cords, and may form a mass large enough to prevent their approximation.

Atrophic rhinitis usually produces a form of inflammation, *laryngitis sicca*, in which small brown scabs adhere to the cords and posterior commissure, but occasionally the disease itself spreads to the larynx, which is covered by large greenish or brownish-black fetid crusts ; more rarely still the crusts extend into the trachea and cause severe dyspnoea, which may prove fatal.

Pachydermia laryngis is a somewhat rare variety of chronic laryngitis, occurring principally in middle-aged men. It is frequently ascribed to alcoholism, perhaps on insufficient grounds, to syphilis and to tubercle ; the diagnosis between pachydermia and these two diseases is, however, often a matter of difficulty. The characteristic epithelial thickenings are probably of the nature of corns, resulting from frequent cough and continued irritation. There is hoarseness of a rough raucous character, but no particular discomfort. The epithelial thickening is pink or whitish and occupies the posterior or cartilaginous region of the glottis from the vocal processes backwards to the posterior commissure. A circumscribed swelling appears on each vocal process, with a small cup or depression at the apex ; the approximation of the cords is better than would be expected, because the prominence on one vocal process fits into the depression at the other. The epithelium of the inter-arytenoid space is thrown into ridges, which fill up the angles between the arytenoid and the posterior commissure, but leave a depression in the middle line. These firm, opaque, symmetrical swellings, without ulceration, are distinguishable from the soft irregular granulations

of a tuberculous lesion, and the cup-shaped swelling on the vocal process, even when more marked on one side, should not be mistaken for an early epithelioma.

Treatment.—The detection and correction of the aetiological factors are the most important part of treatment. Any constitutional disturbance, such as anæmia, rheumatism, gout or dyspepsia, should receive attention. Over-indulgence in tobacco or alcohol, lack of ventilation and exposure to dust must be considered, and with teachers the black-board chalk is a common source of irritation.

Incorrect voice-production is a factor of great importance especially, but by no means exclusively, among those who use the voice largely in their occupations; in such, a course of lessons in voice-production often works wonders. In a large proportion of cases the primary cause of the laryngitis is to be found in the nose, therefore any source of nasal obstruction, catarrh or suppuration must be carefully looked for and treated; any concomitant pharyngitis should also receive attention.

Locally, treatment must begin with rest of the voice, which should be absolute in the case of professional voice-users. Where there is much secretion a saline lotion may be used in a spray—sodium bicarbonate, borax, sodium chloride, 10 grains of each, thymol water, 120 minims, glycerin, 60 minims, water to 1 ounce. Oily solutions are usually preferred, such as menthol, 5 grains, camphor, 2 grains, chlorbutol, 5 grains, or oil of eucalyptus, pine or gaultheria in similar proportions, to an ounce of liquid paraffin. The direct application of pigments is not often called for, and is to be recommended only when pachydermatous changes are present; in such cases the cautious application of a solution of nitrate of silver on a cotton-wool mop once a week may be tried, beginning with 5 grains to the ounce and increasing the strength gradually to 50 or more grains. Dundas Grant advises an alcoholic solution of salicylic acid, beginning with 1 per cent. and increasing to 6 or 8 per cent.

Internally, small doses of potassium iodide, 2 or 3 grains, or the yellow proto-iodide of mercury, $\frac{1}{18}$ grain, three times a day over long periods, is of value.

CONGENITAL LARYNGEAL STRIDOR

In this condition there is an exaggeration of the infantile shape of the upper aperture of the larynx; the epiglottis is sharply folded laterally, the ary-epiglottic folds are almost in contact, and the opening is thus reduced to a narrow vertical slit. As these parts are very flaccid in infancy, they become sucked together during inspiration and, by their vibration, produce the characteristic stridor. This stridor is noticed very soon after birth, it is inspiratory, of a peculiar purring or even musical character, and is most marked during active breathing and crying. The voice is unaffected, and there is remarkably little sign of dyspnoea or distress. These characteristics distinguish the condition from other forms of obstruction found in infants, such as laryngeal webs or papillomata, or "thymic asthma." It tends to disappear during the second year of life, but the prognosis must be guarded in early infancy, for an attack of bronchitis is more than ordinarily dangerous and kills a proportion of these patients.

SYPHILIS

CONGENITAL SYPHILIS

The early, or secondary, form appears in the first few months of life and is rarely recognised, but it may be suspected when the cry is hoarse in an infant with active syphilitic lesions.

Tertiary lesions are rare, and usually make their appearance about puberty, less often during the second dentition. The disease takes the form of diffuse infiltration with or without ulceration; the swelling may produce obstruction, and rarely cicatricial stenosis may ensue. The symptoms are stridor with hoarseness, and tracheotomy may be required.

ACQUIRED SYPHILIS

Symptoms.—Secondary lesions are superficial, cause no symptoms but hoarseness, and seldom come under observation. The commonest manifestation is an erythema which differs from that of catarrhal laryngitis by being more uneven and patchy in its distribution, and may affect one cord, leaving the other normal. Mucous patches are occasionally found on the cords or on any part of the larynx, appearing as superficial erosions with a smooth greyish base and a sharply defined hyperæmic margin. The fauces are nearly always affected at the same time.

Of tertiary lesions, the superficial serpiginous ulcer is occasionally seen with the same characters with which it more commonly appears on the fauces. Diffuse infiltration may attack any part of the larynx, but chiefly, in contra-distinction to tuberculosis, the anterior regions, such as the epiglottis and the front parts of the vocal cords. Subglottic infiltration is fairly frequent and abduction of the cords often limited, so that stenosis is much commoner than in tuberculous disease. The typical circumscribed gumma is distinctly rare; it is single, unilateral, and attacks especially the epiglottis and arytenoids, and usually breaks down rapidly to form a deep excavated ulcer, which may result in perichondritis, exfoliation of cartilage, and, ultimately, in severe cicatricial stenosis. The subjective symptoms are hoarseness, of a peculiar rough "raucous" character, and sometimes dyspnoea with stridor; pain is in general not a prominent symptom, but a gumma on the upper aperture may cause severe dysphagia.

Diagnosis.—From tuberculous disease the diagnosis is discussed under that heading (see p. 1107). From epithelioma a gumma is distinguished by its more rapid evolution; the edge of an epithelioma is thick and everted and its base nodular, whereas these characters are less marked in syphilitic ulceration, the margin of which is hyperæmic and frequently sharply cut; while other parts of the larynx or fauces often show syphilitic lesions. The hard infiltration of secondarily involved glands is characteristic of malignant disease.

Treatment.—General treatment is urgently called for to prevent perichondritis and stenosis. Local treatment is not often required. Tracheotomy should be performed when decided dyspnoea is present; it seems to aid the recovery of the larynx, and the tube can often be omitted in a short

time, when anti-syphilitic medication has removed the obstructing lesion. Necrosed pieces of cartilage must be removed by internal or external operation, and insufflations of orthoform are indicated when dysphagia is present.

LUPUS

Lupus in the larynx is comparatively rare, and is probably always secondary to the disease in the nasal passages.

Symptoms.—The lesions begin on the epiglottis and slowly spread along the aryteno-epiglottic folds; the interior of the larynx is less often attacked and the cords usually escape. The infiltration is composed of tiny red nodules, which develop the typical "apple-jelly" centre and break down to form multiple coalescent shallow ulcers, the smooth base covered by a scanty secretion and with indefinite uninfamed margins. Cicatricial contraction goes on during the progress of the affection, and the epiglottis, if not destroyed, is usually much deformed; but the scars are less thick, and the contraction less severe than in syphilis, and marked stenosis is less common.

Treatment.—The disease shows a decided tendency to spontaneous cure, and in many cases of cutaneous lupus the scars of healed disease can be seen in the larynx. Open-air treatment, as carried out in a sanatorium, with good food, moderate exercise, and cod-liver oil suffices to cure most cases. Arsenic, in large doses, appears to have a specific effect, starting with 5 minims of liquor arsenicalis 3 times a day and increasing the dose gradually to 15 or more minims. Local treatment should be reserved for those cases which general measures fail to cure. If the lesions are confined to the epiglottis, this may be removed; for more diffuse infiltration repeated galvanocautery puncture gives the best results, but over-zealous application will promote stenosis. Good results have been reported from the use of radium, applied externally to the neck in the form of plates.

TUBERCULOSIS

Ætiology.—Primary tuberculosis of the larynx is so rare as to be of no practical importance; in the overwhelming majority of cases the disease is secondary to pulmonary tuberculosis, of which it is a common and important complication. It is probably caused by infection from the sputum, is two or three times commoner in men than in women, and is most frequent between the ages of 20 and 40. St Clair Thomson finds that the difference in sex-incidence is occupational, and that women working in office and factory are as susceptible as men.

Symptoms.—The disease attacks, in order of frequency, the vocal cords, arytenoid region, inter-arytenoid space, ventricular bands and epiglottis; in general the lumen is invaded before the upper aperture, and the posterior rather than the anterior parts of the larynx. The typical infiltration is finely nodular, pallid and soft in appearance; ulcers are shallow, with a smooth speckly base and pale ill-defined margin. On the vocal cord the disease chiefly attacks the posterior half and especially the vocal process, where ulceration readily reaches the underlying cartilage and may produce a deep

triangular excavation. Thickening in the inter-arytenoid region is common; infiltration of the arytenoids results in typical pale semi-translucent flask-shaped swellings, while the epiglottis appears as a firmer red sausage-shaped mass.

Of subjective symptoms, the hoarseness is very characteristic, the voice being weak and effortless and very different from the raucous voice of syphilis. Cough and expectoration are mostly due to the pulmonary disease and not in any considerable degree to the larynx. Pain on swallowing is common and often very intense; there may also be actual obstruction to deglutition and, in a late stage, entry of food into the larynx. Dyspnoea is rare.

Diagnosis.—Although signs of pulmonary tuberculosis are helpful in diagnosis, it is obvious that any kind of laryngeal disease may occur in a consumptive patient.

From simple laryngitis.—In the earliest stage of invasion tuberculous laryngitis may exactly resemble catarrh, but redness of one cord only is certainly not due to catarrh, and the latter quickly improves under treatment. The swollen arytenoids of oedematous laryngitis are less pale and more transparent, while the affection is acute and the entire larynx inflamed. Inter-arytenoid infiltration resembles pachydermia, but the latter is opaquely white, firm and symmetrical.

From lupus, typical tuberculosis differs completely. The former is painless, affects first the epiglottis and upper aperture, is never accompanied by oedema, and tends to cicatrization. But there is a chronic "lupoid" form of tuberculous laryngitis which attacks the epiglottis and is very similar to lupus.

From syphilis.—The tuberculous ulcer has an ill-defined margin without surrounding hyperæmia; the base has a yellow speckled appearance, and on healing there is little scarring or contraction. The superficial syphilitic ulcer has a well-defined hyperæmic margin, with a smooth, flat base; the deep ulcer is "crateriform," with thickened punched-out edge, and, on healing, leaves a dense scar and marked deformity. In general, syphilitic lesions attack the anterior half of the larynx, tuberculous the posterior; the former look firm and dense, the latter soft, translucent and ill-defined.

From neoplasms.—Only the rare tuberculomata resemble innocent tumours. Occasionally tuberculosis attacks one vocal cord in an elderly patient, and may then easily be mistaken for epithelioma (see p. 1111).

Prognosis.—Any tuberculous lesion of the larynx renders the prognosis of a case of pulmonary tuberculosis much more serious. A considerable number of the superficial lesions become healed; but it is doubtful if any cases of extensive infiltration recover, with the exception of a few rare instances where it is confined to the epiglottis and can be entirely removed.

Treatment.—Tuberculous laryngitis is but a complication of pulmonary tuberculosis, and by far the most important part of the treatment is that of the general infection. For the laryngeal lesions the most valuable remedy is complete silence, but it is a severe and depressing measure and should not be insisted on unless there is a prospect of cure; the pain and irritation

in advanced cases are, however, often relieved by vocal rest. Any concomitant catarrh should receive attention; an oily spray containing menthol and chlorbutol (7 grains of each in an ounce of liquid paraffin) is valuable, and irritable cough should be relieved by a simple lozenge, or, if severe, by heroin, $\frac{1}{12}$ gr. or less in a lozenge or linctus. Attempts to cure by active local treatment must only be made when the pulmonary lesions are quiescent or progressing towards arrest, the general health good, and the local lesions not very extensive. Of these methods the galvano-cautery is the most generally useful, and may be employed to the surface of superficial ulcers, or as multiple puncture of infiltrated areas. Chemical caustics may be applied to ulcerated surfaces, especially on the cords and posterior commissure; lactic acid, 50 to 80 per cent., may be used, or Lake's mixture of lactic acid 50 per cent., formalin 7 per cent., and phenol 10 per cent. Ulcers covered with sprouting granulations may be curetted, and occasionally infiltration of the epiglottis or arytenoid may be removed with punch-forceps.

In advanced cases the dysphagia is so distressing that its relief is of great importance. For this purpose the most valuable drug is orthocaine (orthoform), which may be combined in equal proportions with benzocaine (anæsthesine); it is an insoluble non-toxic powder and is used as an insufflation in doses of 3 to 5 grains half an hour before meals; patients readily learn to inhale it into the throat through a glass tube. Cocaine and morphine should be reserved to the last stages. When the dysphagia is due to infiltration of the epiglottis, the greatest relief is afforded by its removal under cocaine with special punch-forceps; and when the pain is caused by a tense swollen arytenoid, the removal of a piece with punch-forceps relieves tension and gives similar relief. Injection of alcohol into the superior laryngeal nerve is a useful method of alleviating pain in cases of extensive disease. Tracheotomy is seldom required, and tuberculous infection of the wound is common after this operation.

TUMOURS

INNOCENT TUMOURS

Singer's nodules are inflammatory epithelial thickenings, and may be considered as a form of pachydermia. They are found in teachers and speakers, rather than in singers, and are caused by faulty voice-production and, especially, by forcing the voice when the cords are inflamed. The appearance is that of a minute pink or whitish nodule on the edge or upper surface of the vocal cord, surrounded by a varying amount of injection; there is usually a nodule symmetrically placed on each cord, but it is frequently larger on one side than on the other. The place where the growth occurs, and which is also the "seat of election" for other innocent tumours, is at the junction of the anterior and middle thirds of the glottis, that is, in the centre of the true vocal cord, for the posterior third of the glottis is cartilaginous. It is here that the cord, if swollen, comes into contact with its fellow on phonation.

Fibromata occur usually on the vocal cord on the same site as the singer's nodule, of which they are in some cases probably a development; or at the

anterior commissure. They are pedunculated, smooth and round, white, pink, or brown from extravasation of blood, and vary from the size of a pin's head to that of a bean. These growths not infrequently become œdematous, when they appear translucent like a nasal polypus, and have been incorrectly described as *myxomata*.

Papillomata are reddish, papillary, pedunculated growths, and occur anywhere on the larynx, but, when single, generally occupy the "seat of election" on the vocal cord, and are seldom found on the posterior half of the glottis. Multiple papillomata occur especially in childhood and, as they present special characteristics, will be considered separately later.

Cysts.—A fibroma may degenerate with the formation of a cystic space in its interior. Apart from this, cysts, which may reach a large size, are found as a rarity on the upper aperture of the larynx, especially on the anterior surface of the epiglottis. They are thin-walled and translucent, with ramifying vessels running over the surface.

Angiomata occur, though rarely, on any part of the larynx either as a flat patch or a raised purple mass resembling a blackberry.

All innocent neoplasms are rather uncommon; in addition to those already mentioned, *lipomata*, *chondromata* and *thyroid-gland tumours* have been observed.

Symptoms.—A tiny growth situated on the vocal cords, or in such a situation as to prevent their approximation, causes hoarseness and a variable amount of aching and discomfort, but even a large tumour elsewhere may cause no symptoms to attract attention. Dyspnoea results in rare cases when a neoplasm is large enough to block the air-way, but it is astonishing how slight a disturbance may be produced by a large tumour if it has grown slowly; inspiration is more difficult than expiration, except when the tumour lies below the glottis. Angiomata cause hæmorrhage, which may be very severe.

Diagnosis.—This is usually easy on inspection, but a growth on the anterior commissure, or one that drops down below the cords, may be readily overlooked. The rare tuberculoma may so exactly imitate an innocent neoplasm as to be only recognisable under the microscope. The important matter of diagnosis from a malignant neoplasm will be referred to under the latter disease.

Treatment.—Tumours situated away from the glottis and causing no symptoms should be left alone. Cysts are treated by making a large hole with punch-forceps or the cautery, for they refill after simple incision. Angiomata, especially if diffuse, are best left untouched, unless bleeding calls for interference, in which case it can usually be checked by the cautery at dull-red heat; diathermy puncture with a fine terminal through a direct laryngeal spatula, or by suspension-laryngoscopy, is a preferable method; if repeated hæmorrhage becomes a danger, the angioma can be excised by an external operation, but is usually more widely spread than appears on laryngoscopic examination. Singer's nodules, if quite small and sessile, frequently disappear under prolonged rest of the voice; the smaller nodules may be lightly touched with the galvano-cautery; larger ones should be removed with forceps. Fibromata and papillomata are removed with forceps; it causes less disturbance to the patient if this be done under cocaine anæsthesia by the "indirect method," i.e. under guidance with the laryngeal mirror

provided that the operator has acquired the necessary skill, but since the introduction of the "direct method" they are usually removed through the tube-spatula. After the little operation, absolute rest of the voice should be enjoined for a few days; in cases of singer's nodule a longer rest is necessary, with training in voice-production and avoidance of dust and conditions of vocal strain.

Multiple papillomata.—This rare but serious condition occurs almost exclusively in children and generally attracts attention between the ages of 2 and 4. The warts may be very numerous, fill the lumen with a cauliflower-like mass, and extend to the subglottic region, down the trachea and sometimes on to the pharynx. The first symptom is hoarseness, and long-continued hoarseness in a child should suggest the possibility of papilloma; dyspnoea ensues later and becomes gradually more severe until tracheotomy is necessary. The growths may disappear after tracheotomy, or spontaneously, or after an acute illness, and tend to vanish or cease to recur about puberty. The growths should be removed through the tube-spatula, or by suspension-laryngoscopy, under general anæsthesia; a preliminary tracheotomy is advisable if there is much dyspnoea; several sittings may be required, and the operation must be repeated as often as the growths recur.

MALIGNANT TUMOURS

Ætiology and Pathology.—Epithelioma is by far the commonest malignant growth in the larynx, but spheroidal-cell carcinoma and sarcoma also occur. Malignant disease of the larynx proper is rare below the age of 40, and is seldom seen in women; but there is a post-cricoid epithelioma, originating on the mucous membrane of the pharynx, which is relatively common in women, and has been known to occur at such an early age as 23. Secondary or metastatic growths are practically unknown in the larynx; on the other hand, owing to the fact that the laryngeal lymphatics do not anastomose freely with other systems, cancers confined to the lumen of the larynx rarely become disseminated and do not readily infect the glands. Therefore, Krishaber's classification into *intrinsic* and *extrinsic* is valuable for treatment and prognosis; the former are those situated within the cavity of the larynx, while the latter affect the upper aperture, epiglottis, arytenoids, and the outer walls.

Symptoms.—Unfortunately, intrinsic malignant tumours do not cause severe symptoms at an early stage and, particularly in hospital practice, frequently do not present themselves for treatment until they have become extrinsic by extension; hence the importance of a laryngeal examination in all patients over 40 with hoarseness which does not rapidly yield to treatment. Hoarseness is the most general, and usually the only, early symptom; owing to the deep infiltration characteristic of malignant disease, it is often more marked than the size of the tumour would appear to warrant. Cough is not a common symptom. Pain is absent in the early stages of intrinsic cancer, but is severe in the later stages and in the extrinsic forms; it radiates to the ear and side of the head, and is made worse by swallowing, speaking and coughing. The later symptoms include involvement of the glands, fetor of the breath, salivation, hæmorrhage, dyspnoea, dysphagia and general cachexia; often the patient dies of septic pneumonia.

On a vocal cord, epithelioma may appear as a definite tumour resembling a papilloma or angioma, or it may begin as a diffuse infiltration, or merely as a localised area of thickening and congestion. On the ventricular band or posterior commissure it usually shows itself as an irregular papillary dusky-red swelling; cancer of the epiglottis ulcerates early and appears as a dirty white or reddish tumefaction. Often the only sign of a post-cricoid growth is a swollen and fixed arytenoid, or merely a pool of saliva in the pyriform fossa; sometimes the upper edge of the growth is visible, but it may be necessary to pass a tube-spatula, or to pull the larynx forward with a probe passed down to the glottis, before it can be seen.

Diagnosis.—In the earliest stage the diagnosis obviously is a matter of extreme importance and sometimes one of great difficulty. The unilateral character of the infiltration is ordinarily sufficient to exclude a simple inflammatory lesion; though pachydermia may be more marked on one side, the lesions are bilateral and the smooth cup-shaped swelling on the vocal process is characteristic. The difficulties of diagnosis are generally between an innocent neoplasm on the one hand, and a tuberculous or syphilitic infiltration on the other. A papilloma or a fibroma on a vocal cord should be regarded with suspicion in a man over 40, and especially if, after removal, the site fails to heal promptly. An innocent neoplasm has a fine pedicle, moves freely in the air-current on phonation, there is no tumefaction at its site of origin, and it is found at or in front of the middle of the vocal cord. A malignant growth may occur in any situation; it is less movable and pedunculated, the cord in the neighbourhood is frequently swollen and may show a leash of tiny vessels running to the tumour; a white spiky appearance of the papillæ is suggestive of malignancy; a sluggish delayed movement of the cord is an important sign, insisted on by Semon, but its absence by no means excludes malignancy, for it only occurs when the growth has infiltrated the muscles, and its presence increases the gravity of the prognosis. Epithelioma beginning as a flat infiltration may resemble a tuberculous or syphilitic lesion, but usually other signs of these diseases are present to aid the diagnosis; in the latter the Wassermann reaction and, still more, the effect of vigorous anti-syphilitic treatment will clear up the doubt. There is a form of senile tuberculosis in which infiltration of one vocal cord may closely imitate epithelioma, more especially as the pulmonary signs are merely those of bronchitis and tubercle bacilli are very scanty in the sputum. Sometimes a piece may be removed for examination, but frequently the growth is too sessile to permit it; the piece removed must not be very small, and a negative finding is of little value, for the tip of a malignant growth cannot always be distinguished microscopically from a papilloma. Biopsy is more important since the adoption of Broders' histological method of estimating the malignancy and radio-sensitivity of tumours, but it should not be performed in cases too advanced for treatment, for it stimulates the growth and does harm.

Extrinsic epithelioma is most likely to be mistaken for tertiary syphilitic ulceration. A gumma grows and ulcerates more quickly, its edge is smoother and sharper, and its base often covered by a yellow slough. Pain has little diagnostic value, for a gumma of the upper aperture may cause severe dysphagia. Palpation with the finger is of great service, for the firm hard feel of an epithelioma is very characteristic.

Treatment.—Malignant disease cannot be removed with certainty by

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the natural passages. Thyrotomy, or laryngo-fissure, is the operation of choice for removal of intrinsic cancer ; it consists essentially in wide excision of the disease from the interior of the larynx after splitting the thyroid cartilage in the middle line. The results in suitable cases are probably better than those of removal of malignant disease in any other part of the body ; there is now practically no immediate mortality in skilled hands, statistics show from 70 to 80 per cent. of permanent cures, and a useful husky voice remains. When, however, the growth has spread at all extensively to the arytenoid or to the subglottic region, laryngo-fissure is no longer possible and a very different picture is presented. By modern methods of laryngectomy the trachea is fixed in the lower part of the neck, aspiration of septic matter is avoided, and the mortality has been much reduced, while the results as regards freedom from recurrence have been greatly improved. The operation leaves the patient in a condition very different from that after thyrotomy ; as he breathes through the tracheal opening in the neck he cannot cough or strain and has no natural voice. Nevertheless he can often produce a fairly audible whisper by means of the air in the pharynx, or with the aid of a tube leading from the tracheotomy wound into the mouth or nose, and a number of patients succeed in passing a surprisingly happy and useful existence. Subhyoid pharyngotomy and lateral pharyngotomy are operations designed to obtain access to cancers at the upper aperture of the larynx ; the latter operation gives very good results in strictly limited tumours of this region. The technique of therapy by means of X-rays and radium has now been put on a reliable basis by determination of the appropriate dosage and by adequate screening ; in many cases of malignant tumours of the larynx, great improvement can be obtained by both these agencies, and a considerable proportion of cures has been effected. A valuable method, in intrinsic cancer, is the insertion of radium tubes through a window cut in the thyroid ala. Great care and judgment are required in the selection of cases for these various operations, and very many come under observation too late. In these much can be done by palliative treatment ; careful attention to the hygiene of the mouth and teeth is of great importance, together with mild antiseptic laryngeal sprays containing hydrogen peroxide, listerine, sanitas or phenol. For dysphagia, insufflations of powdered orthocaine, with or without benzocaine, is the most reliable remedy ; cocaine is disappointing, for its effect is very transient and the resulting numbness interferes with swallowing ; the judicious administration of morphine or heroin is eventually necessary, but the local application of these drugs is useless. Palliative tracheotomy or gastrotomy may be called for, and curettage or partial removal by knife or diathermy gives relief in some cases of cancer at the upper aperture.

PARALYSIS

Paralysis of a vocal cord is a frequent symptom of various diseases of the thorax and of the nervous system, and the laryngoscope is therefore of great value to the physician as an aid to diagnosis ; this is more especially the case in that the common early form, abductor paralysis, causes no symptoms, and can only be recognised by laryngoscopic examination.

The original function of the laryngeal muscles is that of a sphincter to

prevent the entrance of fluid into the lungs, and this sphincter, or adductor, is the only muscle present in the larynx of the most primitive air-breathing animals; the abductors are a later addition, to assist the entry of air. Accordingly, in lesions of the nerve path, the abductor muscles are affected first and the primitive adductors are more resistant. But the function of phonation, much more recently acquired, is associated with adduction, and is under direct control of the will. Functional disturbances, therefore, always cause adductor paralysis, while organic lesions first affect the movement of abduction.

ORGANIC PARALYSIS

The crico-thyroid muscle is supplied by the superior laryngeal nerve, and, when this is injured, the affected cord remains slack on phonation, but, owing to the short course of the nerve, isolated paralysis of this muscle is extremely rare; it results from surgical or suicidal wounds, or by pressure from glands, but most often occurs after diphtheria. In lesions of the vagus above the origin of this branch the signs of this paralysis are obscured by that of the other muscles of the cord. The recurrent laryngeal nerves supply all the other muscles. In any progressive lesion of the nerve-path the muscles become paralysed in a definite order, the enunciation of which is known as Semon's law; the abductors are first affected, then the tensors or thyro-arytenoidei, and finally the adductors.

In *abductor paralysis* the affected cord lies in the middle line; during phonation the sound cord adducts to meet it and the larynx appears normal, but on inspiration it is drawn outwards and backwards and appears longer than its paralysed fellow, which remains unmoved. As would be expected from the course of the recurrent nerves, the left cord is far more often affected than the right. The voice is unaltered, but, although the glottic aperture is reduced by half, there is usually dyspnoea only on exertion, except in children, in whom the narrowing of the naturally small glottis may produce sufficient obstruction to necessitate a tracheotomy.

When the thyro-arytenoid fails, the edge of the cord is concave on phonation, the cord appears narrower than its fellow, and the voice gradually becomes husky. Finally, when *total recurrent paralysis* has occurred, the cord assumes the "cadaveric position" between the middle line and the normal position of rest. On phonation, the healthy arytenoid crosses the middle line and pushes the paralysed cartilage aside; sometimes the latter drops forward and exposes its broad posterior surface, which may be mistaken for a swelling. As the cords are still able to approximate, the voice is not necessarily lost, but is hoarse and easily tired, with a characteristic breathy quality from waste of air, or a diphonic character, due to unequal vibration of the two cords.

In cases of *bilateral abductor paralysis* the cords lie together near the middle line. The voice is good, but the inability to take a full breath gives the speech a peculiar character; dyspnoea is a marked symptom accompanied by inspiratory stridor and severe paroxysmal exacerbations. When the disease progresses to complete *bilateral recurrent paralysis*, both cords remain in the cadaveric position, the dyspnoea becomes less severe but the voice is reduced to a whisper.

Diagnosis.—The diagnosis is almost entirely a matter of accurate

inspection. Obliquity of the laryngoscopic image, due to faulty position of the mirror, may cause confusion. In nervous subjects the cords are sometimes adducted on inspiration, but they will abduct naturally during the involuntary inspiration which follows a prolonged phonation. The only condition which really imitates paralysis is the fixation of the arytenoid cartilage which results from disease in or around the joint; its complete immobility with the presence of swelling or scarring often aids the diagnosis, but in old-standing cases of paralysis secondary fixation frequently occurs.

Ætiology.—The ætiology is of importance, for it is on our know-

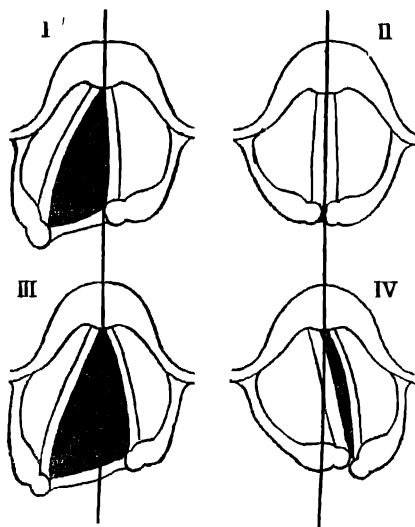


FIG. 97.—Organic Paralysis: I. Abductor paralysis of left cord on inspiration; II. Abductor paralysis of left cord on phonation; III. Total paralysis of left cord on inspiration; IV. Total paralysis of left cord on phonation. (*Lancet*.)

ledge of their causation that the diagnostic value of these lesions depends. The movements of the cords are represented bilaterally in the cortex cerebri, and stimulation of either centre produces movement (adduction) of both cords, from which it follows that no unilateral lesion above the bulbar nuclei can paralyse the larynx, and clinically we find that it is never affected in cases of hemiplegia. The bulbar centres lie in the floor of the fourth ventricle, and here a lesion of one centre causes paralysis of the cord on the same side which, in a gradually progressive lesion, affects first the abductor muscle. Thence the nerve fibres pass in the roots of the bulbar-accessory to the vagus and recurrent laryngeal nerve; the cause of the paralysis may, therefore, be situated (1) in the medulla, (2) at the base of the skull, (3) in the vagus, or (4) in the recurrent laryngeal nerve.

Paralyses of bulbar origin are often, but by no means always, bilateral. In lesions here and at the base of the skull neighbouring nerves are liable to be involved; thus, paralysis of a cord and of the same side of the palate may coexist (syndrome of Avellis), or paralysis of cord, palate, trapezius and sternomastoid from involvement of the spinal accessory roots, or persistent frequency of the pulse due to damage of the cardio-inhibitory centre or nerves. *Tabes dorsalis* is the most frequent cause of paralysis of central origin; it may affect one or both cords and may be associated with anæsthesia, paræsthesia or the spasmodic attacks called "laryngeal crises." In general paralysis of the insane laryngeal palsy is not uncommon. It is the rule in bulbar paralysis, and is usually bilateral, but appears late in the disease.

Syphilitic nuclear disease, pachymeningitis and gummata at the base of the brain are frequent causes, and here the ocular muscles, especially the external rectus, are often attacked.

Peripheral causes usually act by compression of the recurrent nerve, the most frequent being aneurysm, enlarged glands, tuberculous or malignant, and cancer of the œsophagus. Other causes are thyroid tumours, usually but not necessarily malignant, mediastinal tumours, cancer of the lung, pleurisy, and pulmonary tuberculosis in which the nerve, usually the right, may be involved in a lesion at the apex of the lung or by tuberculous bronchial or tracheal glands. Neuritis is a cause of laryngeal paralysis; it may be produced by the toxins of the infectious fevers, usually diphtheria, or by organic poisons, especially lead, and more rarely arsenic and alcohol. Finally, the condition is sometimes the result of traumatism, more especially surgical operations on the thyroid gland and œsophagus.

Prognosis.—Paralysis of one cord is not in itself dangerous to life; but when the cause is undiscovered the prognosis must be guarded, for this paralysis may be for a long time the only sign of serious disease; on the other hand, the recurrent nerve may be involved in some non-progressive lesion, such as a fibrotic bronchial gland, and such cases have been under observation for 20 or 30 years without change.

Treatment.—This depends on the cause. In most cases it is but a symptom of disease elsewhere and does not call for special treatment. In traumatic cases, however, the nerve may sometimes be found and sutured; afterwards, and in cases due to neuritis, strychnine and the local application of the faradic current by means of an intra-laryngeal electrode are indicated. Tracheotomy is advisable in bilateral abductor paralysis, but a plug may usually be worn in the tube, to be removed at night and whenever dyspnoea threatens.

FUNCTIONAL PARALYSIS (FUNCTIONAL APHONIA)

Ætiology.—Functional aphonia is a common manifestation of hysteria, and has been a very frequent symptom of war-neurosis or "shell-shock," but it should be clearly stated that the majority of cases are not purely hysterical. Anything which increases the effort of phonation, such as debility or laryngeal catarrh, predisposes to this affection, which is characteristic rather of feeble neuro-muscular tone than of hysteria; this explains how some women lose the voice completely with every slight cold, while other patients can produce a loud if hoarse voice with severe laryngeal disease.

Symptoms.—Paralysis of the adductors presents a totally different clinical picture from the organic paralysis. It is always bilateral; the larynx appears normal while at rest, but, on attempts at phonation, it is seen that the cords do not adduct into the position necessary for the production of the voice. Very commonly the thyro-arytenoidei are the only muscles which fail to act; the cartilaginous glottis is then properly closed, but an elliptical chink is left between the cords. If the crico-arytenoidei laterales are paretic, the entire glottis remains open to a variable extent, and, very rarely, the arytenoideus is affected alone, when a triangular aperture is left behind the vocal processes. The paralysis is

hardly ever complete; indeed a considerable amount of movement is usually seen, though insufficient to produce phonation. In purely hysterical cases the onset and recovery are sudden, the cough is usually not aphonic and the voice when regained is not hoarse. In some hysterical patients there is also inability to whisper.

Treatment.—In patients suffering from debility the cause should be

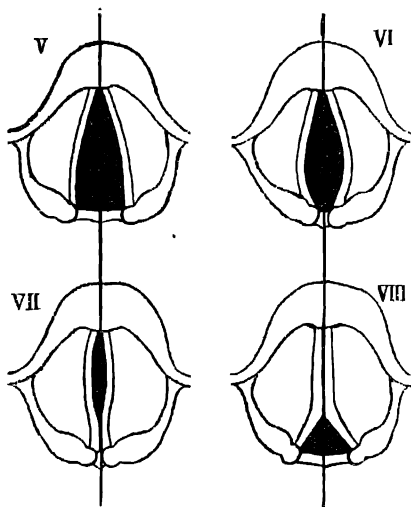


FIG. 98.—Functional Paralysis; all during attempted phonation; V. Paresis of all the adductors; VI. Arytenoides still active; VII. Paresis of internal tensors; VIII. Paresis of arytenoides. (*Lancet*.)

is no serious disease, but that he is not using his muscles correctly, and that he can produce a good voice quite easily when the laryngeal mirror or tongue-depressor is in position. With a little elementary instruction in voice-production this is usually successful, the patient's confidence is restored and the voice does not again fail; in obstinate cases some perseverance in lessons on production is required. These methods have been extraordinarily successful with shell-shocked soldiers, but it must be confessed that such certain and rapid results are not always obtainable in nervous women.

SPASMODIC AFFECTIONS

SPASM OF THE GLOTTIS

Spasm of the laryngeal muscles produces adduction of the cords, for, though the abductors are probably affected, they are overpowered by the stronger adductor muscles.

found and treated; chronic phthisis is such a common cause of functional aphonia that it should always be thought of in this connection. There is frequently a slight degree of laryngitis and in some of these cases the failure of adduction is "myopathic," or due to inflammation of the muscles; in these the local condition must receive appropriate treatment. When the larynx is normal the voice can nearly always be temporarily restored by any powerful local stimulation, such as the intralaryngeal application of chloride of zinc, or any similar drug, or of the faradic current; but the aphonia usually recurs again, and succeeding applications are less effective, so that the most difficult cases to cure are those who have had much local treatment. Far better results are obtained by moral suasion, explaining to the patient that there

Ætiology.—(1) In the majority of cases the spasm is a reflex set up by local irritation : foreign bodies, including the laryngoscopic mirror, irritating gases ; inflammation, ulceration or tumours in or near the larynx, children being much more liable than adults to spasm from local irritation. (2) Spasm is also caused by irritation of the recurrent laryngeal nerves by enlarged glands, mediastinal tumours and, especially, by aneurysm. (3) Central nervous lesions, especially tabes. (4) Functional disturbances, frequently hysterical, often associated with globus hystericus, and sometimes excited by sexual disturbances.

Symptoms.—The attacks vary much in different subjects in severity and duration. The patient is usually aware of its onset, and clutches some support or rushes to the window. The respirations are rapid and shallow, with loud inspiratory stridor, and, in the height of a severe attack, are completely arrested with all the signs of asphyxia. The subjective sensations include a horrible feeling of anxiety, but consciousness is not lost. Many cases are less acute but persist longer, even for several hours.

Prognosis.—The attacks are practically never fatal, unless a foreign body or tumour be present.

Treatment.—During the attack amyl nitrite or chloroform may be inhaled, and ampoules of these drugs should be kept on hand. Between the attacks sources of irritation should be sought for and removed, the upper air-passages brought to a healthy condition, and the general health and mode of life should receive attention. Administration of bromides may be required when the attacks recur frequently.

LARYNGISMUS STRIDULUS

Ætiology.—This is a condition, clinically similar to glottic spasm, occurring in children. It is far commoner than the spasm of adults, and it has been suggested that the asphyxial attacks of laryngismus are caused by collapse of the soft and yielding cartilaginous framework of the larynx, and not solely by spasm of the muscles. It is commonest between the ages of 6 months and 2 years, but may persist later ; it occurs in ill-nourished, unhealthy children, usually in association with rickets, and practically always in association with adenoids.

Symptoms.—The onset is sudden and usually at night. The child wakes gasping for breath, and a series of short noisy inspirations is followed by complete cessation of breathing and terminated by a long, crowing inspiration. There are retraction of the lower ribs and epigastrium, cyanosis and great terror and distress and, in severe cases, carpo-pedal contractions, convulsions and evacuation of urine and fæces. When the attack is over the child is perfectly normal and there is no hoarseness. Slighter and less typical attacks often occur.

Diagnosis.—This is easy if the symptoms are carefully noted ; the sudden attack of dyspnoea, with complete absence of symptoms in the intervals, is quite distinctive.

Prognosis.—The prognosis is somewhat grave in severe cases ; an infant rarely dies in an attack, but is often worn out and succumbs to collapse of the lungs.

Treatment.—During the attacks the face and chest may be freely

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sponged with cold water, and the inhalation of amyl nitrite from a capsule broken in a handkerchief may be tried. The quickest relief can usually be obtained by drawing the tongue forward with a finger passed into the mouth to its base, a manœuvre easily performed by the mother or nurse. The attacks are so short and sharp that there is no time for the hot bath or administration of bromides frequently recommended.

Prevention involves general tonic treatment, fresh air, wholesome food and correction of digestive disturbances. The removal of adenoids is of great importance, even if not large enough to be definitely obstructive, as is the treatment of naso-pharyngeal catarrh with the usual saline lotion which, in small children, may be dropped into the nostrils from a pipette. Bromides are to be avoided if possible as depressing, but 10 to 30 drops of liquid extract of *grindelia* may be given 3 or 4 times a day in milk or sweetened water as recommended by Eustace Smith.

CICATRICAL STENOSIS

Ætiology.—Suicidal and other wounds, gunshot injuries and scalds may produce cicatricial narrowing of the lumen of the glottis. After thyrotomy a web may form across the anterior commissure. In the haste of an emergency tracheotomy, the wound has often been made too high and the cricoid cartilage cut through; in these cases it generally happens that, after the subsidence of the acute condition, dyspnœa follows every attempt to remove the tube, and a stenosis is found to have resulted from swelling and narrowing in the subglottic region. Similarly, a proportion of cases intubated for diphtheria are unable to breathe without the tube, by reason of a subglottic stenosis. Lupus and tuberculosis of the larynx can produce cicatricial stenosis, and especially when the cautery has been extensively employed in treatment. Syphilis is the most fruitful cause of this condition, and the great difficulty of obtaining a cure at this stage is a powerful reason for early and thorough treatment of syphilis of the larynx. Leprosy and scleroma cause stenosis, but are rarely seen in this country. The perichondritis which is an occasional complication of enteric fever, small-pox and diphtheria commonly ends in severe stenosis.

Symptoms and Diagnosis.—The principal symptom is naturally dyspnœa, but in chronic cases it is remarkable how great may be the narrowing before dyspnœa becomes severe. The obstruction and the stridor are most marked on inspiration, in contra-distinction to tracheal stenosis where the stridor is both inspiratory and expiratory. The larynx moves downwards with each inspiration; this "respiratory excursion" of the larynx is a further diagnostic sign of laryngeal obstruction, but is not always present. The patient sits upright, with the head thrown back; whereas in tracheal obstruction he bends the neck forward to relax the trachea.

Treatment.—In all cases with decided dyspnœa tracheotomy should be first performed, and in syphilitic cases it is important that all active disease should be arrested by thorough treatment before any attempt at dilatation be begun, and the stenosis itself will often be greatly improved by such treatment. The administration of iodides is dangerous in these cases, for the increased secretion is pent up behind the stenosis and may overwhelm

the lungs. Difficulty in dispensing with the tube after tracheotomy is sometimes due to nervousness on the part of the child, and can then be surmounted by using a fenestrated tube which is plugged occasionally and by encouraging the patient to breathe through the mouth by blowing soap-bubbles or sounding a whistle. When the tracheotomy wound is too high, a low tracheotomy should be performed and the original wound allowed to close; this is often sufficient to overcome the difficulty.

The successful treatment of severe cicatricial stenosis demands the greatest skill and perseverance on the part of the surgeon as well as the patient co-operation of the sufferer. The whole circumstances of the case should be carefully considered before advising difficult and prolonged treatment. Adult patients can live active lives with a permanent tracheotomy opening, which is not so serious a disability as it is generally considered to be. If the stenosis be not too extreme, a fenestrated tube may be worn which can be kept plugged during the day, so that the patient may have the use of speech and respiration by the natural passages. In children and young people a permanent tracheotomy is more harmful, but the prospect of cure by dilatation is better; the best method of dilatation is by the use of intubation tubes.

HAROLD S. BARWELL.

DISEASES OF THE TRACHEA

INFLAMMATION OR TRACHEITIS

ACUTE TRACHEITIS

Acute tracheitis may occur from any condition leading to irritation of the mucous membrane of the trachea. When it occurs as a result of bacterial or chemical agency, the whole of the upper air-passages are usually involved in greater or less degree, and the clinical manifestations are not confined to the trachea. In some cases, however, the stress of the resultant reaction falls upon this tube, and the condition therefore requires separate consideration.

Ætiology.—1. *Microbic invasion.*—This is the commonest cause. The bacteria usually found associated with tracheitis are the so-called catarrhal organisms, such as the *Micrococcus catarrhalis*, the pneumococcus, the Freidländer pneumo-bacillus and Pfeiffer's *H. influenzae*. It is probable that the primary organism in many cases is of filter-passing type. Frequently a member of the streptococcus group may be found, either alone or in association with one or more of those just mentioned. As with catarrhal inflammation of other parts of the upper air-passages, damp, cold or foggy climatic conditions predispose to tracheitis. It is more common in young and middle-aged adults than in infancy or in old age. Mouth-breathers are more liable to this condition. Exposure to sudden changes of temperature may be a factor in its onset.

Tracheitis may also occur as part of the clinical picture in some of the acute specific diseases, such as enteric fever, diphtheria, whooping-cough

and measles. It is often a troublesome and distressing association or sequel of true influenza.

2. *Chemical agencies.*—Irritating or poisonous fumes and vapours may lead to a very acute form of tracheitis. It may, therefore, occur in certain occupations, unless adequate precautions are taken. The use of "poison gases" in warfare has drawn widespread attention to this form of the condition, since tracheitis was an almost constant result of certain forms of "gassing." The chief chemical irritants used in the War of 1914-1918 were chlorine, phosgene and yperite, or dichloroethyl sulphide, commonly known as yellow cross or mustard gas. Of these the last was perhaps the most irritant to the trachea, and fatal cases invariably showed tracheal lesions. Direct inhalation of steam may also induce an acute tracheitis.

3. *Mechanical causes.*—The presence of a foreign body, or the invasion of the trachea by extension from malignant growth in adjacent structures may lead to a local or even to a general tracheitis. It is noteworthy, however, that the trachea is frequently spared in occupations involving the respiration of dusty air, which leads to deposits in the lungs and bronchial glands with resulting pneumonokonioses. Although a coal miner's lungs are black, yet his trachea may be practically normal.

Pathology.—The changes found in the trachea vary from simple catarrhal inflammation to intense destructive changes with ulceration, and in some cases croupous or membranous exudate. In the catarrhal forms, the mucous membrane shows changes similar to those in bronchitis. It is at first swollen, red and dry, the vessels running across the trachea being engorged and clearly visible. Then, owing to increased activity of the mucous glands, excessive mucoid secretion occurs and the mucous membrane becomes moist, after which resolution may take place, or the process may proceed to a mucopurulent stage, when the fluid on the membrane coheres to form yellowish or green tenacious pellets. Occasionally numerous red blood cells are extruded and the tracheal exudate becomes streaked, tinged or uniformly pinkish.

In some inflammations, such as those induced by poison gases or inhaled steam, the mucous membrane may be intensely engorged and actual destruction may occur, involving even the deeper structures and the cartilages, so that greyish yellow sloughs result, which on separation leave ulcers. In diphtheria the characteristic false membrane composed of necrosing fibrin, leucocytes and bacilli may be found loosely attached to the mucous membrane, as in other localisations of this process. It may be primary or secondary to faucial or laryngeal diphtheria, either by direct extension or through diphtheritic infection of a tracheotomy wound.

In influenza the pink appearance of the trachea is of such constancy in fatal cases that it has come to be regarded as one of the most characteristic post-mortem changes found in this disease. The bright injection generally involves the lower half of the trachea, but it may occur along the whole length of this tube.

In whooping-cough the inflammatory reaction is usually less acute.

In typhoid fever small ulcers may occasionally be found in the trachea similar to those occurring more commonly in the larynx.

Symptoms.—Acute catarrhal tracheitis usually begins more or less acutely, like the common "cold," of which it is to be regarded as one form, with malaise, slight headache, and a mild degree of fever, the temperature

being usually between 99° and 100° F., rarely 101° F. The patient soon experiences a sensation of irritation behind the sternum, rapidly leading to a harsh, dry cough of noisy character. The cough aggravates the retro-sternal discomfort, which develops into a sensation of rawness or soreness, making the cough very painful and distressing. If the larynx is involved at the same time, the voice becomes hoarse and sometimes lost, or reduced to a raucous whisper. In tracheitis alone the voice is usually unaffected. After from 12 to 24 hours the condition passes into the mucoid stage. The cough becomes looser and less painful, and small pellets of tenacious mucus are coughed up, usually greyish or black in town-dwellers, whitish in those in rural conditions; in either case, the mucus may be streaked with blood or even tinged a uniform pink colour; in the more acute forms it sometimes becomes yellow and more purulent. In the mucoid stage, the retrosternal soreness becomes less, the constitutional symptoms abate, while the temperature subsides and becomes subnormal. The patient often feels weak and out of health for some days, and is sometimes left with a noisy morning cough and tracheal irritation which may last for days or weeks. The aspect of the patient shows nothing characteristic. There is the general appearance of fever, malaise and discomfort. The rise of temperature and increase in pulse-rate are usually moderate. In the early stages physical examination of the chest shows no abnormality, but when exudation occurs a coarse wheeze may be audible over the trachea, particularly when the patient takes a deep breath or just before a cough occurs.

Diagnosis.—The association of catarrhal symptoms with a dry, harsh cough and retrosternal soreness, without signs of bronchitis, is almost pathognomonic. In some cases the diagnosis can be established with the laryngoscope or by endoscopy, but in most the discomfort which these examinations entail is unnecessary.

Prognosis.—This is almost invariably good, except in debilitated subjects or in those with cardiac or renal disease, in whom the process may spread to the larynx, bronchi or lungs. The usual course is from 2 days to a week, though cough and expectoration may persist for days or weeks. The condition may become chronic. To some extent the prognosis depends upon the care and treatment in the initial stage. Cases that are neglected are liable to become chronic.

Treatment.—The prophylactic and remedial treatment of acute tracheitis is practically identical with that of acute bronchitis of the larger tubes. Even in mild cases the patient should go to bed, though this may be necessary only for 1 or 2 days; but he should keep to his room till his temperature has become normal. There may be less need for expectorants than in bronchitis, and a simple saline diaphoretic mixture, with the addition later of tinct. ipecac. and tinct. opii camphorata, may be all that is necessary. Sedative inhalations, such as vapor benzoini, are useful, and counter-irritation to the sternal region is comforting and grateful to the patient. When a chronic noisy cough develops, a mixture containing small doses of apomorphine and tinct. chloroform. et morphin. co. often gives relief, or codeine linctus B.P.C.

When tracheitis occurs as part of some specific disease, such as diphtheria or influenza, the treatment should be that appropriate to the primary disease.

In "gassing," every effort should be made to relieve the distressing and

painful symptoms, and for this purpose morphine, either alone or in combination with atropine and strychnine, may be required. Various inhalations may be tried, and useless cough should be checked by sedative mixtures or by a linctus of heroin, morphine or codeine.

CHRONIC TRACHEITIS

Ætiology.—Chronic tracheitis may follow an acute attack, or it may develop insidiously in patients suffering from chronic laryngitis or bronchitis. Inhalation of cigarette smoke is a not infrequent cause. It is also sometimes a sequel of chronic rhinitis, especially of the atrophic form or *ozæna*. A certain degree of chronic tracheitis accompanies the specific lesions of syphilis and tuberculosis, which are described below.

Pathology.—Various degrees of chronic inflammatory lesions may be found. In chronic catarrhal tracheitis, the vessels are distended or engorged, and the mucous membrane of the trachea becomes thickened and more or less covered with mucoid or muco-purulent secretion, the histological changes being those of chronic catarrhal inflammation, namely, shedding of the ciliated epithelial cells, overactivity of the mucous glands, and sometimes thickening and induration of the submucous tissues from proliferative changes. A condition of perichondritis of the tracheal cartilages may, in this case, be observed, and this may result in a mammillated appearance in the internal aspect of the trachea. In *ozæna*, crusts similar to those in the nose and pharynx may form on the tracheal mucosa.

Symptoms.—The symptoms of chronic tracheitis are similar to those of the acute form. There is a sense of discomfort and irritation about the trachea and a chronic, almost dry cough, often worse in the morning. There is generally some scanty, sticky expectoration, mucoid or muco-purulent, darkened by carbon particles and occasionally blood-tinged.

There are practically no physical signs of this condition, except that the tracheal changes can be observed by the laryngoscope or by endoscopy of the trachea.

Diagnosis.—This is concerned chiefly with its differentiation from chronic changes in the trachea due to syphilis, tuberculosis or leprosy and to the effects of new growths. It must largely be made by endoscopic examination.

Prognosis.—The prognosis depends upon the cause. When this can be removed, as by treatment of predisposing conditions in the nose and throat, the outlook is good. When the tracheitis is due to other conditions, such as syphilis and tuberculosis, it depends upon the situation and extent of the other lesions and upon the treatment adopted.

Treatment.—This is, in its main features, similar to that of acute tracheitis, but climatic treatment may be of great importance. The patient may perhaps spend the winter months in a warm or equable and clear climate with great advantage. Vaccine treatment may also yield good results. When other conditions are concerned, such as *ozæna*, syphilis or tuberculosis, the treatment appropriate to them should be employed as well.

CYSTS AND TUMOURS

These are rare conditions, but require careful consideration.

CYSTS

Owing to weakening of the wall of the trachea, local bulging may occur, giving rise to a cystic, air-containing swelling in the neck, in direct communication with the lumen of the trachea. Such cysts are known as "tracheoceles" or "aeroceles." They are resonant to percussion and can often be temporarily reduced by pressure.

Small retention cysts may occur in the posterior wall of the trachea, from obstruction of the ducts of the mucous glands as they pass through the trachealis muscle. They are of pathological interest only, and do not give rise to symptoms.

SIMPLE TUMOURS

The most important is papilloma. It occurs chiefly in children and is usually pedunculated. When it grows in polypoid form it may lead to obstruction of the trachea low down, in which case tracheotomy may fail to give relief, and death results unless the tumour can be removed by endoscopic methods.

Other innocent tumours occur, but are rare. They include enchondrosis from localised overgrowth of cartilage, multiple enchondromata, and osteoma from ossification of a pre-existing enchondroma. Lipoma and aberrant thyroid tumours may occur, but are very rare.

Symptoms.—These tumours produce varying degrees of tracheal obstruction, and can usually only be recognised by endoscopy. Treatment is considered under that of tracheal obstruction.

MALIGNANT TUMOURS

A few cases of primary carcinoma of the trachea have been recorded. Secondary growths are not common, but the trachea is often involved and infiltrated by primary carcinoma in adjacent structures, such as the oesophagus, the thyroid, the larynx, or by the extension of secondary deposits in the cervical or mediastinal glands.

Primary sarcoma of the trachea is also very rare. The growth is usually smooth and not pedunculated. Secondary deposits of sarcoma in the trachea may occur from sarcoma of distant organs, such as the kidney; or it may be invaded directly by sarcoma originating in the thymus or other mediastinal structures, and especially by lympho-sarcoma of the mediastinal glands.

Symptoms.—The tracheal symptoms and signs are usually those of obstruction, accompanied by pain. When the primary growth is in the oesophagus, antecedent dysphagia and sometimes laryngeal paralysis reveal the origin of the tracheal symptoms when they occur. In this case copious frothy mucoid expectoration is frequent, and when ulceration develops with perforation, food particles may enter the trachea, excite cough and soon lead to inhalation broncho-pneumonia or gangrene. When the growth is near the bifurcation, urgent dyspnoea is the rule, and spasmodic attacks may

occur, causing extreme distress. In most cases of tracheal growth the characteristic clanging brassy cough (gander cough) of tracheal obstruction can be heard. The trachea may be pushed to one side and its lumen distorted and obstructed by growth in the cervical glands. In mediastinal new-growth invading the trachea, the pressure signs and symptoms characteristic of that disease usually render the explanation of the tracheal symptoms apparent.

Course.—This is generally rapidly progressive.

Diagnosis.—Intratracheal growths have to be differentiated from other causes of tracheal obstruction, and the diagnosis is considered in detail under that condition. Endoscopy affords valuable confirmation if it is practicable or desirable. In œsophageal and mediastinal new-growths invading the trachea, X-ray examination may assist in diagnosis.

Prognosis.—This is hopeless, death occurring from asphyxia or from some complication or by asthenia.

Treatment.—Treatment can be palliative and symptomatic only. In obstruction, it may be possible in rare cases to give temporary relief by a low tracheotomy, but as a rule this is impossible, owing to the presence of obstruction below any point where the trachea is accessible.

THE INFECTIVE GRANULOMATA

SYPHILIS

. The trachea may be affected in both the congenital and acquired forms.

In congenital syphilis, a progressive cicatrisation may occur, leading to stenosis. In acquired syphilis, during the secondary stage, the mucous membrane of the trachea may become generally hyperæmic, or small raised mucous patches may develop locally. In the tertiary period, gummata may occur in the trachea, the commonest site being towards the lower end. Degenerative processes, leading to necrosis and softening, eventually result in ulceration, sometimes with local sloughing of parts of the tracheal rings. In the process of cicatrisation a progressive stenosis may develop.

Symptoms.—Symptoms are those of chronic tracheitis and tracheal irritation in both the secondary and tertiary manifestations, but in the latter, signs of tracheal stenosis may develop when scarring and healing are in progress. Laryngeal involvement occurring at the same time tends to distract attention from the tracheal lesions or to obscure them.

Diagnosis.—The diagnosis of syphilis of the trachea depends upon a careful study of the history of the case, indications of tracheal irritation, laryngoscopic or endoscopic examination, the coexistence of other manifestations of syphilis, and in their absence, a positive Wassermann reaction.

Prognosis.—If the condition is recognised early, excellent results may be obtained by treatment, but it is obvious that where deep destructive changes have resulted, medicinal measures can only palliate.

Treatment.—Anti-syphilitic treatment should be administered vigorously, and after a course of neoarsphenamine, mercury or bismuth preparations should be given. Inunction seems sometimes of special value in such cases. In cases of stenosis of the trachea from cicatrisation, dilatation of the stricture by means of bougies introduced through an endoscope may be practicable and afford useful help.

TUBERCULOSIS

Tuberculosis of the trachea is occasionally found post mortem in advanced cases of pulmonary tuberculosis, usually in those with extensive laryngeal involvement. Primary tracheal tuberculosis is unknown. The rarity even of secondary lesions in this tube is probably to be explained by the ciliated epithelium preventing lodgment of the bacilli.

Pathology.—Tuberculous lesions may occur at any part of the trachea, but they are more frequent in the lower part and on the posterior wall. When they occur they are usually numerous. There may be some general hyperæmia, or small tubercles, varying in size from a pin's head to a split pea, may be visible. Later, superficial ulceration occurs, forming irregular punched-out ulcers. Occasionally, the process may extend deeper, and erosion of the cartilages may occur, with the formation of sinuses and even fistulous communication with the œsophagus.

Symptoms.—Since tracheal tuberculosis is usually a late manifestation of advanced disease, its clinical indications are slight and are usually obscured by the more obvious laryngeal and pulmonary symptoms and signs, though if the process extends deeply and produces sinuses and fistulous tracks, it may become apparent. The actual tracheal symptoms are those of cough and retrosternal soreness.

Diagnosis.—This condition has to be distinguished from other chronic tracheal lesions, and a diagnosis can only be made from a careful review of the history, the general evidence of tuberculous disease and by the tracheal involvement which may be visible by endoscopy.

Treatment.—This must, from the nature of things, be largely palliative, and is in effect practically identical with that of laryngeal tuberculosis, notably intratracheal insufflation with orthoform and benzocaine (anæsthesine).

LEPROSY

In some cases of this disease, granulomatous lesions occur in the trachea, and these may eventually give rise to tracheal stenosis, owing to the contraction of new-formed fibrous tissue. The diagnosis can only be made from the occurrence of tracheal symptoms in a case with established lesions of leprosy in other parts.

The treatment is symptomatic.

SCLEROMA

Although in most cases this condition affects the nose only, scleromatous lesions may be found in the trachea as a pathological curiosity. The disease in any form is rare in England, and occurs chiefly in Poland and Austria. The nodules of granulomatous tissue in the trachea may cause partial obstruction mechanically, or, on contraction, lead to actual stenosis.

TRACHEAL OBSTRUCTION

Obstruction to the lumen of the trachea may be produced by foreign bodies, by conditions originating in the trachea, and by pressure from without.

FOREIGN BODIES IN THE TRACHEA

The commonest route by which foreign bodies enter the trachea is through the mouth and larynx, in the acts of breathing, laughing, yawning, sighing, or before and after coughing, when food or some foreign substance is in the mouth. A piece of bone, a stud, button, false teeth, chewing gum, peas, articles of food, nuts, grains of wheat, beads or blades of grass are among the substances which may gain entrance to the trachea in this manner. Surgical operations in the mouth and throat may lead to the inhalation of a tooth, a piece of tonsil or a mass of adenoid tissue. Material vomited from the stomach, such as food, blood clot or intestinal worms, may be inhaled into the trachea. A large blood clot in hæmoptysis may temporarily obstruct it. Foreign bodies may also gain access through the tracheal wall, such as small projectiles in wounds of the neck, a piece of new growth, or tuberculous glands by ulceration through the wall.

Unless it becomes impacted, or is too large to enter one of the two main bronchi, a foreign body rarely remains long in the trachea. It either causes death with dramatic rapidity, is coughed out again, or passes down into one or other of the large bronchi or their secondary divisions, where it produces results which are described in the section on diseases of the bronchi.

Symptoms.—These depend upon the mode of entry, the size of the foreign body, and the degree of obstruction to the air current which it induces, but in general the tracheal symptoms are less urgent than those of laryngeal obstruction, and less serious than those of obstruction of one or other main bronchus. There may be intense dyspnoea, with great discomfort and alarm during the actual passage through the larynx of a small foreign body, especially if it is temporarily arrested there; but when it enters the trachea there is an almost instantaneous cessation of the acute distress, though some degree of dyspnoea may persist. The type of dyspnoea is inspiratory in the main, though a minor degree of expiratory difficulty may be apparent if the foreign body is of considerable size. There may be a definite stridor with both phases of respiration, but it is more pronounced in inspiration. If the foreign body remains loose in the trachea, which may occur if it is rounded and too large to engage in one of the main bronchial divisions, a sound of vibratory character may be heard on auscultation of the trachea, sometimes described as the *bruit de grelottement*. This may be produced by friction of the foreign body against the tracheal wall, or more commonly by the air passing over it during respiration. A paroxysmal cough may occur, caused by the foreign body irritating the sensitive posterior wall of the trachea, and during such an attack the foreign body may be forced up to the larynx, obstruct it, or cause reflex spasm with intense dyspnoea and cyanosis and a risk of suffocation, unless it drops back, is coughed out, or removed. When sudden rupture of caseous material into the trachea occurs, the lumen may be blocked and death take place rapidly.

Course.—A foreign body impacted in the trachea may give rise to septic inflammation of its walls, with subsequent cicatrisation after removal, or it may lead to secondary infective processes in the lungs, such as purulent bronchitis and broncho-pneumonia.

Diagnosis.—The history of disappearance of some object from the mouth during coughing, breathing or laughing should give rise to suspicion of an

inhaled foreign body, and this may be confirmed by seeing the object directly by endoscopy, or indirectly by means of the X-rays.

Prognosis.—This depends in the main on the nature of the foreign body, and the time elapsing before its removal. An irregular, rough or soft foreign body is more likely to induce septic complications than a smooth, hard substance. Apart from rapidly fatal results, the prognosis is better with intratracheal foreign bodies than with those reaching the bronchi. If removal is effected within 24 to 36 hours, recovery is usually rapid and complete.

Treatment.—Treatment consists in rapid removal with as little damage to the trachea and larynx as possible. This may be effected by means of forceps passed through a bronchoscope, or rarely by tracheotomy alone, when the foreign body may be coughed out through the opening or be easily removed by forceps. Inversion of the patient in the hope that gravity may assist the expiratory efforts of cough is dangerous and should only be attempted after tracheotomy has been performed. Where rupture of a caseous gland or softening new-growth occurs into the trachea, an immediate tracheotomy may be necessary.

OBSTRUCTION FROM CICATRISATION OF THE TRACHEAL WALLS

Ætiology.—This may result from any condition leading to ulceration of the tracheal walls, with subsequent healing, such as a syphilitic gumma, or less commonly other granulomata, such as tubercle, leprosy or scleroma. Another cause is cicatrization from wounds of the trachea, accidental, suicidal or after tracheotomy, when the incision has been made too near the cricoid, or when the wound has become infected or the tube left in too long. Scarring from damage to the trachea by the inhalation of boiling or caustic liquids or even by inhaled gases may lead to stenosis.

Pathology.—The deformity of the trachea and the obstruction of its lumen depend upon the situation and the extent of the cicatricial contraction of its walls. It may be local, producing an hour-glass constriction, or involve a long extent of the tube. Occasionally, especially in syphilitic lesions, stenosis may occur at two different levels.

Symptoms.—These depend upon the degree of stenosis, the rapidity with which it develops, and the condition of the larynx, bronchi and lungs. When the stenosis is produced gradually, as in cicatrization, a degree of obstruction may result, greater than would be compatible with life if suddenly induced. In the early stages of a progressive stenosis, slight dyspnoea may be present on exertion, and during sleep a faint stridor may be audible, disappearing when the patient is awake. As the contraction progresses, the dyspnoea becomes more marked, and a definite and persistent stridor develops, at first inspiratory only, though expiration may become both noisy and obstructed. The patient may experience a sensation of obstruction referred to the neck or under the sternum, accompanied by pain and irritation, leading to cough, which may be dry, noisy and metallic, or accompanied by more or less frothy sputum, if the primary condition is associated with widespread tracheitis. The voice may lose tone and volume, and the patient talk more quietly than normal and with some evident effort. In advancing stenosis, sudden and alarming attacks of dyspnoea may occur,

leading to cyanosis and threatening suffocation. These attacks are usually due to an accumulation of mucus at the site of the stenosis. The patient in advancing degrees of obstruction cannot lie down, and generally sits leaning forward with chin depressed. It may be noted that the extraordinary muscles of respiration contract forcibly, and yet the laryngeal excursions may be small or hardly noticeable, in contrast with those of laryngeal obstruction in which they are maximal. This distinguishing sign was first pointed out by Gerhardt, and is of value, but unfortunately it is not absolute and cannot, therefore, be regarded as pathognomonic. On auscultation over the trachea, a noisy roar may be audible, of maximum intensity near to the obstruction, whereas the breath-sounds over both lungs may be deficient, although the stridor may be conducted bilaterally.

Course.—The course of cicatricial stenosis is usually progressive, unless arrested by treatment, and the dyspnoeic attacks become more frequent and alarming.

Diagnosis.—Tracheal obstruction from cicatrization has to be distinguished from laryngeal obstruction, in which the symptoms are usually more acute and more urgent. Gerhardt's sign described above may also be suggestive. It has also to be differentiated from obstruction due to pressure from without (*vide infra*). The only reliable method of distinction is by direct inspection with the bronchoscope.

Prognosis.—Early syphilitic stenosis may be arrested by appropriate anti-syphilitic treatment. Obstruction due to other granulomatous conditions varies with the severity and extent of the primary lesions. Caseous material or degenerated growth ulcerating into the trachea is usually immediately fatal, or leads to death within a few days from pulmonary complications.

Treatment.—Rest, avoidance of exertion, smoking and alcohol should be advised. The patient's fears should be allayed and symptomatic treatment ordered, such as sedative inhalations or a linctus to check useless cough. In syphilitic stenosis vigorous anti-syphilitic treatment with neoarsphenamine,¹ mercury or bismuth preparations should be given. A low tracheotomy may be necessary for an intractable stricture high up in the trachea. In some cases where an ordinary tracheotomy cannot be performed below the stricture, it may be possible to insert Koenig's long tracheotomy tube through an opening in the trachea made above it. In other cases, dilatation of a fibrous stricture by bougies passed through an endoscope may be feasible.

OBSTRUCTION FROM EXTERNAL PRESSURE

Pressure on the trachea may occur in the neck or in the mediastinum.

Causes of pressure in the neck.—Strangulation, throttling or garotting leads to death by occlusion of the trachea and suffocation. Enlargement of both lobes of the thyroid body may cause lateral compression of the trachea, until eventually its lumen is reduced to a narrow slit—the so-called "scabbard trachea." Irregular or unilateral enlargements on the other hand cause deviation of the trachea, with kinking of its lumen. Other less common

¹ According to Mr. Harold Barwell, administration of potassium iodide is very dangerous, as it increases the secretion which is pent up behind the stenosis. It may be given in combination with belladonna, but it is better withheld until the severity of the condition has been relieved by neoarsphenamine.

causes of compression of the trachea are enlargement of the cervical glands from tuberculosis, malignant disease, Hodgkin's disease or leukæmia. The trachea may be pressed on from behind by a foreign body impacted in the œsophagus, or by a bony tumour arising from the vertebræ.

Causes of pressure in the mediastinum.—An aneurysm of the aortic arch may press directly upon the trachea at, or near, the bifurcation and cause obstruction. Similarly deep pressure may be caused by a retrosternal goitre, a persistent and enlarged thymus, or a thymic abscess, mediastinal glands enlarged from any cause, usually malignant disease, a dermoid cyst or a bony tumour originating in the sternum.

Symptoms.—The symptoms are in the main identical with those of stenosis of the trachea from intrinsic causes, with the special symptoms due to the primary external condition superadded.

Diagnosis.—This may be simple and obvious, as in those cases due to pressure from tumours in the neck, whereas, in those due to mediastinal pressure, it is usually only possible after a careful survey of all the symptoms, and is in brief identical with that of aneurysm or mediastinal new-growth, to which reference should be made. In some cases X-ray examination may give valuable information.

Prognosis.—This is good in obstruction due to causes in the neck other than malignant disease, but it is almost uniformly bad, indeed hopeless, in obstruction due to mediastinal causes, with the exception of abscess and some thymic conditions.

Treatment.—The treatment is that of the primary condition. In goitre and tuberculous glands, in simple tumours, cysts and some thymic conditions, operation may be possible and may effect complete cure. In those due to mediastinal pressure, especially from aneurysm or new-growth, treatment, in most cases, can be only palliative or symptomatic, and directed to the relief of pain, dyspnoea, cough and distress.

INJURY

Direct violence to the trachea has been known to cause rupture when the chin is raised upwards and the trachea is, therefore, extended.

R. A. YOUNG.

G. E. BEAUMONT.

DISEASES OF THE BRONCHI

BRONCHITIS

Inflammation of the bronchi, or bronchitis, is one of the commonest maladies and may be induced by a variety of causes. These, in the main, fall into three groups: bacterial, chemical and mechanical, similar to the causes of tracheitis, which is, indeed, in many cases, a concomitant or antecedent of bronchitis, so that tracheo-bronchitis would be a more accurate designation of the majority of cases. At the same time it should be recognised

that the trachea may be alone or predominantly affected, while, on the other hand, in many cases of bronchitis of the smaller tubes, the trachea may escape, or be only slightly involved.

Bronchitis is so varied in its extent and in the form and severity of its manifestations that a satisfactory classification is somewhat difficult to formulate. We propose to consider the clinical manifestations of bronchitis according to the following classification :

1. ACUTE FORMS—(a) Catarrhal bronchitis, (1) of the larger tubes, (2) of the smaller tubes ; (b) suppurative ; (c) secondary bronchitis ; (d) bronchitis due to mechanical and chemical agencies ; (e) fibrinous.

2. CHRONIC FORMS—(a) Catarrhal, (b) suppurative, (c) secondary, (d) due to mechanical agencies, and (e) fibrinous.

1. ACUTE BRONCHITIS

ACUTE CATARRHAL BRONCHITIS OF THE LARGER TUBES

Synonyms.—This condition is often called Bronchial Catarrh, or Acute Tracheo-bronchitis.

Ætiology.—*Predisposing causes.*—Climate and latitude undoubtedly play an important part. Catarrhal bronchitis is rare in polar and arctic regions and near the equator, but is very prevalent in damp and foggy climates. In England, attacks are common in late autumn, winter and early spring. It is probable that some degree of hereditary predisposition occurs, since "weakness of the chest" is common in some families. Owing chiefly to greater exposure, the disease occurs more frequently in men than in women. It is most common at the extremes of life, infancy and old age, but it is not infrequent at any age. Fatigue and privation play their part, and exposure to cold, wet or fog so frequently seems to initiate the attack that it is often regarded as the exciting cause. Scoliosis, kypho-scoliosis and other malformations or deformities of the chest predispose to bronchitis, and some of them are induced or aggravated by bronchitis early in life. Chronic cardiac and renal disease both render their subjects more liable to bronchitis, as do also conditions of the nose and pharynx which lead to mouth-breathing, in consequence of the inhalation of air which is unwarmed and unfiltered by the nose. In childhood, dentition seems to be a frequent predisposing condition.

The exciting cause is usually one of the catarrh-producing organisms, and one or more of the following may be found in the sputum: the pneumococcus, Friedländer's pneumo-bacillus, streptococci, *Micrococcus catarrhalis*, staphylococci, *M. tetragenus*, and filter-passing organisms. It may also be caused by the *Spirochæta brachialis*.

Pathology.—The changes induced in the bronchi are similar to those in the nasal mucosa in coryza and in the trachea in tracheitis. Three stages may be described: An initial dry stage, when there is active hyperæmia of the bronchial mucosa, with exudation into the submucous layer, causing temporary diminution of the bronchial secretion from occlusion of the mucous ducts. The second or mucoid stage is associated with copious discharge of mucoid secretion, owing to increased activity of the mucous

glands, this secretion being mixed with shed ciliated epithelial cells and scanty leucocytes. Sometimes in acute cases a few red blood corpuscles are present. The third stage is that of resolution, though not infrequently a muco-purulent stage occurs, when the sputum becomes less copious and greenish in colour from large numbers of pus cells.

In fatal cases the lung tissue may appear slightly distended and red, while the bases may be sodden from cedema. On section, the bronchi appear injected and the mucosa is swollen. On squeezing the lung, beads of mucoid fluid or muco-pus exude from the cut ends of the bronchi. There is no consolidation and the lung tissue floats in water.

Symptoms.—An attack of acute bronchitis generally begins suddenly, with malaise, aching in the limbs, and a sense of oppression in the chest. If the trachea is also involved, there is the characteristic feeling of rawness under the sternum. The temperature rises, varying from 99° to 100° F. in mild cases to 103° F. in more severe ones. The cough is at first dry, irritating and ineffective, but in a few hours it becomes looser. The sputum in the early stage is scanty, tenacious and sometimes streaked with blood; it then becomes copious, mucoid and frothy in character, and is found to contain mucus, shed epithelial cells, leucocytes and red blood corpuscles. Later it lessens in quantity and may become thick, yellow and muco-purulent. With the onset of expectoration there is generally an abatement in the symptoms, the rawness under the sternum disappears, and the feeling of pain or soreness about the pectoral muscles and the costal attachments of the diaphragm lessens. The febrile reaction may last only 3 or 4 days, but the cough and expectoration may go on for 10 days or longer, gradually diminishing, until they are present only night and morning, and then cease completely. *SIGNS*

In the early stage the patient is flushed and the breathing may be slightly increased in rate, but it is rarely or never laboured, unless emphysema co-exists. Vocal fremitus is unaltered, but rhonchal fremitus may sometimes be felt over one lung or both. The chief physical signs are discovered only on auscultation. The breath-sounds may be harsher and higher-pitched, particularly in infants and children, but they remain vesicular, and expiration may be prolonged. The voice conduction is unaltered. As a rule rhonchi, either sonorous or sibilant, according to the size of the bronchus in which they are produced, are audible over both lungs, and during the mucoid stage bubbling râles may be heard, especially at the bases.

Complications and Sequelæ.—Bronchitis may go on to broncho-pneumonia or may be followed by lobar pneumonia, fibroid induration or bronchiectasis. It may lead to chronic bronchitis, or be followed by active tuberculosis. Occasionally acute interstitial emphysema may result from violent coughing.

Course.—This is variable. The patient may be convalescent in from 7 to 14 days, but cough, expectoration and a condition of debility may continue for several weeks, though, in this case, the possibility of pulmonary tuberculosis should always be considered.

Diagnosis.—The diagnosis of bronchitis is usually easy, owing to the characteristic rhonchi, but it is important to differentiate primary bronchitis from bronchitis occurring as a secondary condition in acute specific fevers and other diseases.

Prognosis.—Bronchitis of the larger tubes is rarely fatal, except when it occurs in infants or the aged, or as a complication of advanced cardiac or renal disease.

Treatment.—**PROPHYLACTIC.**—This consists in the avoidance of stuffy, ill-ventilated rooms and places of entertainment when catarrhal infections are rife. In mouth-breathers, steps should be taken to deal with the conditions of the naso-pharynx inducing this habit, and instruction in normal breathing given. In dusty occupations, suitable measures should be taken to minimise the irritant particles in the air, as is now done in most factories and workshops. Where poisonous gases have to be encountered, some form of efficient gas-mask should be utilised.

Prophylactic inoculation by vaccines, either from stock mixtures such as are now available, or from autogenous cultures, is now being extensively used, and with some success. An autogenous vaccine is usually to be preferred, if possible. The dose given depends upon the organism and varies from 1 to 50 or 100 millions. Two or three doses at intervals of 7 to 10 to 14 days are usually given in the case of the stock vaccines, whereas with the autogenous a course of 6 to 12 gradually increasing doses is given at intervals of about a week.

CURATIVE.—No matter how mild the attack may be at the onset, the patient should be kept in bed. This may only be necessary for 1 or 2 days, but he should keep to his room till his temperature has returned to normal. The Turkish bath taken by some patients at the first onset is unwise and should be discouraged. The room temperature should be kept at 60° to 65° F. While the temperature is raised the diet should be the ordinary, simple, liquid diet suitable to febrile conditions, namely, milk, weak tea, cocoa and simple gruels, broths or one of the many invalid foods. The patient is often thirsty, and warm or hot demulcent drinks, such as toast water, fruit juices in hot water, and linseed tea sometimes afford great comfort. The air of the bedroom may be moistened by means of a steam kettle in the dry stage, but the use of a steam tent is to be avoided. Local applications over the sternum, acting as counter-irritants, seem to give some relief to the distressing soreness so often complained of. A mustard leaf or one of the medicated wools is the most easy to apply, but a linseed poultice, kaolin poultice or a liniment, such as camphorated oil or the acetic turpentine liniment, may be ordered. Some patients find a cold or hot compress to the neck comforting. Medicated inhalations may be used, either in a special inhaler or in a domestic substitute, such as a jug. At first vapor benzoini—60 minims to the pint of water at 160° F.—is the most comforting, but in later stages vapor pini (olei pini, min. 10; mag. carb. levis, gr. 10; aquam ad min. 120)—120 minims to the pint—or a dry inhalation of creosote, terebene and spirits of chloroform may be useful. It is often wise to start treatment with an aperient, unless this is contra-indicated. In the dry stage a simple saline diaphoretic mixture may be given, with tinct. ipecacuanhæ or vin. antimoniale in small doses. One-drop doses of tincture of aconite are also sometimes given. When expectoration starts it may be encouraged by saline and stimulating expectorants, such as ammonium chloride or carbonate, combined with squills and flavoured with syrup of tolu or of Virginian prune. For the first night it may be well to give 10 grains of Dover's powder to relieve discomfort and secure sleep.

During convalescence the patient should take care to avoid chill and should be given a more liberal diet. A mixture of strychnine and phosphoric acid may be given for a few days, and a linctus or lozenge containing small doses of diamorphine (heroin) or other sedative, to lessen the ineffective cough, which not infrequently occurs, especially at night. Convalescence is usually shortened by a few days' stay at the coast, especially the south.

When bronchitis occurs as a part of some specific disease, such as diphtheria or influenza, the treatment should be that appropriate to the particular disease.

ACUTE CATARRHAL BRONCHITIS OF THE SMALLER TUBES

Synonym.—Capillary Bronchitis.

It is open to question whether this condition exists as a separate entity. When the finer bronchi and bronchioles are inflamed the alveoli invariably become involved, since very little swelling of the bronchiolar walls is sufficient to occlude the lumen of the tube, with the inevitable production of an area of lobular collapse. The transition from this condition to actual lobular pneumonia is a very small one. In any case, the causes, the symptoms and the treatment of capillary bronchitis and broncho-pneumonia are identical. (See Secondary Broncho-pneumonia.)

ACUTE SUPPURATIVE BRONCHITIS

Synonyms.—Sometimes called Acute Purulent Bronchitis, or Suffocative Catarrh.

This condition was brought into prominence during the Great War. In 1916 and 1917 it appeared in epidemic form amongst the British troops in England and France. Although it was then regarded by some observers as a new disease, it is more probable that it was, in reality, an epidemic form of a condition usually rare and sporadic and previously termed "suffocative catarrh." That name has unfortunately been applied loosely to a number of conditions associated with acute dyspnoea.

Ætiology.—*Predisposing causes.*—The exceptionally severe winter of 1916-17, together with conditions of overcrowding in huts and billets, were undoubtedly concerned in the epidemic just mentioned. The condition affects young adults chiefly, and is much more common in men. Over-exertion, fatigue and debility predispose to it, but the disease may occur in robust and healthy persons. A history of chill may be given, but often no obvious cause can be discovered.

Exciting cause.—The organisms usually found are the pneumococcus and Pfeiffer's *H. influenzae*, the latter being reported in 90 per cent. in some series of cases. The *Micrococcus catarrhalis* is also sometimes present.

Pathology.—A very intense inflammation occurs in the medium-sized and small bronchi, leading to an exudate rich in leucocytes. The inflammatory process may extend to the alveoli, which then contain a fibrinous fluid, with entangled red cells. The condition occurs in both lungs and is usually almost universal, no portion being spared. Post mortem the lungs are heavy and red in colour. On section the bronchi are found to contain a thick yellow purulent fluid. Small areas of collapse and sometimes of broncho-pneumonic consolidation are seen, and there is usually oedema of the

bases. Plastic pleurisy is not infrequent, and the glands at the root of the lung are enlarged.

Symptoms.—The onset is usually abrupt, often in young people apparently in robust health. A definite chill may occur, or only coryza and general malaise, with aching of the muscles. The temperature rises quickly and may reach 104° F. early in the disease. A cough soon develops and extreme dyspnoea is a characteristic feature. Expectoration starts early, often on the second or third day. At first it may be streaked with blood, but it soon becomes yellowish green and nummular; it consists of almost pure pus; there is often as much as 5 or 6 ounces in 24 hours. In most instances there is great prostration. In grave cases the patient becomes unconscious and loses control of the sphincters.

There is intense cyanosis, the face, lips and ears being purple. Respiration is rapid, 30 or 40 per minute, and the accessory muscles are often in full action. Palpation and percussion may not show any abnormality though slight dullness is sometimes present at the bases. At first no signs may be discovered on auscultation, but soon the breath-sounds become largely obscured by medium-sized bubbling râles, often audible from apex to base, both front and back. The pulse is frequent, the right heart may dilate and the heart-sounds become weak.

Complications and Sequelæ.—In severe cases recurrent bronchitis, broncho-pneumonia, fibroid disease or emphysema may follow.

Course.—In favourable cases complete restoration to health results. In severe cases the course is rapid, the patient becomes comatose from toxæmia, expectoration ceases and death occurs from exhaustion in 2 or 3 days from the onset. In other cases the disease may last for weeks and proceed to recovery or death.

Diagnosis.—The early occurrence of marked dyspnoea and cyanosis, the expectoration of copious pus, and the widespread râles without dullness are very suggestive of acute suppurative bronchitis. The disease must be differentiated from other conditions described as acute suffocative catarrh that are associated with extreme dyspnoea and cyanosis.

Acute pulmonary oedema is usually afebrile, and the sputum is albuminous, frothy and copious. The condition leading to it, such as cardiac or renal disease, may be apparent.

Capillary bronchitis or broncho-pneumonia may give rise to difficulty, but in these conditions the sputum is scanty, tenacious, sometimes rusty, and but rarely purulent; moreover, cyanosis and dyspnoea develop late and depend upon the extent of the disease and the condition of the right side of the heart.

Pneumonia of the wandering type may simulate this condition, but the character of the signs, with dullness and tubular breathing, and the rusty sputum, usually render diagnosis easy.

Prognosis.—This is very grave. The mortality is high, often as much as 50 per cent. Cases extending to 3 weeks or more with swinging temperatures usually recover.

Treatment.—Cases of this disease should be isolated. If there are indications of an epidemic spread, prophylactic inoculations with a vaccine made from the special strain of pneumococci concerned may be useful in limiting it. In any future epidemic, a trial should be made of sulphapyridine

(M. & B. 693). The steam tent and the inhalation of medicated vapours, such as vapor benzoini, may give a little relief to the dyspnoea. Oxygen should be administered either by means of an oxygen tent, the double nasal catheter or the B.L.B. mask. Venesection may give temporary relief, but produces no lasting effect. Ammonium carbonate and potassium iodide are generally recommended. Stimulants, such as brandy and strychnine, should be given freely, and hypodermic injections of nikethamide (coramine) or leptazol (cardiazol) may be given.

SECONDARY BRONCHITIS

Ætiology.—Bronchitis, usually of catarrhal type—indistinguishable as regards symptoms and signs from primary acute catarrhal bronchitis—occurs as a definite part of many acute infectious diseases and as a complication in others. Among these may be mentioned measles, whooping-cough, influenza, the enteric group, small-pox, diphtheria, malaria and plague. Acute nephritis of infective origin is often accompanied by acute bronchitis. Other conditions associated with bronchitis are pulmonary tuberculosis, glanders, secondary syphilis, pleurisy and gunshot wounds of the chest.

Diagnosis.—Bronchitis is easy to recognise, but it is important not to overlook the fact that it may not be the primary condition. In all cases of bronchitis in the early stages, the possibility of a primary acute specific infection should be borne in mind. The diagnosis is also of importance in regard to treatment—for example, in malaria, nephritis and syphilis, in which treatment directed to the primary condition may be more helpful than the ordinary treatment of catarrhal bronchitis.

BRONCHITIS DUE TO MECHANICAL AND CHEMICAL AGENCIES

Ætiology.—*Mechanical.*—Attacks of acute bronchitis may be caused by the inhalation of dust-laden air. In occupations where the worker is liable to inspire fine particles of carbon, silica, steel, iron, asbestos or kaolin, acute bronchitis may result, but more often these conditions lead to chronic bronchitis and pneumokoniosis. Pressure on a bronchus by aneurysm or new-growth, or irritation by the presence of a foreign body, may induce local acute bronchitis. The symptoms and signs are practically identical with those of the catarrhal form and need no special description.

Chemical.—Acute bronchitis may follow the inhalation of chemical irritants, either as a result of occupation, accidents, attempts at suicide, or the use of poison gases in warfare. Special attention has been drawn to this subject by the large number of cases of “gassing” dealt with in the War of 1914–1918. Death not infrequently occurred, much acute suffering was caused, and some permanent damage has resulted in many cases which recovered. “Mustard gas” produces its chief effects upon the skin, the eyes and the bronchi. A fibrinous exudate forms on the mucosa as a false membrane, which separates as a slough. The suffocative gases chlorine and phosgene affect the alveoli primarily and more intensely. Chlorine inhaled in a concentration of 1 in 10,000 causes a rapid alveolar flooding with a serous and highly albuminous fluid, and if the victim does not die at once he is liable to suffer from an acute bronchitis. A condition called bronchiolitis fibrosa

obliterans may occur as a sequel. It is often associated with asthmatic dyspnoea.

Symptoms.—These are similar to those of acute catarrhal bronchitis, but there is great pain, distress and almost constant cough, often with copious expectoration.

The treatment is referred to under the heading of Tracheitis, and is, in the main, symptomatic and directed to the relief of pain, useless cough and distress. If there is cyanosis, oxygen should be given continuously if necessary by double nasal catheter or B.L.B. mask.

ACUTE FIBRINOUS BRONCHITIS

Synonym.—Acute Plastic Bronchitis.

Definition.—A comparatively rare acute disease in which there is inflammation of the bronchi, with the formation of casts. These may be hollow or solid, and are coughed up in the expectoration.

Ætiology.—The cause of the disease is unknown. It is more common in males, and is met with both in children and in adults. It may begin as a primary catarrhal bronchitis, or develop as a complication of enteric fever, measles or pulmonary tuberculosis. Such organisms as the pneumococcus or a streptococcus may be found in the casts.

Pathology.—The casts may involve the main bronchi only, or more frequently the smaller ones and the bronchioles. They are greyish white, solid or tubular, and when large, bear the impress upon their exterior of the bronchial walls in which they have been enclosed. Thus, when a cast extends up to the lower portion of the trachea, the indentations made by the tracheal rings may be seen impressed upon it. The fine terminations generally show a spiral moulding. Chemically, they consist of fibrin or of fibrin and mucin. Post mortem the casts may be seen in some places *in situ*; in other areas the bronchi from which they have been expelled may be recognised. The bronchial mucous membrane is at times acutely inflamed, red in colour, with the lining epithelial cells desquamating, or it may appear pale and unaffected. There is usually a certain degree of emphysema, and there may be collapse of lung tissue beyond the site of obstruction.

Symptoms.—The disease generally begins somewhat abruptly with a cough and malaise. In the course of a few days the patient becomes considerably worse, dyspnoea develops and a certain degree of pyrexia, but the temperature is often not more than 99° or 100° F. The dyspnoea becomes more intense, and is the prominent and all-important symptom. The face is seen to be cyanosed, the alæ nasi and the accessory respiratory muscles are in violent action, sometimes with retraction of the intercostal spaces. There may be diminished movement of the chest, either bilateral or unilateral. If there is unilateral pulmonary collapse the heart may be slightly displaced towards the same side. Vocal fremitus may be normal or locally diminished. The percussion note is somewhat hyper-resonant over the anterior chest-wall, but behind there may be some degree of dullness over one or other lobes. If the bronchi are unilaterally affected there may be dullness limited to one lower lobe, with diminution of air entry and no adventitious sounds. Vocal resonance over the affected area is lessened. There is usually some diffuse bronchitis, as indicated by the presence of rhonchi or râles. Marked stridor

is sometimes heard with respiration. A special sign, the "bruit de drapeau," has been described when the cast lies free in the bronchial lumen. It is a dry clicking sound, caused by the flapping of the cast against the wall of the bronchus as the air passes over it. The ordinary sputum does not show any peculiarities. It may, however, show Curschmann's spirals, Charcot-Leyden crystals and eosinophile cells, and it may be absent until the crisis occurs. This consists in the expectoration of the cast after a violent fit of coughing. The cast may be stained with blood, or there is sometimes actual hæmoptysis. The peculiar nature of the expectoration often escapes notice, unless it is examined by floating in water, when a large intact cast is revealed. The dyspnœa ceases immediately after the cast has been expelled.

Complications and Sequelæ.—Emphysema may occur as the result of the violent coughing, or the disease may become chronic, recurring at intervals of varying duration. The most serious complication is laryngeal obstruction, caused by the cast becoming impacted between the vocal cords.

Course.—The disease is generally self-limited, terminating with the separation and expectoration of the cast. The acute stage does not, as a rule, continue for more than 12 to 24 hours.

Diagnosis.—The stridor and respiratory obstruction are suggestive of œdema of the glottis, but auscultation will show that the site of the lesion is lower down the respiratory tract. Asthma, and all causes of laryngeal and tracheal obstruction, must be excluded. The dyspnœa and the presence of signs localised to one lobe may suggest an active lobar collapse, or a lobar pneumonia, but the dyspnœa is more intense than is met with in either of these conditions. Casts are expectorated in diphtheria, pneumonia, chronic disease of the heart, pulmonary tuberculosis and hæmoptysis. The casts of acute fibrinous bronchitis are firmer than those found in these affections, and are expectorated in long pieces, showing the many branches and bifurcations of the bronchial tree.

Prognosis.—The immediate outlook is fair. Death may occur in the first attack, or recurrences may take place, which lead to an increasing degree of emphysema, with its usual results. The ultimate prognosis is, therefore, not good.

Treatment.—The patient should be kept in bed and treated as a case of acute bronchitis. Inhalations of medicated vapours often afford relief. Potassium iodide is believed to expedite the separation of the cast. Intratracheal injections of olive oil or lime water have been recommended, as the casts tend to dissolve in the latter. Tracheotomy instruments should always be at hand in case of laryngeal impaction.

2. CHRONIC BRONCHITIS

Chronic bronchitis is perhaps even more difficult to classify than the acute varieties, each one of which may have its counterpart in chronic form, so that the same classification may be followed. At the same time it must be admitted that, especially in the catarrhal forms, the clinical manifestations are somewhat varied.

CHRONIC CATARRHAL BRONCHITIS

Ætiology.—The causes are practically identical with those of the acute form, of which it is in most cases a sequel.

1. This affection may commence at any age, although it is more common in
2. middle life and with advancing years. Men are more frequently affected
3. than women. It seems also to have a special incidence in some families.
4. It is more common in damp and foggy climates, and is favoured by urban conditions and by dusty occupations. It starts each winter with a more or less acute catarrhal attack, but each year the summer intermission becomes shorter, until the bronchitis persists throughout the year. It tends to produce
5. emphysema and is aggravated in turn by this condition. It is especially
6. favoured by cardiovascular lesions, such as valvular defects and arterial
7. disease; also by gout, chronic nephritis, syphilis and alcoholism. Con-
8. ditions associated with chronic cough predispose to it, notably emphysema, asthma, arrested pulmonary tuberculosis, mouth-breathing and cigarette-smoke inhaling.

The bacteria found are practically identical with those in acute bronchitis, the commonest being the pneumococcus, Friedländer's pneumobacillus, *Micrococcus catarrhalis*, streptococci and staphylococci. Mixtures of two or more of these may be present. A rarer cause is bronchial spirochætosis, from infection with the *S. bronchialis*.

Pathology.—The bronchi show chronic inflammatory changes of a catarrhal nature. The walls are thickened from chronic hyperæmia and also from productive changes in the connective tissues. The mucous glands may be hypertrophied or atrophied, and there may be widespread desquamation of the ciliated epithelial lining of the bronchi. In long-standing cases there is usually some peribronchitis, leading to cylindrical bronchiectasis and distortion of the bronchi by fibrosis. There is almost invariably a greater or less degree of emphysema, which may be generalised or only marginal. Post mortem, the lungs are generally red and somewhat engorged, but if much emphysema has resulted they may be paler than normal. On squeezing the lung after section, pus or muco-pus exudes from the cut bronchi, and there is usually some evidence of œdema at the bases.

Symptoms.—A patient with chronic bronchitis complains of his "chest." By this he means that he suffers from cough, expectoration and shortness of breath on exertion. The cough varies greatly in its severity. During the warm weather the patient may be completely free, and yet suffer for years from a winter cough. It may occur frequently throughout the day and in attacks at night, or only in the mornings and evenings.

The expectoration varies considerably in quality and quantity, so much so that the old classifications of chronic bronchitis were based on this factor. Thus, there may be practically no sputum or only small tenacious pellets, the "crachats perlés" of Laennec; on the other hand, there may be a profuse expectoration resembling unboiled white of egg diluted with water, constituting the form described as "pituitous catarrh" or "bronchorrhœa serosa." Usually the sputum is mucous or muco-purulent and contains greyish-black particles mixed with a frothy fluid. The dyspnoea is largely due to the accompanying emphysema, and so indicates the degree of chronicity of the disease. At first the patient may only notice that he gets out of breath on going upstairs or on mounting slopes, but later even walking on the level causes dyspnoea.

Slight rises of temperature occur in the acute exacerbations of the catarrhal process. Slight cyanosis is frequently observed, especially after

exercise, when the accessory respiratory muscles are called into play. Sometimes rhonchal fremitus is felt. Movement of the chest is restricted by emphysema, and the percussion note then becomes hyper-resonant. On auscultation, expiration is prolonged and sonorous or sibilant rhonchi are heard all over the lungs, with bubbling râles if there is thin secretion in the smaller bronchi. On the other hand, rhonchi may be scanty or only occasionally heard. Voice conduction is unaffected. The fingers may be slightly clubbed, and further evidence of venous obstruction apparent in the dilated venules on the cheeks or along the costal attachments of the diaphragm.

Complications and Sequelæ.—The following changes may occur in the lungs—peribronchial fibrosis, bronchiectasis and emphysema. Asthma or attacks of bronchial spasm sometimes form a complicating factor in chronic bronchitis, especially in the cases of so-called bronchorrhœa. The increased cardiac strain may lead to right-sided dilatation, with basal pulmonary congestion, ascites and œdema of the legs. Late in the disease, as the result of the cyanosis, a peculiar form of confusional delirium is met with, which is worse at night.

Course.—The disease once firmly established, unless relieved by suitable climatic treatment, remains chronic and becomes progressively more severe as further damage is wrought in the lungs with each hibernal exacerbation. As the emphysema develops, a vicious circle is initiated, the aerating power of the lungs diminishes, and finally cardiac failure ensues.

Diagnosis.—Chronic bronchitis must be distinguished from pulmonary tuberculosis, bronchitis secondary to heart failure, and from bronchiectasis. In tuberculosis with bronchitis there is generally wasting, and often flattening of the chest-wall, owing to fibrosis of the lungs. In all cases where the summer intermission of the symptoms fails suddenly rather than lessens gradually, tuberculosis should be suspected. The diagnosis is clinched by the presence of tubercle bacilli in the sputum. In bronchitis secondary to heart failure, in addition to the cardiac signs, the râles in the lungs are chiefly basal and the rhonchi are not so universally distributed. In bronchiectasis the signs are usually characteristic and often limited to one lobe. The X-rays may afford useful aid in diagnosis.

Prognosis.—The immediate prognosis is good, the ultimate is bad. Much depends upon the patient's social condition and opportunities for treatment, especially in respect to climate. The expectation of life of a patient suffering from chronic bronchitis is considerably shortened.

Treatment.—Those subject to chronic bronchitis should live in a warm, equable and dry climate. In England the south-western districts are best, but it is advisable to winter farther afield if possible, either on the Riviera, the north coast of Africa, or in Madeira. High altitudes should be avoided if emphysema is present or if there are cardiac complications. Exposure to wet and chill is dangerous. The question of occupation is often difficult. Much time should be spent out of doors, provided that the patient is not exposed to the inclemencies of the elements; and, further, the work undertaken must not involve severe muscular efforts, or the inhalation of dusty or irritant particles.

In England it is difficult to find an outdoor occupation conforming with these desiderata, consequently light indoor work in a good atmosphere should be advised. Clothing should be warm but light, and afford special

protection to the chest without overloading, as some patients are liable to do. Excesses in diet are to be avoided, also alcohol and heavy smoking. The general nutrition should be well maintained, and many patients, especially those of spare habit, seem to derive great benefit from cod-liver oil during the winter months.

If cough is troublesome and expectoration tenacious or scanty, various combinations of expectorant remedies are useful, such as ammonium carbonate or chloride, tinct. ipecacuanhæ, preparations of squills or senega, with tolu, liquorice or Virginian prune as flavouring agents. A simple saline mixture such as R. Sodii bicarb., grs. 10; sodii chlorid., grs. 3; sp. chlorof., min. 5; aquam anisi ad fl. oz. 1, taken with an equal quantity of hot water in the morning or at night, may help to "clear the tubes" and give the patient a spell of freedom from cough. In older patients the ether and ammonia mixture may be given, and in cases with bronchial spasm potassium iodide with anti-spasmodics, such as stramonium, lobelia, belladonna or grindelia, may be of great value. Various antiseptic drugs, such as turpentine min. 10, terebene min. 5-10, creosote min. 3 in capsules or perles, have been recommended, and the elixir thymi et diamorphin. B.P.C. min. 60. Sedative lozenges, such as compound liquorice, heroin or codeine, are often useful in checking useless cough. Intercurrent attacks of acute bronchitis must be treated on the principles described under that condition and the patient kept indoors or in bed, as may be necessary. When an advanced degree of emphysema coexists the treatment appropriate to that condition should be applied. Sometimes benefit may follow the use of the compressed air chamber. When failure of the right heart ensues, with visceral engorgement, the treatment must be modified suitably as described under emphysema. Liniments applied to the chest-wall, especially those containing camphor, turpentine or belladonna, are soothing and afford relief. Care should be taken that any tendency to constipation is checked. In some cases, especially when the predominant organism is the *Micrococcus catarrhalis* or Friedländer's pneumo-bacillus, an autogenous vaccine prepared from the sputum ameliorates the symptoms when given in small doses. This should be considered especially in cases unable to undergo suitable climatic treatment.

CHRONIC SUPPURATIVE BRONCHITIS

Synonym.—Fetid Bronchitis.

Ætiology.—This condition is not sharply defined and is not a specific and separate nosological entity, but it is a convenient group to include cases with fetid purulent sputum. In some forms of chronic bronchitis the secretion may from time to time accumulate in the bronchi and prove offensive on expectoration. In some instances this condition becomes chronic and the expectoration is fetid up to the time of death.

Pathology.—There is chronic inflammation of the bronchi, with marked peribronchial thickening. The bronchial secretion becomes purulent, and ulceration of the bronchial wall or dilatation of the lumen may occur. Post mortem, the lungs are soft, and on section some broncho-pneumonic areas, with œdema of the bases, may be seen. Pus of an offensive nature exudes from the cut ends of the bronchi.

Symptoms.—These resemble those found in chronic bronchitis, with,

in addition, the unpleasant characteristics of the sputum, in which Dittrich's plugs may be found. These are small, yellowish bodies, with an intensely offensive odour, composed of compact secretion.

Complications and Sequelæ.—Ulceration of the bronchial walls, abscess or gangrene of the lung, and areas of broncho-pneumonia may develop. As with bronchiectasis, pyæmia sometimes ensues, with the formation of secondary abscesses in the brain.

Course.—The disease is progressive, but in the early stages there may be long remissions in which the sputum is not offensive although the bronchitis persists.

Diagnosis.—The sputum is offensive in abscess and gangrene of the lung, bronchiectasis and interlobar empyema. X-ray examination of the chest is of great value in revealing these conditions, and lipiodol or neo-hydriol investigation will usually serve to distinguish between them.

Prognosis.—As the disease becomes firmly established the patient's strength is gradually undermined from the absorption of toxins, and death ensues in the course of a few years, either from exhaustion, toxæmia or pyæmia.

Treatment.—An endeavour should be made to lessen or prevent the offensive character of the sputum. For this purpose creosote or garlic may be administered, as in bronchiectasis. If sputum is copious, postural drainage by means of a Nelson bed may be useful. Creosote vapour baths are also of great value. Apart from this, the treatment is as for chronic bronchitis.

CHRONIC SECONDARY BRONCHITIS

Chronic bronchitis is a common association of chronic cardiac and renal disease, and possibly also of gout. Its clinical characters do not need special description. It is only necessary to emphasise, as in the acute forms, the importance of recognising that the bronchitis is not the essential condition, and that treatment must be directed especially to the primary disease.

CHRONIC BRONCHITIS FROM MECHANICAL AND CHEMICAL AGENCIES

This usually proceeds to interstitial changes in the lung, and these results may be studied more conveniently under the heading of the pneumokonioses.

CHRONIC FIBRINOUS BRONCHITIS

Acute fibrinous bronchitis has been described above. In certain cases of chronic catarrhal bronchitis a fibrinous exudate may occur from time to time, with the formation of intrabronchial casts. There is then cough and dyspnoea, which abate with the expectoration of the cast. It therefore very closely resembles acute fibrinous bronchitis, and the treatment indicated is that described above.

TUMOURS OF THE BRONCHI

Tumours arising in the bronchi may be (a) simple or (b) malignant.

(a) *Simple tumours.*—The following varieties occur: Adenoma, lipoma, myxoma, papilloma and chondroma. Any of these may lead to bronchial

obstruction and, in consequence, to either collapse or bronchiectasis. Adenoma is of sufficient frequency and importance to require separate description.

(b) *Malignant tumours*.—Primary carcinoma or sarcoma may originate in the bronchi. In carcinoma the growth is usually of the columnar type, and arises from the lining epithelium of the bronchi or from that in the mucous glands. Oat-celled tumours also occur, and occasionally squamous-celled carcinoma. In some instances secondary deposits of carcinoma may follow very closely the paths of the main bronchi. Sarcoma may originate in the connective tissue of the bronchial walls.

Although the majority of primary malignant tumours within the lung originate in the bronchi, either from the lining epithelium or from the cells of the mucous glands, their pathological effects and clinical manifestations are in the main pulmonary, and it is therefore more convenient to describe them as tumours in the lung (see pp. 1219–1222).

ADENOMA OF BRONCHUS

Ætiology.—Adenoma of the bronchus occurs about equally in the two sexes, and usually in adults below the age of 40.

Pathology.—The tumour is at first small and of polypoid form, as a rule arising in a main bronchus, but not infrequently in the branch to the lower lobe. It is about twice as common on the right side as on the left. The bulbous end is generally directed towards the trachea. The surface is usually smooth and shiny, but may be nodular. An erroneous diagnosis of carcinoma was not uncommon in the past owing to differences of staining of certain of the constituent cells and their irregular distribution in the connective tissues. Metastases, however, are unknown. An adenoma often projects through the bronchial wall, giving it a dumb-bell or cottage-loaf conformation.

Clinical Features.—Often the earliest symptom is hæmoptysis, and this may be slight or profuse, since adenomata are very vascular and bleed easily. In other cases the tumour causes bronchial obstruction with resultant cough and wheezing, proceeding later to pulmonary collapse or bronchiectasis. Dry pleurisy may be an early result of infection, and at times pleural effusion or empyema may conceal the underlying cause.

Diagnosis.—Other causes of hæmoptysis must be considered, such as pulmonary tuberculosis, mitral stenosis, dry bronchiectasis or bronchial carcinoma. Pulmonary collapse may suggest an unresolved pneumonia. In cases of pleural effusion or empyema the diagnosis is liable to be overlooked. The injection of lipiodol or neo-hydriol, or tomography, will often reveal a blocked or deformed bronchus, but the diagnosis can only be established by microscopical examination of a portion of the tumour removed through a bronchoscope.

Prognosis.—This varies with the stage at which the diagnosis is established. If the condition is recognised early, and treated before the growth has extended outwards through the bronchial wall and before bronchial obstruction and septic infection have occurred, the outlook is favourable.

Treatment.—When the adenoma is recognised before secondary effects have developed, piecemeal removal through a bronchoscope, followed by radon application is often completely successful. There are, however, the

risks of hæmorrhage, primary and secondary, and of local recurrence, especially when the growth has extended outside the bronchus. Deep X-ray therapy has been recommended. If secondary bronchiectasis and fibrosis have occurred, lobectomy, or in rare cases pneumonectomy, may be necessary.

THE INFECTIVE GRANULOMATA

SYPHILIS.—During the secondary stage, a generalised hyperæmia of the bronchial mucous membrane may occur, giving rise to slight bronchial catarrh with the usual symptoms and signs, a condition that has been called syphilitic bronchitis. It is frequently beneficially influenced by anti-syphilitic treatment. In the tertiary stage, gummata may form in or near the large bronchi. They tend rather to fibrosis and contraction than to softening and ulceration, although the latter processes may occur. Contraction may lead to bronchial stenosis, with the symptoms and signs described below, or to extensive peribronchial inflammation and bronchiectasis. If the gummata extend into the lung, as may happen in rare instances, destructive lesions with cough, expectoration and hæmorrhage may result. This condition is more fully described in the section on pulmonary syphilis (see p. 1218).

TUBERCULOSIS of the bronchi occurs as part of pulmonary tuberculosis and does not require separate description.

LEPROSY.—The bronchi may be involved in this disease, with the production of cellular infiltration and even nodule formation. At first, these lesions may produce bronchitis, and they are progressive, leading to cough, expectoration, wasting and asthenia. The general clinical picture may simulate chronic pulmonary tuberculosis, from which it is distinguished by the presence of leprous lesions elsewhere, and the absence of tubercle bacilli from the sputum.

BRONCHIAL STENOSIS AND OBSTRUCTION

Obstruction of the main bronchi or of their subdivisions within the lungs may arise from causes within the bronchi or from conditions outside them, and these require separate consideration. It is important to emphasise the fact that in both conditions the symptoms differ according to whether the obstruction is sudden and complete, in which case collapse of the corresponding lung is the rule, or whether it is partial and more gradual, when bronchiectasis usually results. Obstruction of the smaller bronchi may result from spasm as in asthma (see p. 1148) or from disease as in small-tube and capillary bronchitis (see p. 1133).

(a) INTERNAL CAUSES

These are most conveniently considered in two groups—(1) Foreign bodies ; (2) those due to disease or cicatrisation of the bronchial walls.

(1) FOREIGN BODIES IN THE BRONCHI

These usually gain access through the larynx and trachea by inhalation. Any inhaled foreign body that is small enough to pass down the trachea may

reach a main bronchus, more commonly the right, or if it is small it may pass into one of the secondary bronchi. It may at once become impacted, or be moved by cough, but unless it is expelled in this way, it is sooner or later drawn into the smallest bronchus that will receive it, and there becomes impacted.

The recorded varieties of foreign body thus reaching the bronchi are very numerous, but among the more common are pieces of bone, beads, pins, coins, ear-rings, studs, pencils, fruit stones, grains, grasses, beans, nuts, teeth and pieces of tonsil or adenoid growths after tonsillectomy. Even a living fish has been inhaled into a bronchus. Foreign bodies may reach the bronchi through a tracheotomy wound, or a gland may ulcerate into the lumen of a bronchus. Broncholiths and pneumoliths, calcareous particles originating in the bronchi and lungs respectively, may be inhaled into a bronchus instead of being expectorated.

Pathology.—The pathological changes resulting from a foreign body in a bronchus depend upon the nature of the foreign body, the duration of its stay, the size of the bronchus obstructed by it, and the degree of obstruction induced. If the foreign body is smooth and comparatively little septic, and if it be removed within 24 hours or so, complete recovery after a very mild local inflammatory reaction may be expected. If, on the other hand, the foreign body is rough, or soft and laden with septic organisms, acute pneumonic processes, often septic in character, may develop very rapidly. A soft type of foreign body may swell and completely obstruct the bronchus it reaches, leading to complete collapse of the corresponding lung area, often the whole or half of the lower lobe. If the stay of any foreign body is prolonged to days, weeks, months or longer, irreparable damage almost invariably results. The forms this may take are numerous. Collapse and septic pneumonia have already been mentioned. If the obstruction is partial, septic bronchitis, with stagnation of the bronchial exudate and pus behind the obstruction, leads in turn to peribronchitis, bronchiectasis and fibroid induration of the corresponding lung area. In other cases gangrene of the lung results. Not infrequently an empyema may occur and the foreign body may be found in the empyema cavity. Suppuration round a foreign body may lead to localised intrapulmonary suppuration or abscess. Simple bronchial obstruction, uncomplicated by sepsis, may lead to bronchiectasis, owing to the resultant lowering of intrapleural pressure.

Symptoms.—During the passage of the foreign body through the larynx and trachea urgent symptoms may occur which leave no doubt as to what has happened; but this is not invariable, and the patient may not be sure whether he has inhaled or swallowed it. In any case, after a bronchus has been reached, there may be a latent period which engenders a sense of false security and leads to delay in treatment. In most cases pain, discomfort and cough develop rapidly. The cough may lead to the expulsion of the foreign body, or may cause dyspnoea if it forces it up to the larynx. The cough soon becomes noisy, often paroxysmal, and if local septic changes are set up expectoration occurs, sometimes mucoid and copious, at others muco-purulent. Hæmoptysis is not uncommon. Pain may be absent, but is often severe. The temperature is generally normal for the first few hours, but soon rises, especially if bronchitis, pneumonia or broncho-pneumonia

develops. The further symptoms are those of the reactive changes and complications which ensue.

The physical signs naturally depend upon the bronchus affected and upon the degree of obstruction. They are at first those of deficient air entry. The affected side may show less movement, and there may be some recession of the lower intercostal spaces in young people. If a large bronchus is involved and collapse results, there is some displacement of the heart to the affected side. Vocal fremitus may be diminished or absent, the percussion note impaired, and the breath-sounds and voice-sounds weak or absent over the whole or part of one lung, almost invariably the lower lobe. When bronchiectasis, empyema or other conditions develop, their characteristic signs become apparent.

Complications and Sequelæ.—These have been enumerated in describing the pathological results. Sometimes septic meningitis or cerebral abscess develops.

Course.—Spontaneous relief may occur in two ways, either by the foreign body being coughed up, as may happen within a few hours or days or after an interval of months or years, or the foreign body may track through the lungs and pleura, and be discharged in an abscess bursting through the chest wall. In both cases, if an interval of more than days occurs, irrecoverable damage may have resulted. Apart from these occurrences and from successful treatment the course is very variable. Death may occur quickly from some of the septic complications, or after a longer or shorter interval from bronchiectasis, gangrene or cerebral abscess.

Diagnosis.—The history of disappearance of some article from the mouth in the act of laughing, breathing, yawning, coughing or sighing, should always arouse suspicion of an inhaled foreign body. If signs indicating bronchial obstruction are found, the diagnosis is almost certain. In every suspicious case radiograms of the chest should be taken in two different directions, in case the shadow may be merged in that of the scapula or of the ribs. The possibility of a foreign body should always be borne in mind in cases of unilateral basic bronchiectasis, especially if no obvious cause can be found. When such unilateral lung signs develop after an anæsthetic, or after operations on the mouth or naso-pharynx, the possibility of some inhaled material should always be remembered.

Prognosis.—This is grave unless the foreign body is removed within 36 hours, owing to the various dangerous complications that may ensue. Excluding the few cases in which cure occurs by spontaneous discharge of the foreign body, about 50 per cent. of cases left untreated die within 1 or 2 years.

Treatment.—This consists in removal, if practicable, as soon as possible after the diagnosis is established. If the foreign body is in a main bronchus or one of its principal divisions it can usually be removed by means of the bronchoscope and appropriate forceps. In case of failure the question of pneumotomy may have to be considered. If this is decided on, every effort must be made to localise the foreign body by X-ray examination. If intrapulmonary or pleural suppuration has occurred, this must be dealt with surgically, and sometimes the foreign body can be removed at the same time. The medical treatment of the cases consists in that of the various conditions resulting.

(2) OBSTRUCTION OR STENOSIS FROM DISEASE OR CICATRISATION OF THE BRONCHIAL WALL

Ætiology.—Primary bronchial new-growths, including adenoma, columnar-celled carcinoma, oat-celled tumour and squamous-celled carcinoma lead to bronchial obstruction at an early stage. These conditions produce symptoms and signs practically identical with those of new-growths in the lung (see p. 1220).

A plug of mucus or a blood clot may cause temporary obstruction of a large bronchus.

The causes of cicatrisation are those leading to ulceration of the bronchial wall, with subsequent healing, such as syphilitic processes in and around the bronchi, ulceration from injury produced by a foreign body or in its removal, or by the inhalation of severe irritants. The fibroid variety of tuberculosis may also produce it.

Pathology.—The stenosis may occur in one of the main bronchi, or in one passing to a lobe or to part of a lobe. At first partial, it may progress until the lumen is almost completely occluded at one point. The changes occurring in the lung beyond the obstruction vary with its degree. At first there is retention of secretion in the bronchi, and air may be forced past the obstruction in inspiration, but not expelled during expiration, producing emphysema, with gradual bronchial dilatation. When the obstruction is more complete the air is absorbed, the lung tissue gradually becomes fibrotic, and the bronchi dilate further.

Symptoms.—Cough, not infrequently of paroxysmal character, is an early symptom and is usually a continuation of that caused by the primary condition. It may be dry or associated with mucoid sputum, sometimes blood-streaked. The expectoration may cause dyspnoea, by obstructing the narrowed bronchus. If bronchiectasis develops, the sputum usually becomes fetid.

The physical signs are those of collapse of a part of the lung and are progressive. Local limitation of movement and flattening, with displacement of the heart to the affected side, may be apparent on inspection. The vocal fremitus is diminished, the percussion note, impaired at first, may progress to complete dullness when fibrosis develops. The breath-sounds are weak or even absent, and the voice-sounds diminished. In the early stages a bronchial stridor may be audible. Compensatory emphysema of the adjacent healthy lung tissue often develops.

Complications.—These are similar to those in stenosis from a foreign body, notably fibrosis and bronchiectasis.

Course.—Unless the primary condition causing the stenosis is one which can be arrested by treatment, the condition is progressive, and eventually the area of lung beyond the obstruction becomes permanently functionless.

Diagnosis.—Bronchial cicatrisation must be differentiated from obstruction due to extrabronchial causes, such as pressure from new-growths, aneurysm and the other mediastinal conditions mentioned in the section below. The history, the physical signs and examination by X-rays and, if necessary, by the bronchoscope may help in distinguishing. The Wassermann reaction should be investigated in every case where the stenosis is proved to be of intrabronchial origin.

Prognosis.—This varies with the cause. It is most favourable in cases due to syphilis submitted to treatment at an early stage.

Treatment.—Vigorous anti-syphilitic treatment should be employed in cases due to syphilis. In other cases the treatment is to relieve symptoms by appropriate measures.

(b) EXTERNAL CAUSES

These may be subdivided into—(1) *Mediastinal conditions*, chiefly enlargement of the bronchial or mediastinal glands from tuberculosis, Hodgkin's disease or malignant growth, aneurysm of the aorta, mediastinal abscess, pericardial effusion and oesophageal new-growths. (2) *Intrapulmonary causes*, generally primary or secondary new-growths.

Symptoms.—These are practically identical with those just described, but in addition there are those of the condition causing the pressure.

Diagnosis.—This has been discussed in the previous section. The bronchoscope should not be employed where there is any suspicion of an aneurysm.

Prognosis.—This is extremely unfavourable, except in cases due to tuberculous glands and pericardial effusion, and in some cases of mediastinal suppuration.

Treatment.—This can only be palliative in the majority of cases. Useless cough may be checked by a sedative linctus of diamorphine (heroin) or morphine. Dyspnoea when due to spasm may be lessened by inhalations of creosote and spirits of chloroform, or by administration of oxygen. Pain may be relieved by aspirin or other analgesic drugs.

ASTHMA

The term asthma has been loosely employed to denote any form of dyspnoea of expiratory type occurring in paroxysms. For all conditions other than that now to be described some descriptive qualification should be employed to avoid confusion.

Asthma or true spasmodic asthma is a paroxysmal affection, occurring most frequently in patients of neuropathic inheritance. It manifests itself in attacks of severe expiratory dyspnoea due to excessive vagal discharges, set free by peripheral irritation, chemical agencies or cerebral influences.

Ætiology.—Probably no other disease shows such a varied and complex causation, but studies of idiosyncrasy and anaphylaxis have served to explain many of the obscurities.

Predisposing causes.—*Age.*—The first attack may occur at any age, even as early as the period of the first dentition. The majority of cases begin before the age of 25.

Sex.—Asthma is generally stated to be nearly twice as frequent in the male sex as in the female.

Heredity.—Asthma certainly runs in families. The heredity is not always direct, the nervous instability sometimes being evidenced in other generations by migraine, epilepsy or hysteria. The view that hypersensitiveness to certain proteins is inherited is now discredited, and it is believed that an unduly irritable bronchial centre is the factor transmitted by heredity.

Other diseases.—Gout and syphilis are said to predispose to asthma. Bronchitis not infrequently leads to paroxysms in patients with asthmatic tendencies. Tuberculosis of the lung occasionally induces it, but here again it is probably in patients with the asthmatic diathesis.

Climate and locality.—Asthmatics seem very sensitive to both of these, but no general relationship can be proved, as the effects are most variable. Some patients are better in dry, others in damp, foggy climates, and in regard to locality each patient is a law to himself.

Conditions of the nose and naso-pharynx.—Nasal obstruction from swelling of the turbinates, deflection of the septum, spurs and polypi, and conditions of the naso-pharynx, such as adenoids and enlarged tonsils, undoubtedly predispose to asthma, and may also be exciting causes of the actual paroxysm.

Exciting causes.—Chemical substances.—The emanations from certain animals may be the determining cause. The best known of these are the horse and cat, but rabbits, hares, guinea-pigs, deer, dogs and monkeys may have a similar effect. Even human hair appears capable of discharging the paroxysm. The dust from some substances, such as corn, rice or oats, the smell of certain drugs, such as ipecacuanha, and the scent and the pollen of grasses and flowers may act in a similar fashion, as also may articles of diet, and many drugs. This factor in causation has attracted much attention—in this country by Freeman, Coke and Bray, and in America by Walker. It is claimed that at least 50 per cent. of asthmatics show hypersensitiveness to various protein antigens obtainable from animals, grains, bacterial bodies, foods and drugs, and over a hundred are now available for routine testing of these patients. The analogy with the causation of hay fever and paroxysmal sneezing is obvious. This group has been referred to as “allergic” asthma.

Peripheral irritation.—As already mentioned, irritation of the nose, naso-pharynx and bronchi may be asthmogenic in those of asthmatic tendency.

Gastro-intestinal disturbance.—This is well recognised as a cause, and most asthmatics find by experience the penalties of a heavy late meal and of indigestible articles of diet. It is possible that actual metabolic errors may be a factor, as in the so-called “week-end asthma,” due to altered conditions of diet and exercise at this period.

Genito-urinary conditions, particularly in women, notably ovarian or uterine disorders, sometimes act in inducing asthma.

Cutaneous.—Asthmatics are peculiarly liable to urticaria and eczema, although these conditions usually alternate with the asthmatic attacks.

Nervous factors.—Fatigue, emotion and nervous shock may precipitate an attack. This factor cannot be ignored, even in cases due to protein hypersensitiveness, as is shown by a well-known case in which a patient susceptible to roses developed asthma when handed an artificial rose.

Pathology.—Numerous theories have been propounded to explain the asthmatic paroxysm. Among these may be mentioned vascular turgescence of the bronchial mucous membrane, spasm of the bronchial muscle and increased secretion of the mucous glands. Spasm of the diaphragm or of the inspiratory muscles has also been suggested. That bronchial spasm plays the major part seems to have been established by the experiments of Brodie and Dixon, and this view is strongly supported by their observations on the effects of drugs on the bronchial musculature. Muscarine, pilocarpine and physostigmine produce bronchial constriction and asthmatic symptoms

in animals, while atropine, hyoscyamine and chloroform abolish these effects.

There can now be little doubt that the broncho-constrictor fibres of the vagus are the channel by which the impulses discharging the asthmatic paroxysm reach the bronchi, although the possibility that impulses leading to vaso-dilatation and to increased bronchial secretion are also concerned, must be admitted.

ANAPHYLAXIS.—The important part played by extraneous proteins in the genesis of asthma and the obvious analogy between the asthmatic paroxysm and the symptoms of anaphylactic shock have suggested that in many cases, if not in all, asthma is an anaphylactic phenomenon. Evidence is accumulating in support of this view. It has been shown that the lungs of the guinea-pig killed in anaphylactic shock show extreme constriction of the bronchioles. Asthmatics are well known to show anaphylactic tendencies, and especial care in the administration of antitoxic serums is necessary with them. It is of some interest to note that the Eppinger and Hess group of vagotonics show urticaria, dermatographia, eosinophilia and liability to anaphylactic shock, all conditions which occur in asthmatics. It is tempting, therefore, to assume that the foreign protein or toxin produces the asthmatic attack by inducing vagotonicity. Lastly, the observations of Freeman, Coke and the American workers have demonstrated the cutaneous hypersensitiveness of many asthmatics to special foreign proteins. Further research is needed before it can be accepted that anaphylaxis accounts for all cases of asthma, but it is almost certainly an important factor in many.

Symptoms.—The asthmatic paroxysm most commonly commences about 2 a.m. or later, but it may sometimes develop in the daytime. There are often preliminary indications some hours beforehand, constituting the "asthmatic aura." These include restlessness, irritability, mental exaltation, less frequently depression, itching of the nose or chin, flatulence or polyuria. Some attacks are ushered in by coryza. Such warnings are not constant, and the sufferer usually wakes from sleep with a feeling of suffocation. In early attacks great restlessness, anxiety and alarm occur. The difficulty in breathing and the sense of suffocation increase; the patient sits up in bed, or gets up to throw open the window, and fixes his arms to bring into action all possible muscles of respiration. Respiration, although laboured and difficult, is often slow, inspiration being short while expiration is greatly prolonged. Both are accompanied by loud wheezing sounds, audible at a distance from the chest. The patient appears pale, but the lips are dusky and the expression is anxious and distressed. The jugular veins are distended and prominent. The accessory muscles of respiration are seen to be in violent action, notably the sterno-mastoids, scalenes and pectorals. The skin is moist and there may be marked sweating. The chest is much distended, and at each violent attempt at inspiration very little further enlargement occurs, while there is often sucking-in of the supra-clavicular and lower costal regions.

Percussion reveals marked hyper-resonance and encroachment on the cardiac and hepatic dullness. On auscultation inspiration is short and high-pitched, expiration very prolonged, and both are obscured by abundant sonorous and sibilant rhonchi, and later by bubbling râles at the bases. The

pulse is small, quick and sometimes irregular. There is usually marked epigastric pulsation. A differential blood count during an attack may show an eosinophilia of as much as 35 per cent. Cough does not develop until late in the paroxysm, and is quickly followed in many cases by the expectoration of small pellets, called "perles" by Laennec, and often likened to boiled sago or tapioca. These were carefully studied by Curschmann, and when examined on glass on a black background, prove to consist of a central highly refractive mucinoid coil, with masses and threads of mucin wrapped spirally around it. Microscopically leucocytes, mostly eosinophils, may be seen entangled in the mucus. The sputum frequently contains Charcot-Leyden crystals, which are now accepted as spermin phosphate. With the onset of expectoration the dyspnoea quickly lessens, and the attack subsides. The patient often passes a large quantity of pale urine and then may sleep until morning, awaking in apparent comfort. More frequently he appears pale, tired and anxious.

Course, Complications and Sequelæ.—Such an attack may last from a few minutes to several hours, and may remit and then return. When the spasm is very severe and prolonged into hours, with little or no remission, the condition is often termed "status asthmaticus." The patient may be extremely ill, and death may occur unless the attack remits spontaneously or as a result of treatment. More often the attacks recur at the same time each night for a considerable period extending to weeks, and then pass off, after which the patient may enjoy a period of freedom of weeks or months. The intermissions may become shorter with successive attacks, and increasing emphysema may develop. This in turn leads to secondary bronchitis, which persists, together with some degree of permanent oedema of the bases. Later still the cardio-vascular changes incidental to emphysema occur as sequels, namely, engorgement of the right heart, tricuspid regurgitation, venous stasis, ascites and oedema. Chronic asthmatics frequently present a characteristic appearance. Of thin build, with sallow complexion, anxious expression and nervous manner, they often have a long neck, high straight shoulders, and a forward stoop. Asthma necessarily imposes limitations upon those who suffer from it at all severely, although many asthmatics lead active, useful lives in spite of their disease.

Diagnosis.—This involves the differentiation from other forms of dyspnoea, particularly those of spasmodic expiratory type. The chief forms of paroxysmal expiratory dyspnoea are:

1. *Bronchial asthma or spasmodic dyspnoea complicating chronic bronchitis and emphysema.*—This condition is sometimes a late result of true asthma, but may occur independently. The dyspnoea is more persistent and is more definitely related to the bronchitic attacks, being therefore more common in the winter.

2. *Cardiac dyspnoea or cardiac asthma.*—This, like true asthma, is usually nocturnal, but the signs of failure of compensation in association with valvular or myocardial disease usually make the nature of the dyspnoea clear.

3. *Uræmic dyspnoea or renal asthma.*—This is also not infrequently nocturnal and may be almost indistinguishable from true asthma. Examination of the urine, the urea and non-protein nitrogen content of the blood, usually enable the distinction to be made with certainty. Cardio-vascular changes with high blood pressure are frequently but not invariably present.

4. *Hay asthma* is probably only a severe form of hay fever and is to be regarded as a variety of true asthma.

5. *Pulmonary tuberculosis may be associated with asthmatic dyspnœa.*—The differentiation may not be easy during the attack, but the persistence of apical signs in the interval may give a clue. It is a wise precaution to examine the sputum for tubercle bacilli in all cases of asthma. A low blood pressure in an asthmatic should also arouse suspicion of tuberculosis.

The dyspnœa of laryngeal or tracheal obstruction and of mediastinal pressure can usually be recognised by the fact that it is chiefly of inspiratory type, and may be associated with stridor, instead of wheezing. In all cases of doubt the chest should be examined with the X-rays to exclude aneurysm or new-growth.

Prognosis.—When the disease starts in childhood or in early adult life it may stop spontaneously or be relieved permanently when some causal condition is discovered and treated. During a severe attack the aspect of the patient may be so alarming that a fatal issue may seem imminent, yet death rarely occurs. In chronic cases, the ultimate prognosis is made more serious by the complicating emphysema and bronchitis, and in spite of popular belief, the asthmatic has less than the normal expectation of life.

Treatment.—(a) *During the attack.*—The list of anti-spasmodic drugs and measures employed is a long one, and it is impossible to foretell which will be efficacious, for asthmatics vary as widely in their response to drugs as they do in regard to asthmogenic causes. Drugs may be administered for this purpose by inhalation, by nasal sprays, by the mouth or by hypodermic injection. Adrenaline hydrochloride, in doses of 2 to 5 minims of a 1 in 1000 solution hypodermically, may act with dramatic efficacy if administered sufficiently early, but it should be given cautiously to elderly asthmatics. It may also be combined with pituitary extract, as in the special preparations evatmine, pitrenalin and asthmolysin. In status asthmaticus, the procedure suggested by Sir Arthur Hurst may give relief. A syringe of 1 c.c. capacity is filled with adrenaline solution 1 in 1000. This is slowly injected over a period of several minutes to half an hour or until the spasm relaxes. Ephedrine hydrochloride, in tablets of gr. $\frac{1}{4}$ to $\frac{1}{2}$, has proved itself a useful substitute for adrenaline in some cases and can be given by the mouth. Pseudo-ephedrine in doses of $\frac{1}{2}$ to 1 grain is often helpful where ephedrine fails. Ephetonin, a synthetic preparation of similar character, is also sometimes employed. Adrenaline often proves helpful as a nasal spray, especially in combination with chloretone. A weak solution of cocaine and atropine in an oily excipient has been much employed as a nasal spray, but it is not devoid of risk if used indiscriminately. The fumes of burning nitre paper, or of a powder composed of tobacco, stramonium and nitre, sometimes help to relieve the distress, but they should be avoided in cases with bronchitic complications. Smoking a cigarette or a cigar may be helpful in patients who do not smoke habitually; others are helped by cigarettes containing stramonium. Inhalations of amyl nitrite, ethyl iodide or chloroform may be tried in some cases. Various drugs have been employed, of which potassium iodide and bicarbonate with tincture of stramonium, hyoscyamus, lobelia or belladonna are the most useful. Twenty minims of liquid extract of grindelia every 20 minutes for three doses have been found useful in some cases. Other drugs which have been recommended are chloral hydrate, phenacetin and the other coal

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tar antipyretic drugs, elixir of caffeine tri-iodide (eupnine) in 60 minim doses, and an emulsion of benzyl benzoate, 120 minims every 2 hours. Other measures include drinking a cup of strong coffee, the application of a mustard leaf over the sternum, and placing the feet in hot water and mustard. In very severe cases, if all else fails, it may be necessary to inject morphine or diamorphine (heroin), but this should only be done after careful consideration, owing to the danger of inducing habit particularly with heroin. It is especially dangerous in cases of status asthmaticus.

(b) *Between the attacks.*—The greatest care should be taken to discover and deal with any predisposing or exciting cause. The patient should live in that locality which his experience shows to be most suitable for him, and at present no rules can be formulated in advising on this matter.

Diet requires careful consideration. Any article of diet to which the asthmatic is susceptible should be entirely eliminated, and only the lightest of meals should be taken after midday. Dextrose has proved to be helpful in some cases of asthma in childhood. It is recommended to give 3 teaspoonfuls in lemonade or orange juice 3 times a day, with extra sugar and sweets at meals. Alkalis may also be given at the same time. Fatigue, overwork and emotional stress are to be avoided. Care should be taken to see that the bowels act efficiently. The general health should be maintained by every possible means. Arsenic may be given by the mouth or intravenously or intramuscularly as sodium cacodylate (gr. $\frac{1}{2}$ in min. 15 sterile water). When the patient is having a series of attacks, iodide of potassium with one or more of the anti-spasmodic group of drugs such as stramonium, lobelia, belladonna and grindelia, may be given regularly with great benefit. Any local source of irritation in the nose or naso-pharynx should be dealt with adequately. Sometimes touching the nasal septum with the galvano-cautery may alone be efficacious. In cases complicated by bronchitis, the sputum should be examined bacteriologically, and a vaccine may be made from the predominating organisms, but small doses and very gradual increments should be employed, since asthmatic patients are frequently hypersensitive to vaccines. If these prove to be *Micrococcus catarrhalis*, or Friedländer's pneumobacillus, great benefit may result, but the patient should be told that the vaccine can only help the asthma by lessening the accompanying catarrh. Some cases associated with marked emphysema obtain considerable relief from compressed air baths, at first on alternate days, then daily, the course extending to 1 or 2 months.

Careful investigation of the question of protein hypersensitiveness should be undertaken, and the method of testing by means of the cutaneous application of various antigens is worth a trial. For this purpose, the particular protein antigen, or a series of such antigens, may be applied to the skin of the forearm in the form of powder, solution or paste, and superficial scarification is then effected by means of a sterile needle or scalpel. A positive reaction is shown by the development of an urticarial wheal surrounded by a hyperæmic area. A control scarification with normal saline or a paste free from protein should be made at the same time. A positive result may be expected in about 50 per cent. of asthmatic patients. If such a condition is established to one or more such substances, they should be avoided if possible; if not, the methods of desensitisation may be tried, but the results are frequently disappointing. The specific antigen

may be employed in very minute doses by injection, starting, for example, with 1 minim of a 100:1000 solution and gradually increasing. Peptone given by the mouth or by hypodermic injection is sometimes employed as a shock desensitising agent.

A variety of "shock" treatment which has given good results is the intramuscular injection of sulphur oil (*huile soufrée*), 0.03 gramme in 1 c.c. This is given in doses up to 1 c.c. once or twice weekly over a period of weeks or months.

A gold salt, allochrysin, has been given intramuscularly in doses of 0.05 gramme, followed in a week by 0.1 gramme, and then, if tolerated, up to 0.2 gramme at weekly intervals till a total amount of 2 grammes has been given.

Breathing exercises of expiratory type such as those recommended by the Asthma Research Council are often of great value.

BRONCHIECTASIS

Definition.—Bronchiectasis is a condition of permanent dilatation of one or more bronchi. When it occurs in the finer divisions it is sometimes described as bronchiolectasis.

Ætiology.—Bronchiectasis is invariably secondary, and may result from disease of the bronchi, the lung parenchyma or the pleura. Even the rare congenital cases are probably consequent on malformation, atelectasis or intra-uterine disease.

1. The bronchial conditions which may progress to dilatation are bronchitis, and any affection leading to partial bronchial obstruction, such as inhaled foreign body, tumour (simple or malignant), stenosis from cicatrization and external pressure from new growth or aneurysm. Localised pulmonary collapse thus induced seems to be the commonest antecedent condition. In children, measles and whooping-cough are not uncommon causes, especially when they follow one another in rapid succession, although either alone, if severe, may lead to it.

2. Conditions of the lung parenchyma which may cause bronchiectasis are unresolved pneumonia, broncho-pneumonia, collapse, syphilis and tuberculosis. Syphilis is rare and usually acts by leading to bronchial obstruction or stenosis. Fibroid tuberculosis is a common cause, but the clinical manifestations are as a rule masked by the primary condition. The pulmonary complications of influenza are not infrequently followed by bronchiectasis.

3. The pleural conditions which are followed by bronchiectasis are those which lead to pleural adhesion and those which are associated with pulmonary fibrosis, notably chronic pleural thickening, or empyema leading to prolonged or permanent collapse of the lung.

In a lesion with such diverse antecedents the age relations are necessarily indefinite. It may occur at any age, but is commonest in the third and fourth decades. It frequently commences in childhood, although the characteristic clinical manifestations may not develop until adult life.

Sex.—In most recorded statistics there is a striking preponderance in the male.

Social state.—It is noteworthy that bronchiectasis in its more severe form is more common in the poor than in the well-to-do.

Pathology.—Four factors in the pathogenesis of bronchial dilatation have to be considered. (1) The most important is the localised collapse which leads to secondary bronchial dilatation. (2) Weakening of the bronchial walls. Most of the conditions preceding bronchiectasis tend to induce severe bronchitis and peribronchitis, and thus render the walls more yielding. Where stagnation of secretion occurs, septic and putrefactive organisms develop, producing tryptic ferments which may act injuriously upon the lining membrane. The importance of the infective factor has been stressed by Moll. (3) Increased pressure on the walls thus weakened is the determining factor. This is generally expiratory in origin and due to the strain of cough. The actual pressure of secretion accumulating behind an obstruction may promote yielding of the bronchial walls. In cases of bronchiectasis following on collapse of the lung the force of inspiration has been regarded as contributory, but this is doubtful and in any case is less important than the expiratory strain of cough. (4) The fourth possibility is the traction exerted upon the walls of the bronchi by contracting connective tissue in the surrounding fibroid lung. This obviously postulates the existence of pleural adhesion, which is not invariably present. While this must be admitted as a possible contributory factor, its importance is certainly less than that of the preceding ones.

Congenital bronchiectasis is a pathological rarity and may be confused with congenital cystic disease of the lung (see p. 1222). It is usually unilateral, and the bronchi involved are of small size, although in some cases the lung may show a large central cavity, with smaller spaces around it. Bronchiol-ectasis is also more of pathological than of clinical interest. It occurs chiefly in children, as the result of acute broncho-pneumonic processes. It is said sometimes to follow influenza and possibly tuberculosis. The lung has a peculiar spongy appearance, to which the name "honeycomb" has been applied.

Bronchiectasis of the larger tubes may be either cylindrical or saccular. In the former condition several of the bronchi are more or less uniformly dilated, and when opened out they appear like the fingers of a glove. Sometimes the dilatations are fusiform, at others they show a beaded arrangement, described as moniliform. These forms of dilatation are usually associated with emphysema and chronic bronchitis. Saccular bronchiectasis is generally localised and may be found in any part of the lung, but is most common in the lower lobes and near the base. This is partly due to the fact that the antecedent processes fall with special stress on the bases of the lungs, and partly to the influence of gravity in leading to retention of secretion in these parts. Although it may be unilateral in origin, it often spreads to involve both bases or even all the lobes. There may be one large saccular cavity, or a series of smaller globular dilatations involving the whole of the walls of one or more bronchi. The cavities are usually filled with a fetid secretion, to be described under expectoration. When this is removed away the walls are found to be thin, smooth and formed of a single layer of mucous membrane. In places this may have ulcerated, owing to the caustic action of the secretion, and the lung tissue is thus exposed. If such a cavity may then form, and an aneurysm sometimes develops, as in a saccular cavity. The openings of the smaller bronchi, derived from the bronchus, can often be recognised in its walls. In doubtful cases the

histological demonstration of cartilage and muscle in the walls establishes the bronchial origin of a cavity. The surrounding lung tissue is usually airless and fibroid, and sometimes is almost of leathery consistence. Occasionally, however, it is emphysematous, congested or pneumonic. In the great majority of cases there is a dense pleural adhesion over the area of lung involved.

Other morbid conditions found post mortem include lardaceous disease, gangrene of the lung, empyema, pyo-pneumothorax, suppurative pericarditis and cerebral or spinal cord abscess. Owing to the obstruction of the pulmonary circulation which may result, engorgement and dilatation of the right side of the heart, tricuspid regurgitation and the results of systemic venous stasis are often found.

Symptoms.—The onset is usually insidious, the symptoms developing during the course, or as a sequel, of one of the acute or chronic affections mentioned above. In some few cases, however, they develop rapidly in patients previously in good health. This is particularly the case where bronchiectasis results from an inhaled foreign body or after general anæsthesia, and a rapid onset should lead to the suspicion of this. The cough in well-developed cases is somewhat characteristic and occurs in paroxysms. These are frequently induced by change of posture—for example, bending forward or lying down. They occur with special frequency on rising, and are usually associated with the expectoration of large quantities of sputum, due to the overflow of the secretion, accumulated in the cavities during the night, into a sensitive or relatively healthy bronchus, which excites cough reflexly. They also occur on retiring to bed and at long intervals during the day. The sputum frequently amounts to as much as 20 or 30 ounces in the 24 hours. It is generally extremely fetid, although in the earlier stages it is not invariable. The patient's breath is often also malodorous, and the stench may pervade the room or even the house in which he lives, although it is not persistent. The patient is himself much distressed by the unpleasant character of the sputum, of which he is, as a rule, acutely conscious. On standing in a glass vessel it can be seen to settle into three layers—a surface scum of light frothy mucus, an intermediate stratum of thin, turbid, greenish fluid, and a deep layer of brownish colour consisting of muco-pus, bacteria, anaerobes, spirochætes and putrefactive products, including foul-smelling organic acids. Fetid yellow bodies called Dittrich's plugs can usually be found in the deep layer. Elastic tissue is only present when erosion of the wall has occurred. Hæmoptysis is not infrequent, and may occasionally be fatal. It may be the first and only symptom in some cases, which are referred to as dry or silent bronchiectasis. Dyspnoea is not, as a rule, apparent unless the condition is widespread, or unless the pulmonary or cardiac complications are present. The general condition of the patient is at first but little affected, and there may be no fever for long periods. As the disease progresses, lassitude, anorexia and some wasting slowly develop, while bouts of fever occur, due to retained secretions or to some complication.

Physical signs vary with the extent and degree of dilatation, and also with the amount of secretion present. In the early stages there is at most slight dullness at one base, with diminished air entry, peculiar sticky, "leathery" râles, and diminished vocal resonance. When bronchiectasis is well developed the signs are almost characteristic. The patient may appear well nourished and of good colour, although on cold days, especially

in children, duskiness or cyanosis is often noticeable. There is well-marked clubbing of the fingers, generally of drum-stick character, and pulmonary osteo-arthropathy, involving many joints, sometimes develops. There may be localised flattening or retraction of the chest wall over the affected area, with diminished movement, and the heart is drawn over to this side. The remaining signs vary with the state of the cavity. If this is full, there is diminished vocal fremitus, dullness and weak or absent breath-sounds and voice-sounds. If the cavity is empty or partly empty, the vocal fremitus is increased, the percussion note is boxy or dull, while the breath-sounds are bronchial or cavernous. Adventitious sounds are then generally audible, the most characteristic being sharp metallic or "leathery" râles. Bronchophony and pectoriloquy are marked, and occasionally the "veiled puff" of Skoda can be heard. Signs of bronchitis are often apparent in the adjacent lung tissues; compensatory emphysema may be demonstrable in the unaffected parts of the lung, and on the opposite side. X-ray examination before and after the injection of lipiodol or neo-hydriol serves to define the extent of the disease and the degree of fibrosis. Tomography may also be useful.

Complications and Sequelæ.—The chief pulmonary complications are septic broncho-pneumonia, gangrene and abscess. The pleura may become involved, giving rise to dry pleurisy, which sometimes progresses to empyema and rarely to pyo-pneumothorax, while in other cases pleural adhesion and contraction result. Septic pericarditis may develop and prove fatal. Septicæmia and pyæmia sometimes occur as terminal results. Cerebral abscess constitutes a serious and somewhat common complication, and may be found in the frontal, parietal or temporal regions, the cerebellum or cord. Occasionally multiple abscesses form. Lardaceous disease sometimes develops, especially in the liver, kidneys and intestines.

Course.—This is progressive, but is often slow unless fever or complications develop, though the morbid process may eventually involve the other lung. The sputum at first may be simply purulent, then becomes unpleasant and finally fetid. The disease may start in childhood and not lead to death until well on in adult life. The course is slower in cases due to bronchitis and fibroid lung conditions than in those due to foreign bodies, new growths or aneurysm.

Diagnosis.—In well-developed basic cases this is, as a rule, easy. The history of cough, influenced by posture and associated with copious sputum, is suggestive, especially when variable physical signs are observed. The development of the characteristic sputum with these signs renders the diagnosis almost certain, and the X-rays usually serve to confirm. Radiological investigation after an intra-tracheal injection of 10 to 20 c.c. of lipiodol or neo-hydriol, through the crico-thyroid membrane or between two rings of the trachea, under local anæsthesia, or with care directly between the vocal cords, has greatly facilitated the diagnosis of bronchiectasis. Franklin has recommended the nasal route for the introduction of the lipiodol. One nostril, the oro-pharynx and the larynx are cocaineised, then a gum-elastic catheter is passed along the floor of the nose into the larynx. Some cocaine is injected down the catheter and then the lipiodol follows. An attempt should be made to direct the lipiodol towards the affected side by turning the patient towards that side. The injection should be carried out in the X-ray room and the patient instructed to restrain cough if possible until the films have

been taken. The pictures obtained are strikingly characteristic and of great value. In cases with less characteristic symptoms and signs the distinction has to be made from chronic bronchitis, especially the fetid variety, pulmonary tuberculosis, gangrene or abscess of the lung, and fetid empyema. The distinction from chronic bronchitis may be difficult, especially in the early stages when the sputum is not fetid, but the paroxysmal cough, the copious expectoration with signs including bronchial breathing and sticky râles at the base, may be strongly suggestive. In fetid bronchitis the fetid sputum is not constant, and the cough and sputum may occur only during exacerbations of the bronchitis. Pulmonary tuberculosis may give rise to difficulty, particularly in cases of apical bronchiectasis. Repeated examinations for tubercle bacilli and also for elastic tissue in the sputum should be made. The history, the mode of spread, and X-ray examination may all assist. It should be remembered that the two conditions may coexist and, this may be suspected in some cases of fibroid tuberculosis with basic excavation. Abscess and gangrene of the lung have a more acute onset and course, but the chronic cavities left by these conditions may give rise to difficulty. In such cases the history may be an important aid in diagnosis. In fetid empyema rupturing through the lung, particularly when of interlobar origin, the patient is generally acutely ill, there may be a history of pleurisy at the onset and possibly some evidence of mediastinal pressure or cardiac displacement. The rare condition of congenital cystic disease of the lung may give rise to some difficulty (see p. 1223).

Prognosis.—This varies with the cause. {If due to aneurysm or growth, the duration is determined by these conditions. Bronchiectasis induced by a foreign body is generally permanent, even when the latter is removed, but it is not progressive. If the foreign body is not removed, complications generally supervene, and the course may be rapid. In bronchiectasis due to bronchial or pulmonary disease the course may extend into years, particularly if treatment is followed strictly, but sooner or later toxæmia and general or local complications supervene, with the result that the duration of life is inevitably considerably shortened.

Treatment.—Prophylactic treatment in cases of chronic bronchitis, delayed resolution in pneumonia and other conditions tending to fibrosis is of the utmost importance. This comprises respiratory exercises, climatic treatment, inhalations and vaccines.

The medical treatment of bronchiectasis consists in measures to promote the general health and well-being of the patient, to secure efficient emptying of the cavity, and to lessen or control the putrefactive processes occurring in it. The first of these involves a careful mode of life, adequate rest and change, a good and digestible diet, and medicines such as cod-liver oil, iron, quinine, strychnine or arsenic. The evacuation of the cavity may be promoted by postural methods, such as bending over the edge of the bed or stooping forwards. This can now be effectively secured by treating the patient on a Nelson bed. By X-ray and iodised oil (lipiodol) the exact position of the cavities in relation to the trachea can be determined, and the patient placed in the position best adapted to secure effective drainage. Expectorants, especially of antiseptic character, may be given, such as creosote, terebene, tar preparations, balsam of tolu or Peru, compound tincture of benzoin or the benzoates. If the sputum is tenacious, or if much bronchitis coexists iodides

and alkalis may be given in an ordinary expectorant mixture. To lessen the fetor, creosote is most frequently given in perles of 3 to 5 minims three times a day after food, or in an emulsion with cod-liver oil. Guaiacol carbonate and other creosote derivatives may also be tried. Syrupus *allii*, in 60 minim doses, is sometimes given, and is of value; but it is not always well tolerated by patients on account of its taste and tendency to repeat. Although these drugs are helpful, the amount of antiseptic reaching the cavity by the blood must necessarily be small. Attempts to secure more direct application by intratracheal injection and by inhalation have been made. The former method is now seldom used.

For inhalation purposes, solutions of volatile antiseptics are employed on a Burney-Yeo mask, such as creosote, terebene, menthol or eucalyptol in spirits of chloroform. The mask may be worn almost continuously or at intervals during the day. The creosote vapour bath is, however, the most satisfactory form of inhalation treatment, and is of great value. This should be given in a concrete-floored room without furniture. The patient is covered with a smock, the eyes are protected by closely-fitting goggles, and the nostrils by cotton wool plugs. A small quantity of creosote is heated in a metal dish, on a tripod over a spirit lamp. When the patient inhales the vapour, which quickly fills the room, violent cough is excited and the cavity is emptied. The ensuing deep inspirations carry down creosote-laden air into the air passages. The baths should be at first given on alternate days and last from 10 to 15 minutes. When the patient becomes accustomed to them, they may be given daily for half an hour or longer. The results are often strikingly beneficial. Vaccines made from the predominant organisms found in the sputum have been given with benefit in some cases. Surgical treatment is now more often employed than formerly. Repeated washing-out through a bronchoscope, at weekly intervals, is often helpful, giving comfort to the patient by diminishing the amount of sputum, and lessening or abolishing its fetor. Induction of artificial pneumothorax is sometimes of value, especially in early cases, in which it may be successful. Unfortunately it is often impracticable, owing to adhesions, and even in cases in which it is carried out, the beneficial effect only persists as a rule while the collapse is maintained. Temporary or permanent paralysis of the phrenic nerve has also proved helpful, especially in localised basal cases and in those secondary to abscess of the lung. Lebeotomy or pneumonectomy is proving a satisfactory and effective method of cure in unilateral cases. The mortality from these operations is rapidly lessening with recent improvements in technique.

INJURY

External trauma applied to the chest-wall may cause rupture of a main bronchus. This is especially liable to occur after severe crushing accidents. One or other of the main bronchi may be completely severed from the trachea. The chief clinical feature presented in such a case is emphysema of the neck and upper portion of the chest-wall. Death usually ensues in 2 to 3 days.

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DISEASES OF THE LUNGS

HYPERÆMIA AND ŒDEMA

Hyperæmia of the lungs may be either active or passive. In the former there is an increased supply of arterial blood through the pulmonary and bronchial arterioles. In passive hyperæmia there is engorgement of the pulmonary venous radicles and capillaries. With both forms there is frequently œdema, due to the exudation of serous fluid into the lung alveoli. The term "congestion" is sometimes employed as an alternative to hyperæmia, but owing to its erroneous popular use it is best avoided.

(a) ACTIVE HYPERÆMIA

Ætiology.—This may occur in association with any acute inflammatory process affecting the bronchi, lungs or pleura. It sometimes results from the inhalation of pulmonary or bronchial irritants, such as poisonous gases or heated air. Severe muscular exertion and exposure to extreme cold are described as causes, but the former at least is doubtful. An important variety is that known as *collateral* or *fluxionary hyperæmia*, which occurs when there is obstruction to the circulation in the whole or part of one lung, from conditions such as a large or rapidly developing pleural effusion, an extensive and spreading pneumonia, or in association with pneumothorax. This may develop in the sound lung, or in the unaffected parts of that diseased. A primary form of acute hyperæmia, the "*maladie de Woillez*," has been recognised by French authors, but this is generally regarded as a mild or abortive pneumonia.

The clinical manifestations of acute hyperæmia are merged in those of the processes with which it is associated, and therefore do not need separate description.

(b) PASSIVE HYPERÆMIA

Ætiology.—Passive hyperæmia may be produced by (1) conditions impeding the venous return from the lungs; (2) those leading to increased resistance to the passage of blood through the pulmonary capillaries, and (3) failure of the driving power of the right ventricle. The commonest causes of impeded return are left-sided heart lesions causing overfilling of, and increased pressure in, the left auricle. In mitral stenosis it may occur early and sometimes almost acutely, but aortic and myocardial lesions also lead to it, when the left ventricle fails and the mitral valve yields. Direct obstruction of the pulmonary veins sometimes results from external pressure by aneurysm, mediastinal tumour or enlarged bronchial glands, or from obstruction of the lumen by thrombosis. The passage of blood through the pulmonary capillaries may be impeded by emphysema, chronic bronchitis, pulmonary tuberculosis and fibrosis of the lungs. Failure of the right ventricle occurs in the late stages of right-sided heart lesions, with tricuspid regurgitation, and as a late sequel of left-sided failure.

Passive hyperæmia is obviously in the main dependent on mechanical

factors; it is not surprising, therefore, that gravity seems to play a part in the localisation of its effects, which are usually most marked in the bases or most dependent parts of the lungs. In bedridden, enfeebled or old patients, particularly if myocardial weakness or degeneration coexists, this factor becomes of great importance. Not infrequently some degree of œdema of the bases develops, and the condition is then called hypostatic congestion. If such an area becomes infected the resulting process is known as hypostatic pneumonia. Basal hyperæmia and œdema of the hypostatic type also result from toxæmia due to diseases such as enteric fever, from poisoning by drugs such as morphine, and as a terminal event in many cerebral lesions causing increased intracranial pressure.

Pathology.—The pulmonary veins and capillaries are engorged, with the result that the lung is darker in colour and heavier, while the alveolar walls and septa are swollen. If the condition persists for some time, pigment derived from the hæmoglobin of red corpuscles escaping by diapedesis is deposited in the epithelium of the alveoli and in the fibroblasts in the inter-alveolar septa. In long-standing cases the lung is firmer than normal and brownish-red in colour, a condition described as *brown induration*. If any degree of œdema is present, serous fluid is found in the alveoli on post-mortem examination, and on section of the lung frothy serous fluid exudes, which may contain some of the pigmented alveolar cells, constituting what are called “cardiac cells.” Although congested and œdematous lung is heavier than normal, it usually floats in water.

Symptoms.—In slight degrees of hyperæmia these may be absent or negligible. In more advanced cases, they are those resulting from the impeded circulation through the lungs and the deficient aeration which this entails. Dyspnoea is the most prominent symptom, and it is generally a measure of the degree of hyperæmia. It is markedly increased by exertion of any kind, and in extreme degrees it is distressing and eventually alarming. It may be inspiratory or expiratory in type, and in the latter case it is sometimes described as cardiac asthma. In severe cases there is usually orthopnoea. Cough is almost invariably present, and there is usually some expectoration of frothy fluid, which may be blood-stained. The pigmented cells referred to above as “cardiac cells” may be found in it. Cyanosis is common and indicates the degree of anoxæmia. This may be associated with distension of the jugular veins, and there is often obvious distress. As in other forms of cyanosis there is usually some increase in the number of red corpuscles. The vocal fremitus at the bases may be diminished, the percussion note impaired, the breath-sounds weak and accompanied by rhonchi, crepitations or bubbling râles, although these signs are for the most part due to the associated œdema. In addition, the signs of the primary condition in the lungs or heart will be apparent.

Complications.—Pulmonary œdema and infarction are the chief complications.

Course.—If the venous engorgement cannot be removed, it usually tends to become progressively worse, whereas when it results from temporary cardiac embarrassment, recovery is usually complete as soon as the heart function is restored.

Prognosis.—This condition has to be distinguished from (1) chronic bronchitis, in which case there may be some rise of temperature and the

physical signs are more variable and more disseminated; (2) infarction, in which pain and hæmoptysis of sudden onset are the rule.

Prognosis.—This is so entirely dependent upon the nature and degree of the condition responsible for the engorgement that no general rule can be formulated.

Treatment.—In elderly patients, or those likely to be confined to bed for long periods, attention should be directed to the decubitus. This should be changed frequently, and if possible the patient should be permitted to sit up or to get into a chair, and encouraged to take a few deep breaths several times during the day. If the hyperæmia is associated with cyanosis and engorgement of the right heart, bleeding to the extent of 8 to 12 ounces may be helpful. If this is not practicable, the application of 6 leeches over the liver, or dry cupping of the bases of the lungs may be tried. Free purgation and the administration of diuretics, notably injection of mersalyl (salyrgan) or neptal may also help indirectly to relieve the engorgement. In cases associated with cardiac failure, the administration of cardiac tonics, such as digitalis, strophanthus or squills, the injection of strychnine, camphor in oil or nikethamide (coramine) may all be of assistance. Moderate hæmoptysis should not be checked, and cough, if effective, may be promoted by suitable expectorants. In cardiac cases a "régime lactée" or strict milk diet is advocated by some French physicians.

(c) ACUTE OR HYPERACUTE PULMONARY ŒDEMA

In this condition flooding of the alveoli with the serous exudate from the pulmonary capillaries occurs with great rapidity.

Ætiology.—It is more commonly met with after the age of 40 than before, although cases have been recorded in children. It is considerably more frequent in the female than in the male sex. Arterial disease and hypertension are the most common antecedents, but acute or chronic renal disease and pregnancy may all act as predisposing factors. It sometimes occurs in diabetes. The actual exciting cause is often obscure, and probably varies in different cases. A heavy meal, an epileptic fit, or the administration of an anæsthetic may be the immediate cause in those predisposed. In other cases it may be a manifestation of angio-neurotic œdema. Sometimes paracentesis of a pleural effusion is quickly followed by œdema, no doubt as a result of a collateral hyperæmia. It has occurred after "gassing" by chlorine. In diabetes the lipæmic condition which sometimes occurs has been suggested as the determining factor, possibly causing multiple fat embolism. Coronary occlusion and acute left ventricular failure are noteworthy causes. In some cases dissociation of the action of the two ventricles has been supposed to be the cause, the right contracting forcibly while the left is in an enfeebled or asystolic condition. In support of this contention may be added the fact that acute pulmonary œdema has been observed after rupture of the chordæ tendinæ of the mitral valve.

Pathology.—The alveoli are found to be flooded with a thin serous exudate. The lungs are heavier than normal, sodden, and on squeezing exude large quantities of greyish-yellow or pinkish fluid. Frothy fluid of similar character is found in the bronchi and even in the trachea and nasopharynx in hyperacute cases.

Symptoms.—The onset is sudden, and generally occurs when the patient is lying down, hence being most frequently observed at night. The patient awakes with intense dyspnoea, and a sense of suffocation, then frequently rolls or rushes about in the endeavour to breathe, even clutching at the throat. Cyanosis is present, and the aspect is one of anxiety and alarm. Frothy fluid, often pink in colour, may soon stream from mouth and nose, or be brought up in great gulps. The chest movements are hurried, and the accessory respiratory muscles are in violent action. Vocal fremitus is diminished over the lower lobes. The percussion note soon becomes impaired over the lungs, commencing at the bases. The breath-sounds are at first vesicular or harsh with prolonged expiration, then become faint and may be obscured by bubbling râles or crepitations, audible all over the chest. Voice conduction is diminished.

Complications and Sequelæ.—Owing to its acute and rapid course, complications do not occur. Bronchitis may result as a sequela.

Course.—The malady usually lasts only minutes or hours. Unless it remits, or treatment affords relief, the patient rapidly becomes unconscious and death follows, the heart continuing to beat after respirations have ceased.

Diagnosis.—The affection is usually so characteristic that the diagnosis is obvious. In the more protracted cases the dyspnoea and the physical signs are not unlike those of acute suppurative bronchitis or suffocative catarrh and broncho-pneumonia; but in both of these there is some degree of fever and the expectoration is less copious, and when it occurs is usually of purulent or muco-purulent character. The nocturnal onset of œdema may suggest asthma; but the physical signs and the late and scanty expectoration in the latter suffice to distinguish it.

Prognosis.—The prognosis is always very grave; but prompt treatment has saved some cases. Death may occur in less than 10 minutes, or be delayed for 24 or 48 hours. In the angio-neurotic type repeated attacks may occur.

Treatment.—The most successful treatment is the immediate subcutaneous injection of gr. $\frac{1}{4}$ morphine. Good results have also followed the injection of gr. $\frac{1}{100}$ – $\frac{1}{80}$ atropine sulphate hypodermically. These are often given together. Oxygen inhalation by nasal catheter or special mask such as the B.L.B. variety may be used. Prompt venesection has been recommended, and should be tried if possible.

(d) CHRONIC PULMONARY ŒDEMA

This is usually the sequel of chronic passive hyperæmia, and the causes and symptoms are those of that condition. It may also occur in chronic renal disease. In marked degrees of œdema, however, the signs may closely simulate those of pleural effusion, save for the displacement of the cardiac impulse. It is important to remember that some degree of hydrothorax may occur as a complication, and increase the difficulty in diagnosis.

INFARCTION OF THE LUNGS

Infarction of the lungs or “pulmonary apoplexy” results when a branch of the pulmonary artery becomes occluded by embolism or thrombosis.

Ætiology.—*Embolic forms.*—The obstructing plug may originate in any part of the systemic venous system, in the right side of the heart or on its valves or in the pulmonary artery itself. The commonest peripheral cause of embolism is detachment of a thrombus in cases of thrombo-phlebitis. This may occur in the veins of the lower extremity, or in those of the uterus after childbirth. Thrombosis with embolic detachment may also develop in prolonged or wasting diseases, such as enteric fever, tuberculosis and cancer; in acute processes, such as influenza, septicæmia and pyæmia; and in localised septic lesions, such as otitis. Pulmonary embolism is not infrequently observed after abdominal or pelvic operations, and after the radical cure of hernia or hæmorrhoids.

Intracardiac thrombi from the right auricle or ventricle, becoming detached, lead to embolism, and this occurs especially in cases of right-sided heart failure secondary to left-sided valve lesions. Vegetations forming on the tricuspid or pulmonary valves in septic endocarditis on detachment produce pulmonary infarction. Rarer causes are fat embolism after injury to bone or to a fatty liver, the entry of pieces of new-growth or hydatid daughter-cysts into systemic veins, and even air embolism.

The exciting cause of embolism is not infrequently sudden movement or strain leading to detachment of a thrombus or vegetations.

Thrombotic forms.—Thrombosis occurs as a secondary process around pulmonary emboli; but it is probable that some cases of infarction are due to a primary thrombosis. This condition may be produced by some acute or chronic pulmonary disease, such as gangrene, tuberculosis and fibrosis, and by atheroma of the pulmonary artery. Any process leading to chronic venous hyperæmia may also cause it. A rare cause is thrombo-phlebitis migrans.

Pathology.—Although the pulmonary arteries are not strictly speaking end arteries, since there is some degree of anastomosis between them and the bronchial arterioles, yet the result of their obstruction is to produce infarcts comparable with those in other organs. The origin of the blood in the obstructed area has been much discussed. Cohnheim regarded it as the result of regurgitation from the veins, a view subsequently disproved, since the infarct is hæmorrhagic even when the veins are also obstructed. It is now regarded as due to influx from the anastomosing bronchial capillaries into the pulmonary capillaries, and the escape of this blood from the latter owing to their altered nutrition. It is generally accepted that embolism is much more common than thrombosis. It has been suggested that some infarcts are not obstructive, but are the result of hæmorrhage *per rherin* in cases of extreme passive hyperæmia, and that the shape is due to the alveolar distribution of the bronchial area affected. If a large embolus has caused sudden death, it will be found arrested at the bifurcation of a large branch of the pulmonary artery, or even in one of the main divisions of that vessel. In such cases there has not been time for pulmonary changes to occur, and the chief post-mortem condition found is engorgement of the right side of the heart.

In post-mortem examination of cases where smaller emboli have led to infarction, the infarcts are usually found in the lower lobes, more commonly in the right lung. They extend to the surface in the majority of cases, and can be seen before section as slightly raised, dark red areas, with the over-

lying pleura a little roughened from inflammatory exudate. They feel hard and firm, and on section are typically wedge-shaped, with the base on the surface and the apex centrally placed. In the rare deep-seated infarcts a spheroidal form is the rule. When recent, an infarct is dark red in colour, and suggests hæmorrhage with clot formation, hence the term "pulmonary apoplexy." In some cases infarcts have a purplish hue, and are said to resemble the colour of damson cheese; later they change to brownish-red. Infarcted areas sink in water. There may be a single large infarct almost occupying one lobe, sometimes only a small one, or several of varying size and age scattered throughout the lungs. In some cases a fortunate section may reveal the embolus with its ensheathing thrombus, but sometimes a thrombus only is found. Microscopically, the alveoli and finer bronchioles are filled with red blood corpuscles, and there is a sharp delimitation from the healthy lung. If the embolus is infective, suppuration occurs, and abscess or empyema ensues.

Symptoms.—If a large embolus blocks one of the main divisions of the pulmonary artery, there is sudden intense dyspnoea, pain in the chest, distress, cyanosis, and rapid unconsciousness, death resulting in a few minutes from asphyxia. In other cases the patient gives a short cry, and falls unconscious, death occurring almost immediately from syncope. In some cases unconsciousness develops so rapidly, and the respiratory symptoms are so little apparent, that a cerebral vascular lesion may be suspected. On the other hand, life may be maintained for several minutes or even hours, the patient being unconscious or in acute distress and anxiety with urgent dyspnoea, lividity and cyanosis. Respiration is deep and laboured, but fails to give relief to the sense of suffocation. In such cases also, death may result eventually from asphyxia or syncope, or the patient may slowly recover. In less severe forms, such as occur in cardiac and in some post-operative cases, there is sudden pain with difficulty in breathing, followed in a few hours or in a day or two by cough with hæmoptysis or by the expectoration of deeply blood-stained mucus persisting for some days, and slowly clearing up. If the embolus is infective, fever, often of hectic type, results, sometimes delayed for a day or more.

In the severe cases there is cyanosis, distension of the veins of the neck, acute anxiety with exophthalmos and cold, clammy skin. The only physical signs apparent are the deep, laboured breathing, the harsh breath-sounds, and the evidence of cardiac embarrassment with feeble, failing pulse.

In less severe cases the signs are also not characteristic. There are evidences of cyanosis and distress of less urgent character, possibly some limitation of movement on the affected side, increase of vocal fremitus, localised dullness, with weak or absent breath-sounds, and sometimes a pleural rub. In some cases definite bronchial or tubular breath-sounds may be audible. A few fine râles are sometimes present in the adjacent lung areas.

Complications and Sequelæ.—Localised dry pleurisy is almost invariably present. With infective emboli, abscess or gangrene, and later empyema may result. In organisation an infarct leads to a localised area of fibrosis.

Course.—As already described, death may occur from asphyxia or syncope in the course of a few minutes or hours, although recovery occurs in some very severe cases. In the less severe forms, after the initial urgent symptoms

have passed off, recovery is often rapid and uneventful, save for pain, cough and bloodstained expectoration.

Diagnosis.—The dramatic onset, the history and the associated lesions of the veins or heart render diagnosis easy as a rule; but it may be necessary to eliminate other causes of hæmoptysis, notably pulmonary tuberculosis and chronic venous hyperæmia.

Prognosis.—This depends largely upon the initial shock. The prognosis is very grave when the patient rapidly becomes unconscious. As there is less likelihood of sepsis in cases due to cardiac lesions than in those due to localised venous thrombosis, the prognosis is rather better in the former; but, on the other hand, organisation of a clot in a vein may completely remove the source of the emboli, while the source often persists when they are derived from the heart.

Treatment.—The coagulability of the blood may be lowered by the administration of 30 grains of sodium citrate three times daily. This is a wise prophylactic measure in prolonged illness, especially when a milk régime is being enforced. When thrombosis has occurred in a peripheral vein, such as in the leg, the affected limb should be immobilised until organisation of the clot has taken place. Morphine is useful in quieting a patient if there is much mental distress when a pulmonary infarct forms; but usually the patient is collapsed and stimulant measures are indicated. An injection of morphine gr. $\frac{1}{2}$, atropine gr. $\frac{1}{100}$, and strychnine gr. $\frac{1}{30}$ is found of value in some cases. If there is dyspnoea oxygen should be administered. Venesection to the extent of 10 or 12 ounces may be tried in cases where there is marked lividity with a forcibly acting heart. Hæmoptysis, when it occurs, should not be checked. Pain may be relieved by leeches, cupping or by application of iodine. In cases with heart failure the appropriate treatment by cardiac tonics should be administered. A few cases are on record in which immediate surgical aid has been available and the operation of embolectomy has been successful.

COLLAPSE OF THE LUNGS

In collapse of the lungs the alveoli are completely or partly devoid of air. The condition may be congenital, and due to non-expansion of the lung, when it is referred to as atelectasis. On the other hand, collapse may be the result of removal of the air from lung tissue previously expanded, when it is called apneumotosis or acquired collapse. The three terms—collapse, atelectasis and apneumotosis—are, however, used as synonyms by many writers.

ATELECTASIS OR CONGENITAL COLLAPSE

Ætiology.—This condition occurs in still-born and in premature infants, and probably persists to some degree for weeks or even months in weakly children. It may result from immaturity or from weakness of the inspiratory muscles, and from obstruction of the air passages by mucus and meconium. It may be a consequence of disease, such as congenital syphilis or lesions and developmental defects of the nervous system.

Pathology.—Atelectasis is due to failure of the respiratory mechanism

to draw air into the alveoli and expand them, as occurs normally with the first few inspiratory efforts of the newborn infant.

Atelectatic lungs are solid, airless and small. They are usually described as presenting appearances similar to those of adult liver as regards colour and consistence. In partial atelectasis the lung appears mottled, and small expanded areas of pinkish colour may project from the surface. The condition is chiefly of medico-legal and pathological interest.

APNEUMATOSIS OR ACQUIRED COLLAPSE

Collapse of previously expanded lung may be active or passive, the former being due to active shrinking of the lung owing to defects in the inspiratory musculature, the latter to conditions disturbing the pressure relations within the thorax.

1. ACTIVE PULMONARY COLLAPSE.

Synonyms.—Active Lobar Collapse ; Massive Collapse.

Ætiology.—This condition was first described by William Pasteur in 1890 in cases of diphtheria associated with paralysis of the diaphragm. In 1908 he pointed out that it occurred also as a sequel of operations, especially of those upon the abdominal organs, less frequently of those upon the neck and pelvis. It is highly probable that many post-operative lung conditions formerly recorded as pneumonia were in reality due to active collapse. It may also follow after injuries, such as those resulting from falls from a bicycle or a horse. During the War of 1914–1918, when chest wounds were collected in special hospitals, it was found that massive collapse was not infrequently an important complication of penetrating and non-penetrating wounds of the chest. It was also noticed in some cases after severe wounds of the buttocks and pelvis.

Pathology.—The mechanism by which deflation results is obscure, and is the subject of controversy. Pasteur regarded the condition in the diphtheritic cases as due to paralysis of the diaphragm through the phrenic nerves or their nuclei, and in the post-operative and traumatic cases as a consequence of reflex inhibition of this muscle. Briscoe, on experimental, pathological and clinical evidence discards Pasteur's explanation. He maintains that the deflation is caused by an exaggeration of the normal phenomena of breathing in the supine position, in which he states that the crural portion of the diaphragm alone contracts, the costal portion being in abeyance. In the supine position, with quiet breathing, deflation of the lower lobes occurs, and this is promoted by conditions of debility, toxæmia or operation. The clinical manifestations described by Pasteur are regarded by Briscoe as the result of superadded pleurisy, or of inflammation of the crura of the diaphragm.

Boland and Sheret have put forward the suggestion that massive collapse is due to obstruction of the bronchi, followed by removal of the air in the corresponding lung areas by absorption into the blood stream. The obstruction is supposed to be due to increased secretion and the inhibition of the cough reflex.

Post mortem, the lower lobe of one lung is usually found to be deflated and retracted towards the spine. Sometimes the whole of one lung may be affected, or both lower lobes. The collapsed area is bluish-red, firm, does not crepitate and sinks in water. Pleurisy or pneumonic changes may be

seen, and these are regarded by Pasteur as secondary to infection of the deflated lung, the resistance of which is lowered, and by Briscoe as the essential factor in the production of the symptoms. In massive collapse the heart and mediastinum are displaced towards the affected side, and the sound lung is often bulky and distended.

Symptoms.—The symptoms commonly commence within 24 or 48 hours of the injury or operation, although they may rarely be delayed for 5 to 7 days. The onset is generally sudden, with pain in the lower part of the thorax or behind the sternum. Severe dyspnoea quickly follows, and the patient appears dusky, cyanosed and alarmingly ill. Cough, with viscid mucoid expectoration, generally develops in a few hours. The latter may become copious and muco-purulent if pneumonic changes ensue. The pulse and respirations are markedly increased in rate, and the temperature not infrequently rises to 103° F. Occasionally the onset is more gradual with pain and cough, and in some cases of wounds it may give rise to few symptoms and be discovered only on routine examination.

Examination of the chest shows diminished movement on the affected side, and often absence or reversal of epigastric excursion with respiration, whereas the movement on the other side may be exaggerated. In other respects the signs usually simulate those of lobar pneumonia. Over the collapsed lung the vocal fremitus is increased, the percussion note is dull, the breath-sounds are tubular, and bronchophony and whispering pectoriloquy are present; but as a rule there are no adventitious sounds, although occasionally rhonchi and a few fine râles may be heard. In some cases the breath sounds are very weak or almost absent, and voice conduction is diminished. Over the healthy lung, loud and harsh breathing is audible. The displacement of the cardiac impulse towards the collapsed lung is a point of cardinal importance. It is noteworthy that in certain cases of gunshot wounds of the chest the collapse affects the contra-lateral lung.

Complications and Sequelæ.—Bronchitis, lobar pneumonia, or pleurisy may occur as complications. There are usually no sequelæ, except that pleural adhesions may occur.

Course.—The course of the affection is rapid. After periods extending from 2 to 5 days the temperature falls to normal, the symptoms disappear, the lung quickly re-expands, the heart returns to its normal position, and there is complete recovery.

Diagnosis.—The most important conditions from which this malady has to be distinguished are lobar pneumonia, pulmonary embolism, pneumothorax and pleural effusion. The position of the cardiac impulse is often the deciding factor: in collapse it is displaced towards the lung involved, in pleural effusion and pneumothorax it moves away from the affected side, whereas in lobar pneumonia there is usually no cardiac displacement, although there may be dilatation. Labial herpes and blood-stained expectoration are frequently seen in pneumonia, but not in collapse. When in right-sided collapse there is marked distension of the left lung with obliteration of the normal cardiac dullness, the signs superficially resemble those of a left-sided pneumothorax; but with careful examination no such error should be made. The distinction from pulmonary embolism may be difficult at first, but the localisation of the signs, and the blood-stained expectoration, may give useful indications.

Treatment.—**PROPHYLACTIC.**—The administration of morphine and atropine before the anæsthetic, propping up of the patient in bed as soon as possible after it, and the insistence upon periodic deep breaths are useful measures in preventing the onset of lobar collapse. When the "collapse attack" develops oxygen should be administered and pain relieved by local applications, such as leeches or poultices, or by an injection of morphine, atropine and strychnine. Expectorants may be given if the cough is ineffective, and cardiac tonics, such as digitalis, strophanthus and caffeine if there is much cardiac embarrassment. Injections of strychnine or coramine are useful if the patient is collapsed at the onset. Bronchoscopic aspiration has been recommended in cases of post-operative massive collapse.

2. PASSIVE PULMONARY COLLAPSE.

This form of collapse may affect the whole of one lung, or be confined to one lobe or to groups of lobules.

Ætiology.—Total collapse is generally the result of pleural effusion, empyema, pneumothorax or obstruction of a main bronchus. In a large effusion and in pneumothorax collapse is complete, unless the shrinkage is prevented by adhesions. In a smaller effusion, the process may be limited to the lower parts of the lung. Other causes of lobar or partial lobar collapse are conditions leading to complete obstruction of a main bronchial division, particularly new-growth, aneurysm or foreign body. It also occurs in aged or bedridden patients, or in those with enfeebled inspiratory muscles, when prolonged fever has enforced a dorsal decubitus. Abdominal distension from tympanites or ascites can also cause collapse of the bases of the lungs.

Lobular collapse results from any condition impeding the air entry to the smaller bronchi or bronchioles, such as bronchitis, broncho-pneumonia, pulmonary tuberculosis, whooping-cough and diphtheria. Obstruction of the naso-pharynx by enlarged tonsils and adenoids may cause partial collapse, especially in the upper lobes.

Pathology.—The deflation of the lung area may be produced in three ways—(1) By complete obstruction to the air from blocking of a bronchus or bronchiole, the residual air being absorbed; (2) by enfeeblement of the inspiratory mechanism similar to the process in active collapse; and (3) by disturbance of the intrapleural pressure by fluid or air, the lung at first contracting in virtue of its elasticity until the intrapleural pressure becomes equal to that of the atmosphere, when any further accumulation of fluid or air causes positive pressure and compression of the collapsed lung.

Post mortem, in complete or lobar collapse the appearances are similar to those in active collapse. In lobular collapse the deflated areas are contracted and depressed below the level of the healthy lung. They are dark red or slaty in colour, while the adjacent areas are pinkish and often emphysematous. The collapsed areas do not crepitate.

Symptoms.—Total collapse of the lung or of a lobe being usually a secondary process, the symptoms and signs are masked by those of the primary condition, such as pleural effusion, pneumothorax, growth or aneurysm. It can, however, usually be demonstrated by X-ray examination, the collapsed lung being apparent as a fairly dense shadow lying alongside the vertebral bodies. Not infrequently, however, in pleural effusion definite tubular breath sounds, with bronchophony and pectoriloquy, may

be audible in the relatively dull area above the level of the fluid posteriorly, and these signs are due to the collapsed lung. In aortic aneurysm, or less commonly in mediastinal, pulmonary and bronchial neoplasms, distinctive signs due to the local collapse may be apparent. These consist of slightly diminished movement of the corresponding part of the chest wall, with diminution of vocal fremitus and impairment of percussion note or actual dullness. Breath-sounds are weak, as a rule, but may be bronchial or tubular. Voice conduction is increased, and in incomplete collapse crepitations are often audible. The cardiac impulse may be displaced towards the affected side, but this is less apparent than in active collapse, and it is not infrequently displaced to the opposite side by the primary condition. Lobular collapse gives rise to no symptoms which can be differentiated from those of the condition inducing it.

Complications and Course.—The lung usually re-expands wholly or in part when the condition causing collapse has been removed. Thus a lung that has been maintained continuously collapsed by artificial pneumothorax, with repeated refills for as long as 4 years or more, will re-expand when the gas in the pleural cavity is not replaced. In chronic effusion, or in large or neglected empyemata, re-expansion may be incomplete, and some falling in of the chest wall results. Fibroid changes may occur in lung tissue which has been long collapsed.

Diagnosis.—This is frequently a matter of inference, owing to the nature of the primary disease. Valuable help may be afforded by X-ray examination.

Treatment.—No special treatment apart from that of the condition causing the collapse is required. If a pleural effusion is slow to absorb, the necessity for paracentesis or gas replacement, to avoid pleural thickening, may have to be considered.

HÆMOPTYSIS

It should be recognised that hæmoptysis is a symptom, not a disease. It is here considered separately because the accurate diagnosis of its origin is essential to its treatment, which differs widely in different conditions.

Definition.—The term hæmoptysis is arbitrarily restricted to the expectoration of blood, entering the air passages from structures below the larynx or from the larynx itself. When the blood is derived from the naso-pharynx or mouth it is sometimes described as spurious hæmoptysis.

Ætiology.—1. Pulmonary tuberculosis is the commonest cause, the blood being derived from an aneurysm in a pulmonary cavity, or from ulceration of a small vessel, or congestive processes around the early lesions.

2. Chronic venous congestion, particularly in mitral stenosis. These two conditions account for the majority of cases.

3. Inflammatory and destructive diseases of the lungs, air passages or pleura, such as pneumonia, broncho-pneumonia, especially the influenzal variety, abscess, gangrene and bronchiectasis with ulceration of the walls. A latent bronchiectasis without sputum may cause recurrent hæmoptysis (*forme hémoptoïque sèche*). Pneumokoniosis, streptotrichosis and ulceration of the larynx, trachea or bronchi from tuberculosis, gumma or new-growth

may also be associated with hæmoptysis. Breaking down of a caseous or calcareous bronchial gland is a rare cause, as also is rupture of an empyema through a bronchus.

4. Infarction of the lung from embolic or thrombotic obstruction.

5. New-growths of the lung, bronchi or mediastinal glands.

6. An aortic aneurysm may cause hæmoptysis by "weeping" through an eroded bronchus, or by direct rupture, the latter being of course immediately fatal.

7. Traumatic causes.—Injury may cause hæmoptysis, by fractured ribs wounding the lung, by contusion and by breaking down of healed tuberculous lesions. Hæmoptysis occurs frequently in wounds of the chest, both penetrating and non-penetrating. A foreign body, such as a piece of shrapnel, may lie dormant for years, and then cause recurrent hæmoptysis.

8. Certain abnormal blood conditions, chiefly leukæmia, purpura, hæmophilia, scurvy, minor degrees of vitamin C deficiency and occasionally pernicious anæmia. Hæmoptysis occasionally occurs in the malignant specific fevers, especially small-pox and measles.

9. Parasitic causes, such as pulmonary distomatosis and spirochætosis, are common in Asia but rare in Europe. Hydatid disease of the lung may cause repeated slight hæmorrhages.

10. Vicarious menstruation.—Some cases in women have been regarded as vicarious menstruation, and this view dates back to Hippocrates. It is probable, however, that most cases are to be explained as due to leakage from obscure pulmonary lesions.

11. Hæmoptysis occasionally occurs in apparently healthy persons. In some, with high systemic arterial tension, it is probable that the pulmonary arterial pressure is also raised, and the condition may be regarded as analogous to the epistaxis which occurs more commonly in such patients. Sometimes the hæmoptysis is due to leaking from an old arrested tuberculous lesion.

12. Rupture of an hepatic abscess or hydatid cyst through the diaphragm into a bronchus is an occasional cause.

Spurious hæmoptysis is usually due to staining of the saliva or the pharyngeal secretion with blood, generally derived from the gums, which are spongy and congested, often from early pyorrhœa. The condition is common in anæmic girls, and is, as a rule, observed in the morning. Hæmorrhage from an enlarged pharyngeal vein is often suggested as a cause, but is rarely seen. Hæmorrhage after tooth extraction, and staining of the mucus expectorated after epistaxis, are other causes of spurious hæmoptysis.

Pathology.—From the list of causes it might be inferred that the origin of the blood differs in different cases. It may come from the pulmonary or bronchial vessels in pulmonary tuberculosis and other lung or bronchial conditions, and also in chronic venous congestion or infarction. It may come from the thoracic aorta direct, or from some of its branches, in aneurysm and mediastinal new-growth, and from the hepatic vessels in abscess of the liver. In cases due to disease of the trachea and larynx it comes direct from the vessels supplying them.

Post mortem, the larynx, trachea and bronchi may contain clots, or blood-stained froth and mucus, and their walls may be stained in places. Dark reddish areas of lobular distribution, due to inhaled blood, may be seen

in various parts of the lungs, particularly at the bases. Sometimes this may induce bronchitic changes, described as hæmoptoic bronchitis. Careful search in cases of profuse hæmoptysis will usually reveal the source of the hæmorrhage, and in pulmonary tuberculosis this is generally a ruptured aneurysmal dilatation in a cavity or an ulcerated vessel. The aneurysm may be small and escape notice unless many cuts are made into the lung.

Symptoms.—In hæmoptysis, the patient often experiences a tickling in the throat, followed by a gush into the mouth with a salt taste, and on expectoration notices blood. The alarm and anxiety this occasions lead to restlessness and rapid action of the heart. If the bleeding is profuse, cough is frequent, and large clots, together with liquid alkaline blood, may be expectorated to the extent of 20 or 30 ounces in a few hours. The bleeding may cease temporarily, to recur at intervals for several days, until the patient becomes blanched, weak and syncopal, with rapid, weak pulse. In any profuse hæmoptysis, death may occur in a few minutes, either from asphyxia or syncope. In the former case, the blood, at first bright and arterial, is soon dark and frothed, while the patient becomes cyanosed and livid. In slighter degrees of hæmoptysis there may be only streaks, small clots or liquid blood mixed with ordinary sputum. After the actual bleeding has ceased, the sputum may be blood-stained for some days, owing to the expectoration of blood inhaled into other parts of the lungs. This can be recognised by its colour, which varies from dark red to brown, owing to the changes undergone by the blood pigment.

Diagnosis.—This involves two problems—first the differentiation from hæmatemesis and spurious hæmoptysis, and secondly the recognition of the cause of the hæmorrhage. If the patient is seen at the time of the bleeding the first of these is easy. The nature of the blood, and its association with cough and possibly with pulmonary or cardiac signs, are conclusive. When the diagnosis has to be made upon the history given by the patient or by friends it may be difficult, especially in the absence of physical signs.

In hæmatemesis there is frequently gastric pain and faintness before the vomiting, the blood is acid in reaction, dark in colour, even brown from acid hæmatin, and is sometimes mixed with food. The fact that in hæmoptysis blood may be swallowed and subsequently vomited increases the difficulty. Patients often give very dubious answers to questions as to whether the blood was coughed or vomited up. They should then be questioned as to whether sputum was brought up on the following day, and, if so, whether it was blood-stained. In cases of doubt the investigation of the pulmonary and abdominal physical signs, when the patient's condition permits, may decide the diagnosis.

The utmost caution should be exercised to exclude tuberculosis before making a diagnosis of "spurious hæmoptysis." Only when there are no pulmonary symptoms, signs or X-ray indications, and when some obvious cause, such as anæmia or pyorrhœa, is found, is it safe to do so.

While distinguishing between the various causes of hæmoptysis it is well to regard and to treat it as due to pulmonary tuberculosis until some other cause is conclusively established. The sputum should be examined for tubercle bacilli on several occasions, the temperature recorded and the physical signs including X-ray appearances most carefully watched.

The presence of a valvular lesion, especially mitral stenosis with signs of

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pulmonary engorgement, may render the cause of hæmoptysis clear. When tuberculosis and cardiac disease can be excluded, a careful study of the history, the symptoms and signs, may throw light on the diagnosis or suggest some investigation which will serve to establish it, e.g. examination of the sputum for parasites and hydatid hooklets, the cytological examination of the blood and an X-ray examination.

In other cases, as in bronchiectasis, abscess or gangrene, the history, the physical signs and the nature of the sputum are often characteristic.

In the latent or silent form of bronchiectasis (*forme sèche*), the condition may be revealed only by lipiodol injection.

Bronchoscopy may be of great value in revealing the presence of adenoma or carcinoma.

Prognosis.—Apart from hæmoptysis, which is rapidly fatal, due to aneurysm or pulmonary tuberculosis, the immediate prognosis in cases of pulmonary hæmorrhage is not unfavourable, even when it continues for days. The ultimate prognosis depends upon the cause.

Treatment.—This is so entirely dependent upon the cause and origin of the bleeding that reference should be made to the corresponding diseases.

EMPHYSEMA OF THE LUNGS

Emphysema of the lungs, or alveolar-ectasis, is a condition of distension of the alveoli; it is usually progressive and is associated with definite changes in the inter-alveolar walls. The following varieties are generally recognised—(1) Large-lunged or hypertrophic; (2) small-lunged or atrophic; (3) compensatory; (4) acute vesicular; and (5) acute interstitial emphysema. The last named condition has no relation to true emphysema except in name, but will be described in this group for convenience.

1. LARGE-LUNGED OR HYPERTROPHIC EMPHYSEMA (SUBSTANTIVE OR IDIOPATHIC EMPHYSEMA)

This is a chronic affection and is usually bilateral.

Ætiology.—*Predisposing causes.*—It may occur at any age, even in childhood, but is most frequently seen in middle and late adult life. It is commoner in men than in women, probably because they are more exposed to the conditions inducing it. Although not strictly hereditary, it often shows a familial incidence. Certain occupations are credited with being concerned in its production, notably those involving violent or prolonged muscular effort with closed or partially closed glottis, such as blowing wind instruments and lifting heavy weights. Dusty occupations also favour its onset by leading to bronchitis and cough.

The common *exciting cause* seems to be the strain of prolonged and repeated cough, induced by chronic bronchitis, bronchiectasis, asthma, whooping-cough, cigarette smoke inhaling, and other causes of irritation of the upper air passages.

Pathology.—The pathogenesis of emphysema has been much debated and various explanations have been offered. (1) **Primary degeneration theory.** Villemin suggested that the essential lesion was a fatty degenera-

tion of the alveolar walls, while Cohnheim believed that there was a congenital defect of the elastic tissue of the lung. (2) The inspiratory theory, first suggested by Laennec and developed by Gairdner, postulates the force of inspiration as the distending agent. (3) The expiratory theory, first enunciated by Mendelssohn, was independently brought forward and established by Jenner. The distension of the alveoli is regarded as due to the effect of forced expiration and cough. Jenner pointed out the special and early involvement of the apices, the anterior and lower margins of the lungs; in other words, the parts least supported by the thoracic cage. (4) Freund regarded the changes in the lungs as secondary to calcification of the costal cartilages, the chest becoming fixed in the inspiratory position and the lung permanently expanded in consequence. The expiratory explanation is now generally accepted, and emphysema is regarded as the result of increased intra-alveolar tension, due to violent expiratory efforts, acting on walls weakened by congenital defects, by inflammatory processes or by toxic agents, such as alcohol (Nothnagel).

The characteristic conformation of the chest is usually apparent (see Symptoms), the costal cartilages are often calcified, and on opening the thorax post mortem, the lungs bulge instead of retracting, so that the pericardium may be almost completely obscured. They are pale in colour, even in town-dwellers, a condition called albinism of the lung by Virchow. They are soft and pit on pressure, and, as described by Laennec, give the sensation of a down pillow. The surface of the lung under the pleura shows a finely vesicular appearance, due to the distension of the alveoli, the vesicles often being nearly as large as pins' heads. Not infrequently large bullæ or blister-like protuberances, varying in size from a pea to a Spanish olive, occasionally much larger, may be seen projecting from the surface, particularly at the apices and margins. These bullæ when incised show fine fibrous bands crossing them, the remains of inter-alveolar walls and of atrophied blood vessels. It was formerly customary to refer to such cases as bullous or marginal emphysema and to describe those in which the dilatation is less obvious but more widely diffused as general emphysema; but the conditions are so commonly associated together in varying degrees that little is gained by so doing. On section the lungs are pale and dry, except at the bases, where there is frequently some cedema in advanced cases. The bronchi may show some general dilatation, although less commonly than might be expected from the close similarity of the causal factors of emphysema and bronchiectasis. When bronchitis coexists, muco-pus can be squeezed from the cross-sections of these tubes. As pointed out by Fowler, pleural adhesion is relatively uncommon. The infundibula and alveoli are dilated, and the inter-alveolar walls are thin and atrophic, even disappearing wholly or in part. The distension and coalescence of adjacent alveoli result in the formation of bullæ. The calibre of the pulmonary capillaries is diminished by stretching of the alveolar walls, and where atrophy of the inter-alveolar septa occurs the capillaries are destroyed. These two processes result in a considerable diminution in the total aerating surface, and cause the dyspnoea and cyanosis characteristic of the disease. Moreover, the normal anastomoses between the terminal bronchial and pulmonary capillaries increase considerably, and some of the blood in the latter may therefore fail to reach the alveoli and so escape aeration. Atrophic changes in the elastic tissue

have been described. In order to maintain the circulation through the diminished capillary area, the right ventricle hypertrophies and the resultant raised blood pressure sometimes induces atheroma of the pulmonary artery. Emphysema being a progressive lesion, and the defective aeration of the blood perhaps interfering with the nutrition of the heart muscle, cardiac failure eventually ensues, causing tricuspid regurgitation, engorgement of the right auricle, and the visceral effects of venous engorgement, such as "nutmeg" liver. Cabot states that true emphysema is often not found post mortem in cases so diagnosed during life, and prefers to designate the clinical entity here described as "the Barrel Chest."

Symptoms.—Dyspnoea of varying degree is the most characteristic symptom. In uncomplicated cases of moderate extent it is only present on exertion, unless bronchitis coexists. In advanced emphysema, dyspnoea is marked and becomes extreme in the bronchitic or "asthmatic" attacks and in foggy weather. Cyanosis is common, and is to some extent a measure of the degree of emphysema. Varying degrees of polycythemia may be observed. The patient may walk about with a more extreme degree of cyanosis than in any other condition except congenital heart disease. Clubbing of the fingers of moderate degree is common. Cough is usually due to the associated bronchitis, and is worse in the winter and in foggy weather. It is frequent, noisy and often hacking and paroxysmal. Expectoration is also the result of the bronchial catarrh, and varies from a few grey mucoid pellets to copious mucopus.

The chest is enlarged, particularly in the antero-posterior diameter, the upper thoracic spine is rounded and kyphotic, the sternum protrudes forward, and the angle of Louis is prominent, the general effect being the so-called barrel-shaped chest. The ribs run forward more horizontally and the intercostal spaces are wider than normal, the chest being as a whole in the inspiratory position. The respiratory movements are much restricted, the patient elevating the rigid thorax with little expansion on taking a deep breath, so that the inspiratory increase at the level of the nipples may be only half to 1 inch, instead of the normal $2\frac{1}{2}$ to 3 for an adult. There is often filling and even bulging of the supra-clavicular hollow, while the neck appears short, the sternomastoids stand out, and the jugular veins are full. A zone of dilated venules, the "emphysematous girdle," is often present along the line of the costal attachment of the diaphragm, but is not pathognomonic. The cardiac impulse is not visible as a rule, and may only be felt with difficulty, but epigastric pulsation is usually apparent. Vocal fremitus is diminished, and the percussion note is hyper-resonant. The superficial cardiac dullness is greatly diminished or even absent, and the lower limit of pulmonary resonance may extend to the costal margin, back and front, the hepatic dullness being encroached on or obliterated.

It is said that in bullous emphysema the breath-sounds are harsh over the outer part of the upper lobes in front, and weak at the bases. In general emphysema the breath-sounds are weak everywhere, inspiration is short, and expiration is greatly prolonged. A loud rumbling, from contraction of the thoracic muscles, may entirely obscure the breath-sounds. A few fine bubbling râles may be heard at the bases or at the sternal margins. If bronchitis is present, scattered rhonchi may be audible. Vocal resonance is generally slightly diminished. The heart-sounds are weak and distant,

and in late stages a tricuspid systolic murmur may develop. The "vital capacity" of the lungs, measured by a spirometer, is often reduced to one-half or less. Examination by the X-rays shows increased extent, and undue transradiancy of the lung tissue. They show the diaphragm lower in position and flattened, and the costophrenic angle widened. The liver is sometimes palpable, possibly from downward displacement by the bulky lung, but more often from enlargement due to passive hyperæmia. The spleen may also be depressed and enlarged.

Complications.—Bronchitis is the commonest, and often constitutes a vicious circle. Asthmatic attacks, so-called "bronchial asthma," are common in later stages; on the other hand, spasmodic asthma may be the cause of the emphysema. Pneumothorax and interstitial emphysema may occur from rupture of the bullæ, although these accidents are surprisingly rare. Pulmonary tuberculosis is an occasional complication of emphysema, which, contrary to popular opinion, is not antagonistic to it, although it may mask and obscure the early stages. Right-sided cardiac failure, with its train of consecutive changes, is a late and often terminal complication.

Course.—Emphysema is progressive, unless the cause is removed or the effects of the disease are mitigated by residence in a warm, dry climate, especially in the winter. Conversely, residence in unsuitable districts, persistence in detrimental employment, and repeated attacks of bronchitis accelerate its course.

Diagnosis.—This is never difficult in advanced cases. The slighter degrees may be more difficult, and the diagnosis is then largely a matter of inference from the association of chronic cough and dyspnoea, with physical signs of hyper-resonance and prolonged expiration.

Confusion may occasionally arise in regard to pneumothorax and pulmonary tuberculosis. Careful record of the symptoms and signs and the investigation of the sputum generally suffice to distinguish these conditions. In doubtful cases the X-rays may assist.

Prognosis.—This depends upon the degree of emphysema and the circumstances of the patient. If progressive, it exerts an increasingly crippling effect, and it certainly shortens life under urban conditions. A "vital capacity" of less than 50 per cent. of the normal is of serious import. The advent of severe bronchitis or of cardiac complications may affect the prognosis gravely.

Treatment.—Emphysema may be arrested but cannot be cured. Attention must be directed to prevention of the causes of chronic cough and increased intra-alveolar tension. In any person with hereditary tendency to emphysema or to winter cough, the questions of occupation and place of residence should be carefully considered. When the disease is established, the patient, if in a position to afford it, should spend the winter in a warm, more equable climate, either abroad or at the south-west coast of England.

Various attempts have been made to increase the respiratory ventilation of the lungs, e.g. by compression of the chest during expiration, by expiring into rarefied air, by breathing compressed air or by expiratory breathing exercises. The patient enters a special iron chamber fitted with a window, and the air pressure is raised during the course of half an hour to $1\frac{1}{2}$ atmospheres. He remains at this pressure for an hour, and is then decompressed to normal during the next half-hour. These baths may be given every other

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day and gradually increased in duration and frequency. This treatment is often helpful in cases of emphysema associated with bronchial spasm or with bronchitis. Cases with marked arterial disease or with much rigidity of the chest-wall are unsuitable for this form of treatment.

The diet should be simple and easily digestible, especially in the later stages. If there is spasmodic dyspnoea or asthma, no late meal should be permitted. Cod-liver oil or halibut liver oil in the winter seems to help some patients. Clothing should be warm, but the excess of under-garments, often worn in fear of chill, is harmful.

In other respects treatment is largely symptomatic. In acute bronchitic attacks the measures to be adopted are in no way different from those in bronchitis uncomplicated by emphysema. In the more chronic bronchitis so commonly present in the winter, iodides with alkalis and balsamic expectorants seem beneficial. Terebene (min. 10) in emulsion or in capsule has been recommended. Counter-irritation to the chest by liniments, such as the lin. terebinthinæ aceticum, is often comforting to the patient, when cough is troublesome. When asthma or paroxysmal dyspnoea occurs, antispasmodic drugs and measures similar to those used in spasmodic asthma may be employed. When cardiac failure supervenes, the appropriate treatment must be vigorously applied. If there is marked cyanosis and venous engorgement, oxygen administration, venesection, leeching, purgative and diuretic drugs may be employed, and digitalis and other cardiac tonics administered. American authorities have suggested the use of an abdominal belt to increase the intra-abdominal pressure and raise the diaphragm.

2. SMALL-LUNGED EMPHYSEMA (ATROPHIC OR SENILE EMPHYSEMA)

Ætiology.—This condition occurs in old age and forms part of the general atrophy of the tissues.

Pathology.—The alveolar walls become thinned and disappear, so that adjacent alveoli coalesce. The condition is primarily atrophic, and therefore differs from true emphysema, although the result is to produce a diminished area for aeration. Post mortem the lungs are small and do not bulge or obscure the pericardium. They are often deeply pigmented, and are more spongy than normal, but although bullæ occur they are small. On section the lung tissue is bloodless and friable. The bronchi may be slightly dilated and show catarrhal changes.

Symptoms.—These are slight and are masked by the enfeeblement due to the general atrophy and debility. There is shortness of breath only on exertion, or on exacerbation of the chronic bronchitis which is frequently present. The chest is small, flat and thinly covered, the movements are poor and there is elevation of the chest as a whole, with poor expansion. Kountz and Alexander maintain that there is very little diminution in vital capacity, that the movements of the diaphragm are increased and that the intervertebral discs are abnormal. There is little cyanosis, and no clubbing. The vocal fremitus is unaltered or slightly diminished. The percussion note is hyper-resonant, but there is no encroachment on the cardiac and hepatic areas of dullness. Breath-sounds are weak, and there is but little prolongation of expiration. Rhonchi and râles may be heard, especially if bronchitis is present, or if the heart is failing.

Diagnosis.—The condition is generally so obvious that no difficulty arises.

Treatment.—This is chiefly a matter of careful regimen and diet, with treatment of coexisting bronchitis or cardiac failure.

3. COMPENSATORY EMPHYSEMA (LOCALISED OR SECONDARY EMPHYSEMA)

Ætiology.—Localised emphysema is a sequel to some process inducing collapse, contraction or destruction of areas of lung tissue. It may be lobular in distribution in bronchitis, broncho-pneumonia, tuberculosis and diphtheria. It may affect one or more lobes, or the whole of one lung, especially in cases of fibrosis following tuberculosis, pneumonia, chronic pleural effusion and empyema.

Pathology.—It is generally conceded that the inspiratory theory of Laennec and Gairdner satisfactorily explains the genesis of this condition. When shrinkage of an area of lung occurs, the chest wall may fall in, if there is pleural adhesion, but otherwise inspiration tends to expand the normal parts of the lungs. None the less, it must be admitted that the expiratory strain of cough may assist in its production.

Although it may be compensatory and physiological at its inception, it is doubtful whether a true hypertrophy takes place after adolescence. In any case it soon leads to atrophy of the alveolar walls, as in true emphysema, and thus becomes pathological and harmful. Post mortem the condition may be found in an upper lobe around contracted scarred lung tissue, or in a lower lobe when the upper lobe is contracted or disorganised. In cases where one lung is fibroid and contracted, compensatory emphysema may be found throughout the sound lung. The resulting adaptations caused by enlargement of one part and shrinking of another may produce some striking displacements, the lower lobe extending upwards nearly to the clavicle, or the anterior margin of the sound lung crossing the mid line. The general appearances are closely similar to those of ordinary emphysema, except that bullæ do not occur, at any rate until the process is advanced and definitely pathological.

Symptoms.—This condition does not produce symptoms that can be differentiated from those of the primary disease. When it affects a lobe or the whole of one lung, there is hyper-resonance over the area involved, which often contrasts strikingly with the dullness due to the primary lesion. The hyper-resonance may extend across the sternum and even for an inch or more beyond it. The heart is displaced towards the side where fibrosis is in progress. Vocal fremitus and vocal resonance are little altered, but may be increased at first and subsequently diminished. In the early stages, when there is alveolar dilatation without degenerative mural changes, the breath-sounds are exaggerated, harsh or puerile, but when such processes develop, they become weak and there are indications of dyspnoea and cyanosis on exertion.

Diagnosis.—This is easy, owing to the difference between the diseased and “compensatory” areas, and to the indications of contraction and displacement.

Treatment.—No special treatment apart from that of the primary condition is required.

4. ACUTE VESICULAR EMPHYSEMA

Although custom has included this condition with emphysema, it is in reality only a temporary acute distension of the alveoli resulting from any condition causing widespread obstruction of the smaller bronchi. It is sometimes observed after death in cases of acute bronchitis, whooping-cough or asphyxia and in anaphylactic shock, and its existence may be inferred in severe asthma. Post mortem the lungs are bulky and the alveoli distended.

The symptoms are dependent upon the primary condition, although dyspnoea is invariably present. The chest is found to be fully expanded, the vocal fremitus is diminished, the percussion note is hyper-resonant, and the breath-sounds vary with the condition inducing it.

5. ACUTE INTERSTITIAL EMPHYSEMA

In acute interstitial emphysema air is present in the stroma of the lungs, and in the subpleural connective tissues. It may follow external trauma, such as fractured ribs, or wounds penetrating the lungs. The alveoli may rupture with violent expiratory efforts, as occur in whooping-cough or influenzal broncho-pneumonia. It may occur in diphtheria. The air sometimes tracks along the pulmonary roots to the mediastinum, and appearing in the neck or on the chest-wall gives rise to surgical emphysema.

Post mortem, subpleural bullæ may be seen containing air, and on section of the lung minute air bubbles may be found in the inter-alveolar connective tissue. A diagnosis cannot be made unless the physical signs of surgical emphysema are present. The air is usually completely absorbed, and a perfect recovery takes place. No special treatment is required beyond keeping the patient at rest, and giving sedative drugs to allay cough.

ABSCESS OF THE LUNG

Definition.—Abscess of the lung includes any circumscribed collection of pus formed in the lung tissue, but softened tuberculous areas and bronchiectatic accumulations are usually excluded.

Ætiology.—*Predisposing causes.*—These include any diseases producing general cachexia or malnutrition, notably diabetes and chronic alcoholism, also any conditions leading to diminished resistance locally in the lung, such as injury, disease or exposure.

Exciting causes.—These are pyogenic organisms, which reach the lung by inhalation, by extension from adjacent suppurative processes, or by the blood stream, either directly or in septic emboli. The common organisms found are streptococci, staphylococci, the pneumococcus, Friedländer's pneumo-bacillus, *Bacillus welchii* and the *Bacillus coli*—sometimes acting in conjunction with putrefactive bacteria. Spirochætes, *Bacillus fusiformis*, treponemata and anaerobic organisms are often present, especially after rupture has occurred. Pulmonary abscess may form under the following conditions:

(1) After inhalation of foreign material into a bronchus. This may be a foreign body, or may occur in association with septic conditions in the nose,

nasopharynx and larynx, or during and after operations in these regions. These are referred to as inhalation abscesses, though some post-operative cases are regarded as due to embolism and not to inhalation. (2) As a result of lobar or lobular pneumonia, especially after the deglutition and aspiration varieties of the latter. Such abscesses are sometimes called metapneumonic. (3) Embolic causes—in pyæmia, or following on septic pulmonary emboli due to right-sided septic endocarditis, or derived from distant septic processes, such as otitis media, and infective thrombo-phlebitis. Amœbic abscess occurs occasionally after dysentery, and pulmonary abscess may be found as a rare complication of enteric fever. (4) From infection of the lung tissue due to spread from adjacent disease. This may occur in bronchiectasis, in ulcerating new growths of the lung, bronchi, œsophagus or mediastinal glands, in caries of the vertebræ or ribs, and in suppurating mediastinal glands. Rupture of an empyema, of a subphrenic abscess, of a liver abscess, or of infected hydatid cysts of the lung or liver may also lead to pulmonary suppuration. Ten per cent. of cases of abscess are due to new growths. (5) As a sequel of perforating chest wounds, or of fractured ribs piercing the lung.

Pathology.—Abscess of the lung is generally single and basic when consequent on pneumonia, whereas embolic abscesses are often small and multiple and may be found in any part of the lung. Abscesses due to extension from adjacent disease are generally solitary, and are often large and irregular. The walls of acute abscesses are generally formed of congested and œdematous lung tissue, or of a zone of unresolved pneumonia. Since acute abscesses commonly rupture quickly into a bronchus, a fibrous capsule is unusual, but in chronic abscess there is often considerable fibroid change in the neighbouring lung tissue. The pleura may become involved over superficial abscesses, leading to empyema, or to pyo-pneumothorax if rupture follows.

Symptoms.—Abscess may develop insidiously, with comparatively slight symptoms. More commonly they are an intensification of those due to the primary or antecedent condition. The patient often appears seriously ill, the fever becomes of septic type, remittent or intermittent in character, and of a high range. Rigors and sweating are common. The pulmonary symptoms at first may be only slight cough with scanty muco-purulent expectoration. Dyspnoea may be present and pain of acute character develops if the pleura is involved. Hæmoptysis occurs in 70 per cent. of cases of abscess. A considerable leucocytosis, up to 20,000 or 30,000 may be found, and occasionally the breath may be offensive, even before rupture into a bronchus occurs, followed by the sudden expectoration of a large quantity of pus. The pus is sometimes unpleasant or offensive-smelling, but has not the extreme fetor of gangrene. Microscopical investigation will demonstrate the presence of pulmonary debris, especially elastic tissue, together with pus cells and micro-organisms. After the expectoration of the pus, the temperature usually falls and the general condition of the patient is much improved, though cough and expectoration persist. In chronic cases after rupture the temperature may become irregular and periodic, a few days of normal temperature being followed by a period of fever and later by increased expectoration. The physical signs in a deep-seated or small abscess are often inconspicuous, and comprise slight dullness over a small area, weak breath-sounds and possibly a few râles in the surrounding infiltrated or œdematous lung tissue. With a large or a

superficial abscess, the signs before rupture may be those of consolidated or collapsed lung. After evacuation occurs, the characteristic signs of excavation usually develop at once. In multiple embolic abscesses the signs are usually those of disseminated broncho-pneumonia.

Complications and Sequelæ.—The commonest complication is dry pleurisy. This may progress to empyema, or to pyo-pneumothorax, if rupture into the pleura occurs. In some cases mediastinitis or pericarditis may develop. Gangrene is described, but is a rare sequel. Metastatic abscesses may be produced in other parts of the body, especially in the brain, and meningitis is a rare and serious complication. The most important sequelæ are fibrosis of the lung, with bronchiectasis, pleural adhesion, and rarely indurative mediastinitis.

Diagnosis.—This is difficult before rupture, but abscess may be suspected from the gravity of the symptoms in relation to the history and signs, especially if leucocytosis and fetor of the breath are present. X-ray examination may be helpful, by demonstrating a localised shadow before rupture, and excavation afterwards, and also by establishing the situation of the abscess. A fluid level can often be seen in films taken in the erect position. The sudden expectoration of pus, followed by retrogression of symptoms and signs of excavation is very suggestive of abscess. After rupture has occurred the differential diagnosis has to be considered from:

1. *Interlobar empyema.*—This may be very difficult or even impossible. In this condition the signs are generally most marked in the region of an interlobar septum, there may be some cardiac displacement, and the sputum, though purulent, does not contain elastic tissue.

2. *Bronchiectasis.*—The history, the characteristic cough and sputum, and the variation of the physical signs with the state of the cavity usually suffice to distinguish this condition. An X-ray examination after lipiodol or neo-hydriol will distinguish in doubtful cases, since they do not as a rule enter the abscess cavity and the appearances in bronchiectasis are characteristic.

3. *Gangrene of the lung.*—The distinction is not always easy in acute abscess, since the two processes are closely related. The extreme gravity of the patient's general condition and the horrible fetor of the breath and sputum are the most characteristic features of gangrene.

4. *Tuberculous excavation.*—The history, the distribution of the signs, and the characters of the sputum, including the presence of tubercle bacilli, are the distinguishing indications.

5. *Purulent bronchitis.*—The history, the widespread physical signs, and the absence of elastic tissue from the sputum usually serve to establish the diagnosis, and lipiodol or neo-hydriol investigations may be helpful.

In multiple or pyæmic abscesses, it is often impossible to recognise the condition, though it may be suspected from the severity of the symptoms and signs. In any doubtful case an X-ray examination or tomography should be carried out, if the condition of the patient permits. The possibility of malignant growth as a cause of abscess should be borne in mind and, when necessary, bronchoscopy as well as lipiodol investigation carried out. Exploratory puncture as a means of diagnosis is dangerous and should be avoided.

Prognosis.—The prognosis, though grave in many cases, is better than

might be anticipated. Many of those in which rupture into a bronchus occurs recover. Death is inevitable in the pyæmic cases.

Treatment.—(1) *Medical.*—In acute abscesses medical measures should be employed, since in a considerable proportion of cases, recovery may occur after rupture, especially when the abscess is in the upper lobe. Before rupture the treatment should be similar to that for acute pneumonia. After rupture, evacuation should be promoted by “tipping,” or postural drainage. The Nelson bed is of great value in this connection when the relation of the abscess to the nearest patent bronchus has been determined by X-ray examination. Expectorant drugs and antiseptics, such as creosote, should be employed. Antiseptic inhalations may also be used on a Burney-Yeo mask, as for bronchiectasis, or creosote vapour baths may be given. In like manner intratracheal injections of menthol, guaiacol and olive oil have been employed with benefit. In cases with spirochætes, treponemata and anaerobic organisms in the sputum, intravenous injections of salvarsan or neo-arsphenamine may be administered with benefit.

(2) *Surgical.*—If spontaneous rupture does not occur after the abscess has become localised and encapsulated, operation is indicated in order to prevent the walls becoming thick. Repeated bronchoscopic aspiration is sometimes employed after rupture, and at times gives satisfactory results. If, after rupture, there is not satisfactory progress towards cure within 6 weeks, clinically and radiologically, surgical treatment should be considered. Thoracotomy and open drainage is the operation generally employed. This is now usually carried out in two stages: (a) a preliminary exposure of the pleura by rib resection and packing with gauze, to ensure adhesion of the pleura; (b) some days later the abscess is opened along the course of an exploring needle. Artificial pneumothorax has been recommended, especially in deep or centrally placed abscesses. There is a risk of rupture into the pleura, more particularly when re-expansion of the lung is permitted. For this reason, this form of treatment is rarely advisable. Phrenic evulsion may aid in the evacuation of a chronic abscess, either alone or after thoracotomy. Pneumolysis, lobectomy, and thoracoplasty are also used in the treatment of chronic abscess. Lobectomy seems likely to be the most satisfactory in chronic cases in which it is practicable.

GANGRENE OF THE LUNG

In this condition localised or diffuse areas of lung tissue undergo putrefactive necrosis.

Ætiology.—*Predisposing causes.*—These include old age, over-indulgence in alcohol, general debility, diabetes and insanity. In certain rare cases, especially after broncho-pneumonia complicating measles, gangrene of the lung is met with in children.

Exciting causes and associated conditions.—These are, in the main, identical with those of pulmonary abscess (see p. 1183). In addition, the pressure of aneurysm or of new-growth on branches of the pulmonary artery may lead to gangrene. The causal organisms are also very similar to those found in abscess of the lungs, and include staphylococci, streptococci, sarcinæ, the *Micrococcus tetragenus*, the *Bacillus coli communis*, the *B. pyocyaneus*, the

B. fusiformis with its associated spirochæte, and various anaerobes. In some instances acid-fast bacilli, classed as streptothrices, occur. Some of these organisms yield putrefactive products, with the liberation of phenol, indole and skatole compounds in the lung.

Pathology.—It is not quite clear what are the factors determining whether abscess or gangrene occurs in an infected area of lung. Doubtless the general resistance of the body, the degree of local vascular disturbance, and the virulence of the infecting organisms all play their part. Laennec first described the two varieties of gangrene, the circumscribed and the spreading or diffuse. Around the former there are indications of a line of demarcation, formed by congested lung tissue, which may present the appearance of red hepatisation. The surrounding lung tissue is invariably somewhat œdematous. The gangrenous area is soft and pulpy, and its colour varies from reddish-brown to greenish-black. As the necrosis advances, putrefactive liquefaction occurs, with the formation of a horribly reeking fluid, containing shreds and masses of necrotic lung tissue. When this is discharged, excavation results, and isolated vessels may be seen running across the resulting cavity, the walls of which are rough and covered with fetid pus. The diffuse variety of gangrene is less common; there is no attempt at a zone of demarcation, and the whole of a lobe or of one lung may be affected. In both forms, the overlying pleura is intensely inflamed, and empyema or pyo-pneumothorax may be produced.

Symptoms.—These are similar to those occurring in abscess of the lung, but are more acute. The patient is desperately ill, rigors are more common and sweating is more profuse. The breath has a peculiar fetor, which, on account of the presence of the skatole group of putrefactive products in the gangrenous lung, has an almost fecal odour. The sputum is intensely offensive, and on standing separates into three layers, similar to those of the expectoration in cases of bronchiectasis. Elastic tissue is usually present, but it may undergo rapid disintegration. Hæmoptysis is not infrequent and may prove fatal. In rare cases gangrene is not accompanied by fetid expectoration, especially when developing in the insane, in young children, and in diabetics, or after pulmonary embolism. The physical signs closely resemble those present in cases of pulmonary abscess, and are those of consolidation before liquefaction occurs, and of excavation afterwards. The signs of the antecedent condition such as bronchiectasis, aneurysm, or malignant disease may also be present.

Complications and Sequelæ.—These are similar to those met with in pulmonary abscess, but owing to the rapid course and greater fatality of gangrene, they are not so common. Cerebral abscess may occur.

Course.—The course is usually rapid, unless the diseased area is small and circumscribed. In rare cases of localised gangrene of small extent, resolution and subsequent fibrosis occur.

Diagnosis.—The differential diagnosis is as for pulmonary abscess, the distinguishing features being the extremely critical condition of the patient and the revolting fetor of the breath and expectoration. X-ray examination may give great assistance if the patient's condition permits it to be made.

Prognosis.—This is always extremely grave, though a few cases of localised gangrene recover spontaneously. The prognosis is improved by

early operation in suitable cases. The outlook is said to be worse if the condition is apical, and diffuse gangrene is invariably fatal.

Treatment.—Operation is indicated when the general condition of the patient permits, if the gangrenous area can be localised by physical signs or X-ray examination. Exploratory puncture should not be carried out. The other operative procedures are similar to those for abscess of the lung. Operation is contra-indicated in cases of diffuse gangrene. The medical treatment is in all respects similar to that for pulmonary abscess. Injections of neo-arsphenamine, in doses of 0.3 g., have given good results, especially in cases due to fuso-spirochaetosis.

PULMONARY FIBROSIS

Synonyms.—Fibroid Disease of the Lung; Chronic Interstitial Pneumonia; Cirrhosis of the Lung.

Definition.—Pulmonary fibrosis is a late sequel of many acute and chronic inflammatory or irritative processes affecting the bronchi, lungs and pleurae. It is therefore rather of pathological than of clinical interest, and in no sense constitutes a separate disease, although the end-results are remarkably similar in different forms. It is described here partly in deference to tradition, and partly to point out the methods of diagnosis between the various causes producing such strikingly similar effects.

Ætiology.—(1) The commonest cause is pulmonary tuberculosis, particularly the fibroid and fibro-caseous varieties. (2) The group of pneumoconioses contributes a considerable number of cases, and possibly some varieties of gas poisoning may induce fibroid changes. (3) Bronchopneumonic processes, particularly the forms associated with measles and whooping-cough, may be followed by widespread fibrosis, especially in children. (4) Although fibroid induration is commonly described as a sequel of lobar pneumonia, this disease is one of the rarer causes. (5) Localised fibrosis may occur around any circumscribed pulmonary or bronchial lesion, such as that produced by syphilis, leprosy, glanders or streptotrichosis. Similarly it occurs about infarcts, pulmonary abscesses and parasitic cysts. (6) Chronic venous congestion, if prolonged, leads to fibroid change, which is referred to as "brown induration." This is usually of moderate degree and does not affect the clinical manifestations. (7) Chronic pleural affections, particularly those leading to adhesions or causing pulmonary collapse, may induce fibroid changes within the lung, and these forms are described as "pleurogenous cirrhosis." (8) Any condition causing obstruction of a bronchus and leading either to collapse or to bronchiectasis may, if long continued, cause fibrosis of the corresponding lung area. Among such may be mentioned inhaled foreign body, new-growth, cicatricial contraction and thoracic aneurysm.

Pathology.—The fibroid overgrowth may be: (1) Massive or lobar; (2) localised or insular; (3) peribronchial; and (4) reticular.

Any part of the connective tissue framework of the lungs and bronchi may contribute to the fibrosis. In the massive form, which generally affects the whole or the major part of a lobe or even of one lung, the appearances in cases due to tuberculosis differ from those due to other causes. In the

tuberculous variety the primary distribution is usually apical, and evidence of other tuberculous processes may be apparent in the form of large or small dried-up cavities, inspissated caseous material or calcareous masses enclosed in fibrous strands. In non-tuberculous processes, the early localisation is commonly basic, and although the primary cause may be obvious in the form of bronchial obstruction or some pleural condition, this is not always the case. On the other hand, non-tuberculous processes may involve the upper lobe primarily and fibroid tuberculosis may fall with special stress upon the lower lobe. In both forms of fibrosis, bronchiectasis may result, although this is more common in the non-tuberculous cases. Apart from the special tuberculous lesions, the end results are very similar in both forms. The affected area of the lung is shrunken and often devoid of air except for that in the bronchi and in the cavities. It is dark in colour, very firm and hard. On section it presents a mottled appearance owing to the strands of blue-grey fibrous tissue traversing it, contrasting with the pigmented, condensed, airless lung tissue. The fibroid area may be honeycombed by cavities or may present one large excavation, due either to tuberculous cavitation or to bronchiectatic dilatation. There is nearly always thickening and adhesion of the pleura. The contraction of the abnormal fibrous tissue leads to marked displacement of the heart and mediastinum.

The localised form is commonly due to healed tuberculous processes at an apex. There may be simple puckering with or without pleural thickening and adhesion, or a dense mass enclosing dried-up caseous matter or calcareous spicules. In bronchitic or broncho-pneumonic processes a patchy fibrosis may occur, described as insular fibrosis by Fowler.

Reticular fibrosis is a rare condition in which the fibrous tissue in the interlobular septa seems to become increased as well as that around the bronchi. It is at present only of pathological interest.

Symptoms.—The symptoms of pulmonary fibrosis are, in the main, expectoration and dyspnoea together with those of the primary affection. In the non-tuberculous cases, bronchiectasis is so frequently associated that the symptoms and signs found are practically those of this condition. Even in tuberculous cases, some degree of bronchial dilatation is the rule, although the sputum is rarely offensive. The cough is generally periodic and associated with change of posture. The expectoration is abundant, and if bronchiectasis is present, it has the usual characteristic features. The dyspnoea is proportional to the extent of lung involved. It may be extreme in the later stages, when the heart becomes embarrassed and begins to fail. Fever is usually absent, except when complications occur.

The patients are generally spare, although nutrition may sometimes be well maintained until late. They may show signs of deficient aeration in duskiness, cyanosis and congested cheeks. Polycythæmia of some degree is the rule. Clubbing of the fingers is almost constant. Evidence of contraction is generally forthcoming in the flattening and retraction of the affected side, with the dropped shoulder and compensatory spinal curvature. Movement is greatly restricted, contrasting with the increased expansion of the other side. The cardiac impulse is sometimes much displaced, especially in left-sided cases, when it may be in the left posterior axillary line or even under the angle of the scapula. In right-sided cases, it is drawn

to the right of the sternum, even sometimes under or outside the right nipple. Vocal fremitus is usually diminished and percussion gives dullness of varying degree over the fibroid area, while the unaffected parts may be hyper-resonant from "compensatory" emphysema. The diaphragm may be drawn up, and the liver or stomach correspondingly displaced. The breath-sounds are often weak or inaudible unless there is bronchiectasis or cavitation, when the characteristic signs of these conditions can be recognised. The vocal resonance is diminished if there is much pleural thickening, increased if cavities are present. Adventitious sounds may be entirely absent, and when present vary from rhonchi and bubbling râles to coarse metallic râles, according to the presence or absence of excavation. X-ray examination gives useful confirmation, showing displacement, excavation and pleural thickening.

Course.—The course is invariably chronic, and may extend to years, even ten or twenty.

Diagnosis.—The diagnosis is usually easy. The evidence of contraction and of mediastinal displacement towards the affected side, especially if signs of cavitation are also present, is highly suggestive. In the absence of the cavitation some difficulty may arise in regard to chronic pleural effusion or empyema. In the earlier stages the contra-lateral displacement of the cardiac impulse should prevent any mistake, but where partial absorption has occurred, this may be very slight or absent. In such cases an exploratory puncture or an X-ray examination may be helpful.

When the diagnosis of pulmonary fibrosis has been made, the differentiation of the cause is an essential to prognosis and treatment. If the condition is apical, there is a presumption in favour of tuberculosis; if basilar, some other cause is more probable. Repeated examinations of the sputum should be made for tubercle bacilli, and if these prove negative, X-ray examination may reveal some cause such as new-growth, aneurysm or even foreign body. In some cases a careful consideration of the history may afford a clue to the diagnosis.

Prognosis and Treatment.—These depend upon the primary condition, but in most cases the latter is mainly symptomatic.

PNEUMOKONIOSIS

Synonyms.—Pneumonokoniosis; Dust Disease of the Lung.

Definition.—Pneumokoniosis comprises all the pathological changes induced in the bronchi, lung and pleuræ by the inhalation of dust particles.

Ætiology.—*Predisposing causes.*—Pneumokoniosis is one of the occupational diseases. It is practically limited to men, and usually develops between the ages of 25 and 40. Defective ventilation, bad hygienic conditions and alcoholism promote its incidence.

Exciting causes.—Various forms of dust, both inorganic and organic, serve to produce pneumokoniosis, and in general the harder and more gritty the particles, the more marked are the changes induced. Organic forms of dust lead especially to bronchitic changes, the inorganic forms to pulmonary fibrosis.

The following varieties are recognised :

1. *Anthracosis* from coal dust (coal-miners' phthisis).
2. *Siderosis* (silico-siderosis), from the inhalation of fine particles in tin, copper, lead and iron miners, and in grinders of steel goods (grinders' rot).
3. *Silicosis* or chalicosis, met with in workers in quartz, gannister and slate quarries, also in potters (quarts-miners' phthisis, and potters' asthma). Gold-miners' phthisis, the most serious form of pneumokoniosis, and especially prevalent in the South African gold mines, is due to the fine dust caused by the rock drills.

4. *Byssinosis*, a rare variety, is met with in cotton workers, felt-hat makers, and the employees in shoddy mills.

5. *Asbestosis*.—A condition found occasionally in those working in the manufacture of asbestos articles. Asbestos is composed of compound silicates of iron and magnesium.

Pathology.—The lungs of persons living under rural conditions are practically free from deposited pigment. A certain amount of carbon is invariably present in the lungs of town-dwellers, giving them a dark-grey mottled appearance, but producing no pulmonary fibrosis. In coal-miners this occurs to such an extent that the lungs are black (anthracosis), although even here little fibrosis occurs, except in miners of hard coal or anthracite. In siderosis and silicosis, fine sharp particles of metallic oxides or silica are deposited in the lung tissue. According to Stewart, siderosis is in effect silico-siderosis, the damaging agent being silica inhaled at the same time. In asbestosis, curious irregular discoid structures of golden yellow colour and containing iron, now called "asbestosis bodies," are found in the lungs and in the sputum. There is also much fibrosis in pneumokoniosis, and tuberculosis is liable to be a later development.

It is generally accepted that these particles are conveyed to the bronchi and alveoli by inhalation. In normal breathing, most of the coarse particles are detained in the nose, and are discharged with the nasal mucus, whereas in mouth-breathers they readily gain access to the trachea and bronchi. Even then, the coarser particles may be discharged, in the expectoration through the agency of the ciliated epithelium, but, owing to the catarrhal processes induced by the irritation of the inhaled dust, this epithelium may be desquamated and the absorption of the particles is promoted. As a further consequence of this initial bronchitis, the finer particles may reach the alveoli, and passing between the epithelial cells, gain access to the tissue spaces, or in some cases they may be taken up into special "dust cells." In silicosis, particles of crystalline silica become deposited in the connective tissue, and chronic peribronchial and perialveolar fibrosis develops. The bronchial glands also become enlarged by the deposition of similar particles conveyed by the lymphatics. Other changes more or less constantly present are emphysema, pleural adhesion and bronchiectasis.

The relationship to tuberculosis has been much debated. It is now established that pneumokoniosis is non-tuberculous in origin, and that it may remain so throughout its course. On the other hand, certain forms undoubtedly favour the development of tuberculosis. In England and Wales coal-miners suffer less from tuberculosis than do all other males. On the other hand, gold-miners are extremely liable to it. It would appear that the determining factor is the presence of particles of silica. Silicates, as

in clay, do not induce tuberculosis. Workers in freestone develop this disease, limestone workers do not. Slate quarries do not acquire tuberculosis very readily, while metalliferous miners working in quartzite very frequently suffer from it. The Miners Phthisis Bureau recognises two types of silicosis. (1) Simple silicosis, the damage found being due to dust alone. It is non-progressive if exposure ceases. (2) Tuberculo-silicosis, in which most of the damage is due to dust, and tuberculosis is secondary. Haldane and Mavrogordato demonstrated that particles of coal are absorbed by the "dust cells" whose movements are thereby stimulated, with the result that they appear in the black spit, which is therefore a healthy sign. The particles of silica are also absorbed by these "dust cells," but no stimulus to their movement is induced and they remain *in situ*. Kettle and Gye have shown that a silica colloid is slowly formed, which leads to breakdown of the tissue defences and thus favours the activity of tubercle bacilli.

Post mortem, the lungs are generally firm and pigmented, the colour varying with the cause, being black in anthracosis, reddish-brown in siderosis, and greyish-black in silicosis. The pleura is generally adherent, especially at the bases. On section the lungs are firm, and often gritty. Small hard nodules may be felt with the finger. Fibroid changes are especially marked in silicosis. The bronchi are inflamed and sometimes dilated. Some degree of emphysema is usually apparent. If tuberculous lesions are also present, these vary from fibroid areas to miliary nodules. Destructive processes resulting in cavitation may also be seen. Microscopically, the alveolar walls are thickened, the connective tissue is everywhere found to be increased, the "dust cells" may be seen in the connective tissue or in the alveoli, and particles of pigment or silica are found widely deposited in the connective tissue cells.

Symptoms.—The onset is insidious, bronchial irritation and cough, especially in the morning, may be the first indications, but increasing shortness of breath and debility are frequently early symptoms. The expectoration, at first scanty and mucoid, becomes more abundant and may present characteristic features as in the "black spit" of anthracosis. Tinging of the sputum and later hæmoptysis occur, but these suggest the possibility of superadded tuberculosis. The patient may appear healthy and be but little troubled except by the shortness of breath, but later emaciation and an appearance of premature old age are not uncommon.

The physical signs are not characteristic; at first they are simply those of persistent bronchitis, then emphysematous changes may become apparent. Later, signs of fibrosis appear, very similar to those described in the preceding section. Even when tuberculosis develops the signs are often but little characteristic, and repeated sputum tests may be necessary to establish the diagnosis. Examination by X-rays may be helpful; at first there is an increase in the reticulation and later nodulation, somewhat like that of miliary tuberculosis. At a still later stage, the nodules become larger, and there is increased fibrosis. The changes characteristic of tuberculosis may be super-added.

Complications and Sequelæ.—The most important complication is tuberculosis, which forms the terminal stage of many cases of silicosis. This may be suspected when fever, night sweats, hæmoptysis or emaciation develop. Bronchiectasis of considerable degree sometimes results as a

consequence of the fibrosis, and leads to the symptoms and signs characteristic of that condition.

Course.—This is progressive, unless the sufferer is removed from the exciting causes. Anthracosis runs a very chronic course, siderosis somewhat less so, while gold miners only live a few years (5 to 6—Oliver) after the onset of the disease.

Diagnosis.—The diagnosis can usually be made from the history of shortness of breath, cough and expectoration, developing in a worker in a dusty occupation. In the early stages, cigarette-smoker's cough and bronchitis may give rise to difficulty. In the later stages, the possibility of a primary fibroid tuberculosis has to be considered.

Prognosis.—This is unfavourable except in anthracosis. Lyle Cummins suggests that the finely divided carbon particles absorb the toxins of the tubercle bacillus. If recognised early, and if the patient is taken from the dusty conditions, recovery may be anticipated. The development of tuberculosis affects the outlook very gravely.

Treatment.—*Prophylactic.*—Every means should be adopted to avoid the dusty conditions leading to the disease. Mines should be well ventilated, and respirators should be worn where practicable in dusty occupations. Factories and workshops should be provided with apparatus to draw away dust. Sprays or jets should be used with drills to moisten the dust produced.

Curative.—Directly the condition is diagnosed, the patient should be advised to change his occupation. Nutrition should be kept at a satisfactory level. Symptoms and associated conditions, such as bronchitis or tuberculosis, should be treated on general principles.

PULMONARY TUBERCULOSIS

Synonyms.—Phthisis ; Consumption ; Decline.

Pulmonary tuberculosis embraces all the abnormal conditions induced by infection of the lungs, pleuræ and bronchial glands with the tubercle bacillus.

Ætiology.—**PREDISPOSING CAUSES.**—**Age.**—The maximum age incidence is between the 15th and 45th years, although the disease may be encountered at any age. Senile tuberculosis is more common than is generally recognised.

Sex.—The disease is more frequent in males, but between the ages of 5 and 15 the female sex shows a preponderance.

Heredity.—Pulmonary tuberculosis certainly occurs with undue frequency in certain families. Since the direct transmission of the tubercle bacillus to the infant is extremely rare, two explanations seem possible—(1) Children born of tuberculous stock may inherit an increased susceptibility or diminished resistance, the tuberculous diathesis ; or (2) they may contract tuberculosis on account of their exposure to massive infection in early life.

Race.—Differences in racial susceptibility probably depend upon the degree of inherited resistance acquired by the race from infection of previous generations. Native races suffer severely when first exposed. In Europe the Irish are particularly susceptible, whereas the Jews are relatively immune.

Climate.—Tuberculosis occurs in all climates. The prevalence of strong

rainy winds and defective subsoil drainage may tend to increase its incidence.

Occupation.—The highest mortality from tuberculosis occurs in England amongst the workers in dusty occupations, thus Cornish tin miners head the list. On the other hand, coal miners are notably free from the disease. Any conditions leading to overwork or to underfeeding increase the liability to tuberculosis.

Environment.—Overcrowding, defective sanitation, dampness, dirt, lack of sunlight and insufficient ventilation are most potent factors in the spread of the disease, causing both lowering of the resistance and increased facilities for direct infection.

Trauma.—Trauma, involving the chest-wall, may be followed by active pulmonary tuberculosis. This is probably because the injury leads to activity of previously arrested disease, rather than to fresh infection at a spot of lowered resistance.

Gassing.—In certain cases the inhalation of poison gases causes rapid activity and spread in latent disease, or it may possibly prepare the ground for reinfection, but it is not a factor of great ætiological importance.

The influence of other diseases and conditions.—The following diseases predispose to the development of pulmonary tuberculosis: measles, especially when complicated by broncho-pneumonia, whooping-cough, influenza, pneumokoniosis, alcoholism, diabetes, syphilis, congenital heart disease and insanity. Tuberculosis may manifest itself for the first time during prolonged lactation or after repeated pregnancies; when previously existent it often remains quiescent during pregnancy, but it may spread rapidly after childbirth. Contrary to the usual belief, pulmonary tuberculosis not infrequently coexists with mitral stenosis. Cases apparently following pneumonia, pleurisy or bronchitis are usually tuberculous from the onset.

EXCITING CAUSES.—The causal organism is the *Bacillus tuberculosis*, discovered by Koch in 1882. It exists in four main forms, human, bovine, avian and reptilian; only the two former usually occur in man, but avian infection has been recorded. The human type is found in over 97 per cent. of pulmonary tuberculous lesions, though a higher proportion of the bovine type has been found in Scotland. In glandular tuberculosis up to the age of 5 years, over 80 per cent. of the bacilli isolated conform to the bovine variety. In tuberculosis of bones and joints up to the same age, 29 per cent. of the cases are of bovine origin.

The bacilli may gain access to the body by inhalation, by alimentary ingestion, through the tonsils, through the skin, or possibly, in rare instances, by hereditary transmission. It is probable that in the majority of cases of pulmonary tuberculosis in adults, the organisms are carried direct to the lungs in the inspired air, and Ghon showed that in children, who had died of tuberculosis of the lungs, a primary focus was present in the lungs in 92·4 per cent. As, however, extensive tuberculous lesions are frequently found in the bronchial glands in cases of pulmonary tuberculosis, it is believed by some that the glands are primarily affected, and that the bacilli pass from them to the lungs, either against the lymphatic flow or in the blood stream. Calmette and others have demonstrated that the bacilli may gain access to the bronchial glands from the alimentary tract through the

thoracic duct, or from the tonsils through the cervical and mediastinal glands. Cases have been recorded in which primary cutaneous infections have been followed later by active pulmonary tuberculosis. Direct transmission of the tubercle bacillus is believed to occur only when the mother is suffering from advanced tuberculosis, and even then is of great rarity.

The incubation period tuberculosis of is uncertain, owing to the difficulty in determining when infection takes place. It is now believed by many authorities that the majority of individuals are originally infected in infancy or early childhood, either from the ingestion of tuberculous milk, or by the inhalation of bacilli from dried sputum. Pulmonary tuberculosis is thus regarded as a late manifestation comparable with the tertiary stage of syphilis. On this hypothesis, active pulmonary disease in adult life may result either from reinfection or from the activation of a dormant lesion in the body. As the organisms found in early life are frequently of the bovine type, whereas in pulmonary tuberculosis they are almost invariably of the human variety, it is probable that reinfection is the more common, since mutation of type has not so far been proved.

Provided that the proper precautions are taken, the risk of infection from adult to adult is not great, and only exists in "open" cases of tuberculosis, *i.e.* in cases with tubercle bacilli in the sputum. The occurrence of conjugal disease, which is less common than might be expected, has been explained by mating of those with hereditary diathesis.

Pathology.—The earliest lesion in the lung is the formation of tubercles, whose structure is described in the general article on tuberculosis. They usually appear first near the apex. This may be due to the relative immobility of this portion of the lung, possibly as the result of calcification of the first costal cartilage (Freund), but in some cases the bacilli may spread from the cervical to the supraclavicular glands and thence to the adjacent lung. In some cases the earliest lesion is found in the subclavicular region well below the apex. It may commence in a subacute manner. In such cases, an area of localised deposit may be seen on radiological examination—known as Assmann's focus (Redecker's "*früh infiltrat*"). The initial deposit is usually in or around the small bronchioles of the third to fifth degree (Hirschfeld's bronchioles). The inflammatory swelling of the bronchioles obstructs their lumen, leading to collapse of the alveoli beyond and the formation of bronchopneumonic areas. At the same time peri-bronchiolar inflammation develops. In children there is, in the majority of cases, a primary lung focus (Ghon's focus), with secondary deposits in the bronchial glands.

SECONDARY CHANGES.—1. *Caseation.*—The tubercle is avascular, and owing to this, and possibly also to the action of tubercle toxins, coagulation necrosis and fatty degeneration frequently ensue. This combined process is known as caseation and results in the formation of a structureless, cheesy mass. Further changes may now occur, either softening, with the development of a "cold abscess" filled with tuberculous "pus," or calcification, with the subsequent formation of gritty masses known as "pneumoliths."

2. *Cavitation.*—Cavities result from the liquefaction of caseous areas, and the expectoration of the resulting debris. They may be no larger than a pea, or may occupy the whole of one or more lobes. A recent cavity has an irregular outline, with rough, shaggy walls and a vascular line of demarcation. It is often traversed by trabeculae, formed by bronchi and vessels

which may be partly or completely obliterated, while sometimes the trabeculae consist of condensed lung tissue which originally separated adjacent cavities. In chronic cases, the cavity is surrounded by fibrosed lung tissue forming a pseudo-capsule, and its interior becomes lined by a thin, smooth, false membrane. Small aneurysms may be found, arising either from vessels running in the walls or in the trabeculae of the cavity, the former being the more common. In some cases, where hæmoptysis has occurred, rupture of such an aneurysm is the cause.

3. *Fibrosis*.—Reactive changes in the lung stroma lead to the formation of fibrous tissue. This may occur early or after caseation has taken place. Such changes are often classified as productive, whereas infiltration and the earlier reactive changes are referred to as exudative.

In the majority of deaths from all causes, old tuberculous lesions are found post mortem near the apex of one lung. These consist of small nodules of arrested disease, with thickening and dimpling of the adjacent pleura.

DISSEMINATION IN THE LUNGS.—The disease may spread from the primary peri-bronchial deposit—(a) By direct infiltration; (b) By the peri-bronchial lymphatics and capillaries, leading to a racemose appearance or to peri-bronchial fibrosis; (c) By the subpleural and interstitial lymphatics, with localised miliary dissemination; (d) By inhalation into a bronchus of tuberculous material, which is then carried to other parts of the same or to the opposite lung—this not infrequently happens after hæmoptysis and in cavitation; (e) By the blood vessels, *e.g.* generalised miliary tuberculosis may result from erosion of a caseous tubercle into a vein.

The pathology of the clinically distinguishable forms of pulmonary tuberculosis will now be described.

1. ACUTE MILIARY TUBERCULOSIS.—A primary caseous focus may be discovered at the apex of one lung, in the bronchial glands, or at some distant spot in the body. Local erosion of a vein may be found, accounting for the dissemination of the disease. The lungs are usually studded with minute grey tubercles, the smaller ones requiring a hand lens for their recognition. In very acute cases death occurs before any secondary broncho-pneumonic changes take place. Miliary tuberculosis may develop as a terminal event in chronic fibro-caseous or fibroid tuberculosis. The tubercles are then found in large numbers around the old foci of disease, but to a less extent in the more remote portions of the lung.

2. CHRONIC MILIARY TUBERCULOSIS.—The lungs are studded uniformly with firm nodules varying in size from one to several millimetres in diameter. They are grey or white, project from the cut surface of the lung, and in some cases are calcified. There may be, in addition, a diffuse fine fibrosis. Miliary tubercles are at times found scantily distributed in the spleen, kidneys and liver. Not infrequently there is evidence of terminal acute miliary tuberculosis involving the brain and meninges.

3. ACUTE CASEOUS TUBERCULOSIS.—Large areas of consolidation form rapidly, which differ histologically from the common chronic tuberculous broncho-pneumonia in that the alveolar exudate is more definitely inflammatory and contains fibrin. In the rare lobar cases, the rapid caseation and the presence of tubercle bacilli show that the caseous pneumonia is a specific process. Firm yellowish patches, which may be confluent, are seen,

usually scattered throughout both lungs. The affected areas are airless and sink in water. Softening is generally present in varying forms up to actual cavity formation, which may be extensive, involving even a whole lobe.

4. **FIBRO-CASEOUS TUBERCULOSIS.**—This is the commonest variety of the disease; the appearances of the lung vary with the relative preponderance of the caseous and fibrotic changes. The early lesions are miliary or bronchopneumonic, but areas of caseation in varying stages, including cavitation, are almost always present. The older lesions show considerable fibrosis, the strands of sclerotic tissue being pigmented and glistening. The earliest lesion is usually near the apex of the upper lobe at the back, more rarely a little lower and towards the front. The apex of the lower lobe is next affected, and the disease then spreads in the direction of the interlobar septum; the apex of the upper lobe of the opposite lung is next involved (Fowler's law of spread). Pleural adhesions are usually present over the oldest lesions, and in the interlobar fissures. An open cavity, from which infected sputum is discharged, is a danger, being a frequent cause of spread.

5. **FIBROID TUBERCULOSIS.**—Fibrosis may be localised around a small arrested lesion, or may spread throughout a lung in which caseation or excavation has occurred. One lobe or the whole lung is then contracted and firm. In the interstices of the fibrous tissue, which is usually pigmented, inspissated caseous material, calcareous patches, or cavities are seen. The shrinkage may lead to bronchiectasis, especially in the lower lobes. The overlying pleura is much thickened and adherent, and the mediastinum is drawn over towards the affected side. The opposite lung, or the sound portions of the fibrosed one, may show compensatory emphysema.

The bronchial glands.—The tracheo-bronchial glands are affected in all forms of pulmonary tuberculosis. They are enlarged, sometimes pigmented, and may present miliary, caseous, calcareous or fibroid changes, in some cases primary, in others secondary to the lesions in the lungs.

The pleura.—This, too, is almost constantly affected. The commonest changes are an early dry pleurisy, and a later thickening with adhesions which may completely unite the visceral with the parietal layers. In acute disease or active spread, the pleura may be studded with miliary tubercles, leading to a large serous effusion.

The post-mortem appearances of the lesions situated in the other organs, found as complications of pulmonary tuberculosis, are described in the respective sections dealing with them, and include tuberculous meningitis, peritonitis, enteritis and genito-urinary tuberculosis. There is usually atrophy of the skeletal muscles, sometimes lardaceous and fatty degeneration of the liver, and hypoplasia with fatty degeneration of the heart.

Symptoms.—The symptoms fall into three groups (Pottenger)—(1) pulmonary, such as catarrh, expectoration, hæmoptysis and pleurisy; (2) reflex, such as pain, cough and laryngeal irritability; (3) toxæmic, including malaise, tachycardia, pyrexia and loss of weight.

ONSET.—The mode of onset is very variable, but certain forms can be recognised.

(a) *Insidious.*—The early symptoms may be malaise, anæmia, amenorrhœa, cardiac irritability, progressive loss of weight, and slight rise of temperature, generally towards evening. Cough and expectoration often appear only when the signs in the chest are quite apparent. When there is intestinal

stasis, the cutaneous pigmentation may suggest the diagnosis of Addison's disease.

(b) *Catarrhal*.—A series of febrile "colds" may usher in the disease, and such a sequence is always suspicious.

(c) *Phthisis ab hæmoptoe*.—Hæmoptysis may first draw attention to the lungs. It may be slight, and is then due to early congestion around the focus of infection. If it is more marked, it indicates breakdown of an old arrested lesion, or may afford dramatic evidence of extensive disease which had not been recognised previously.

(d) *Laryngeal*.—Hoarseness or aphonia may be the first symptom, but laryngeal tuberculosis is usually secondary to pulmonary disease, although the latter may have been unsuspected.

(e) *Gastro-intestinal*.—Anorexia and flatulence often occur early. When they are accompanied by slight loss of weight and pyrexia, the possibility of pulmonary tuberculosis should be suspected.

(f) *Pleural*.—Dry pleurisy is a frequent manifestation of latent pulmonary tuberculosis. When a serous effusion develops, its tuberculous character can be determined by laboratory investigations. Pneumothorax, developing in a previously healthy individual is a rare but often serious clinical mode of onset.

(g) *Pneumonic*.—"Gallopings" consumption often begins with pneumonic manifestations, especially in the young.

(h) *Traumatic*.—Pulmonary tuberculosis may follow injury or "gassing," as described under ætiology.

(i) *Neurasthenic*.—Neurasthenia may occur as a complication of tuberculosis; but in some cases an initial neurasthenia dominates the picture, and the pulmonary lesion is only detected on careful examination.

(j) *Malarial*.—Regular attacks of sweating and fever may occur, especially in those who are or who have been residing in malarial climates, suggesting malaria, but in reality due to tuberculosis.

(k) *Associated with other diseases*.—Tuberculosis may follow immediately on an attack of measles, influenza or whooping-cough, especially if complicated by broncho-pneumonia. In some cases it develops at a later period after the acute disease.

(l) *Senile*.—In old people an insidious onset is common. The disease may be of bronchitic type, and the signs are often masked by emphysema. There may be little or no rise of temperature.

THE CHIEF SYMPTOMS of pulmonary tuberculosis are—

Cough.—This varies considerably in different types of disease. It may be very slight or absent in generalised miliary tuberculosis, or in any form in the insane. It is sometimes dry, persistent and ineffective, especially in miliary extension in the lungs from an old focus of disease, in bronchial gland tuberculosis, or in pleurisy. When there is associated bronchitis or caseation, the cough is usually accompanied by expectoration, which, if very tenacious, may lead to retching or even to vomiting, particularly in the morning. In laryngeal tuberculosis the cough is husky and frequently painful.

Expectoration.—In early disease there is usually no sputum, and in some cases, more especially in the fibroid type, widespread lesions may be present with practically no expectoration. When caseation is in progress, or when

there is secondary infection with bronchitis, the sputum may be abundant and amount to as much as 20 or more ounces in the 24 hours. It may be clear or mucoid, or thick tenacious muco-pus. If mucoid, it often contains small particles, the size of a pin's head or larger, of yellow caseous material. Nummul sputum may be met with in active caseous disease, especially with excavation. This consists of flat rounded masses of muco-pus, with a somewhat distant resemblance to coins. In tuberculosis the sputum is usually inoffensive, but may have the characteristic sickly odour which is also noticed to emanate from the patient himself (odor phthisicus). If bronchiectasis or gangrene occurs as a complication, the expectoration becomes typically malodorous. Pulmonary calculi or pneumoliths, composed chiefly of calcium carbonate or phosphate, are sometimes expectorated. They vary in size from a pin's head to a pea, are irregular in outline and sometimes branched, being derived generally from the walls of a cavity. Although the occurrence of these does not necessarily indicate fresh activity in the lungs, yet such a possibility should always be suspected, and a careful watch maintained on the temperature during the next few days. In some cases larger pneumoliths, as big as a cherry, may be coughed up, and those are frequently derived from calcified tracheo-bronchial glands. They may give rise to alarming symptoms at once, and be the forerunner of fresh activity in the lungs.

Microscopical examination.—The presence of tubercle bacilli in the sputum is the most decisive test of the existence of this disease. The small yellowish caseous particles should be selected from the sputum, and appropriately stained. If no tubercle bacilli are found, samples from the whole sputum of the 24 hours, concentrated by the antiformin method, can be examined. Droplets collected on a laryngeal mirror by cough induced by it may be examined for the presence of tubercle bacilli, especially in children or in patients who habitually swallow sputum.

Sputum culture by the Loewenstein method may be of value when tubercle bacilli are not found in smears. The cells present are usually of the mononuclear type, either mononuclear leucocytes or altered alveolar epithelial cells. The presence of elastic tissue indicates that destructive pulmonary lesions are in progress. Secondary infecting organisms may be demonstrated by cultural methods.

Dyspnœa.—Slight dyspnœa occurring early in the disease may be due to diminished movement of the diaphragm on the affected side. In more advanced cases the degree of dyspnœa is proportional to the amount of lung tissue involved. In addition, cough and pyrexia play a part in its production. Complications such as pleurisy, pleural effusion, pneumothorax and cardiac failure increase the shortness of breath. It is rare to find orthopnœa even in acute and rapidly spreading disease. In arrested cases the dyspnœa is proportional to the extent of fibrosis.

Cyanosis.—This is not an early symptom of tuberculosis. It is dependent upon the amount of lung tissue involved, but is increased by the coexistence of emphysema or cardiac failure. The typical "hectic flush" of tuberculosis is a vasomotor effect caused by toxæmia.

Pain.—Not every sufferer from tuberculosis experiences pain, even in the acute stages of the disease. The commonest cause of pain is dry pleurisy. When the diaphragmatic layer of the pleura is affected, pain may be referred to the epigastrium or to the corresponding shoulder. In chronic fibroid

phthisis there is frequently a dull, aching pain in the chest, which is more noticeable in damp weather. This is caused by the contraction of the condensing fibrous tissue. Cutaneous tenderness of the chest-wall is met with in some cases of advanced disease, and is probably due to a cachectic neuritis. A "cold abscess" forming along one of the ribs or costal cartilages is a rare cause of localised pain in the chest-wall. Cough may be painful, especially when paroxysmal or frequent, the pain being referred to the costal attachments of the diaphragm and upper abdominal muscles. The sudden occurrence of pneumothorax may cause such severe pain as to induce collapse; but when of more gradual onset no severe discomfort may be experienced. Tuberculous laryngitis may be the cause of very great suffering.

Night sweats.—Although not pathognomonic, night sweats occur more frequently in tuberculosis than in other diseases. They are met with in all stages of active lesions, and may be of great severity.

Loss of weight.—This is often an early symptom. It is most marked in acute disease and in the late stages of chronic fibro-caseous tuberculosis.

Fever.—Pyrexia is one of the most important indications of activity at any stage of pulmonary tuberculosis, although it does not follow that the disease is arrested when there is no fever. During treatment the temperature should be recorded at certain definite hours in the day. (a) On waking. The normal mouth temperature at 7 or 8 a.m. is 97° or 98° F. in the mouth, and 97°·2 to 99° F. in the rectum. This temperature should be taken in bed, before eating or drinking. (b) At 1 p.m., after the hour's recumbent rest. (c) At 6 p.m. (d) At 9 p.m., after retiring to bed. The maximum temperature is usually reached between 4 and 6 p.m., but may be delayed to 8 or 9 p.m. In most Continental sanatoria the rectal temperature is taken, and a centigrade thermometer employed. The temperature is dependent upon the extent and the activity of the disease, and upon the amount of exercise taken.

(a) In acute miliary tuberculosis it may be continuous or remittent, and the "typus inversus" is not uncommon, the morning temperature being higher than the evening. This is generally regarded as a sign of grave prognosis.

(b) In acute caseous tuberculosis the high temperature at the onset is continuous, and the record resembles a pneumonic chart. When caseation occurs it becomes hectic or intermittent, with a daily swing of 4° or 5° F. This is probably due to the action of tubercle toxins, and not to the presence of a secondary infection.

(c) In chronic fibro-caseous tuberculosis there is no characteristic temperature record. There may be only a very slight rise occurring at intervals of a few days. On the other hand, the patient may be afebrile while resting, but febrile when ambulant (Stage 2. Inman's classification). Further an afebrile ambulant patient may over-exert himself, and by excessive auto-inoculation develop a sharp rise of temperature which subsides in a few days with rest. The temperature chart is thus a guide to prognosis and to treatment, and if acute miliary tuberculosis or caseation occurs, a typical temperature variation ensues.

(d) In fibroid tuberculosis the temperature is usually normal, unless excessive auto-inoculation results from exercise, or the disease advances. The occurrence of hæmoptysis may have a very definite effect upon the temperature. In some cases it is not followed by pyrexia, but if the inhaled blood leads

to a hæmoptoic bronchitis, there may be a slight degree of fever lasting for a few days. When a definite and persistent pyrexia follows, it usually indicates activity around an old focus of disease, or fresh spread by inhalation of blood containing tubercle bacilli to distant parts of the lung.

A premenstrual rise of temperature may occur; but as it is also met with in healthy women it is not pathognomonic.

Hæmoptysis.—Hæmoptysis occurs at some stage of pulmonary tuberculosis in about 50 per cent. of all cases. With early lesions the sputum is only streaked. This may result from the congestion of tuberculous bronchitis, or from a small area of collapse or broncho-pneumonia. In the pneumonic or broncho-pneumonic forms rusty sputum may be seen. Profuse hæmoptysis generally occurs in chronic disease; but it is occasionally met with in acute caseous forms. Recovery may take place after coughing up 2 or 3 pints, or death may ensue rapidly from suffocation before any considerable quantity of blood has been expectorated. After the cessation of bleeding the sputum may be blood-stained for several days, the colour becoming darker. The source of profuse hæmoptysis is generally an aneurysm of a branch of the pulmonary artery lying in a cavity or in a fibroid lung, although occasionally ulceration without previous aneurysm formation may occur. In the majority of cases hæmoptysis begins while the patient is lying down or resting, so that exercise or work are not frequent exciting causes.

The patient notices a salt taste, feels a warm gush in the mouth, and then expectorates the blood. He is usually greatly alarmed, flushed and sweating, with rapidly beating heart. The blood at first is as a rule bright and frothy but some clots may be present; later it is mixed with muco-purulent expectoration, in the form of clots or streaks.

Circulatory system.—The heart may be small, but the right side often hypertrophies in chronic fibroid cases. Tachycardia may be due to nervousness, but when constant it generally indicates active disease or over-exertion on the part of the patient. The blood pressure is usually low in the stages of activity, and a steady rise during treatment is a favourable sign. If tuberculosis is coexistent with other diseases, such as atheroma, which raise the blood pressure, higher readings are naturally obtained.

The blood.—The red cells are usually normal in number, but there may be a slight anæmia. On the other hand, when there is much cyanosis, or after sanatorium treatment, the red cells may be increased. The colour index is usually low. In the early stages the leucocytes may be slightly increased. A polymorphonuclear leucocytosis occurs in caseation and in early cavity formation, and at times with secondary infection of the lungs. A special differential count of the polymorphonuclear leucocytes themselves may be made by subdividing them into groups, according to the number of their nuclear lobes, as suggested by Arneth. An increase in number of immature cells with only one or two nuclear lobes constitutes a deviation to the left from the normal, and raises the Arneth index. This lævo-deviation is said to indicate toxæmia, and if it is not present, the disease will probably be chronic. The von Bonsdorff modification in which the number of lobes of the nuclei in one hundred polymorphonuclear leucocytes is counted, is sometimes used. The normal figure is 274, a figure less than this suggests activity. The lymphocyte monocyte ratio is said to be of prognostic value, an absolute and relative increase in the monocytes being unfavourable.

Alimentary system.—The tongue is usually clean and the appetite good even in cases with marked fever. When tuberculosis of the larynx is present, there is frequently severe dysphagia. Dyspepsia may be complained of, usually of a nervous type. Anorexia, flatulence, distension with nausea are the commonest symptoms, pain being rarely noticed. There may be marked intolerance of fat in the diet. Atonic dilatation of the stomach may occur in some cases towards the end of the disease. Constipation is common; on the one hand, diarrhoea may occur apart from intestinal ulceration or lardaceous disease.

Nervous system.—The classical “*spes phthisica*” is distinctly rare, but when present is very striking from its contrast with the realities of the disease. It is most commonly seen in acute caseous tuberculosis. More often the patient becomes emotional and self-centred, depression is common and hard to combat, and melancholia with delusions occasionally develops. Neurasthenia is frequent and, as mentioned above, may lead to errors in early diagnosis. Insomnia may be due to cough, pyrexia, night sweats or pain, especially in laryngitis. With marked cachexia, a definite peripheral neuritis may occur.

Genito-urinary system.—In the early stages there is often an increased sexual desire, and this may recur when arrest is taking place. This is probably in part due to the therapeutic régime, the rest, abundant food and lack of interesting occupation reacting upon the nervous system of young adults. In advanced disease, all sexual desire is lost. Menstruation often ceases early, and the patient may seek advice for amenorrhoea. Women remain fertile even in advanced disease. The urine is normal in the early stages, later a febrile albuminuria may occur, or in advanced cases an amyloid nephrosis with generalised oedema may develop.

THE PHYSICAL SIGNS OF EARLY DISEASE.—The general appearance of the patient may be healthy, or may be that of malnutrition with the characteristics of the “*habitus phthisicus*,” the hair being lank and lustreless, the skin white, thin, dry and shiny, and the thorax of the alar or phthinoid type. Certain stigmata are described, which although useful, are not pathognomonic. The eyelashes may be long, dark and curling, the back hairy and the thoracic cutaneous venules dilated. When present around the upper thoracic vertebral spines, they are sometimes known as “the varicose zone of alarm.” Deficient movement may be observed below one clavicle, at the point of one shoulder, or at the lower costal margin. The corresponding shoulder may be slightly drooping, with flattening above or below the clavicle, and slight hollowing of the supra-spinous fossa with wasting of the trapezius muscle may be observed. Pottenger regarded these shoulder signs, when not due to scoliosis or kypho-scoliosis, as reflex, and comparable with the fixation of a tuberculous joint and wasting of its adjacent muscles. In women it may be noticed that the breast on the affected side is smaller and hangs at a lower level.

Palpation confirms the diminished expansion, and reveals a slight increase of vocal fremitus over the affected area of the lung, usually at one apex. The normal increase in fremitus of the right apex over the left must be borne in mind, in order to prevent mistakes.

With light percussion slight dullness and a small increase in the sense of resistance can be detected. This is usually most apparent in the

supra-spinous and upper interscapular regions. The extent of pulmonary resonance above the clavicle, known as "Krönig's isthmus," may be diminished by $\frac{1}{2}$ to 1 inch on the affected side.

Various types of breath-sounds may be heard over the affected portion of lung. They are—(a) weak inspiration, with expiration vesicular or inaudible; (b) cog-wheel inspiration, with expiration vesicular, prolonged or rarely jerky; (c) the "granular" breathing of Grancher, the breath-sounds being somewhat coarse and irregular, suggesting fine or distant râles, although none can be definitely detected; (d) harsh inspiration, with expiration vesicular or prolonged; (e) broncho-vesicular breathing; (f) definite bronchial breathing when early consolidation is in progress.

Often there are no adventitious sounds. Occasionally a few small rhonchi or fine crackling or bubbling râles may be heard with the first few deep breaths, or only with the inspiration immediately following coughing. If râles are constantly heard, it indicates that the lesion is already of some extent. Care must be taken to differentiate them from atelectatic râles, emphysematous râles audible along the sternal margin, and pleural friction or fascial creaks. There is usually a slight increase in the conduction of both the spoken and whispered voice, and the more definitely this extends away from the trachea in front, and from the vertebral spines behind, the more reliable is the sign as an indication of disease.

Mensuration is seldom practised in routine examination, but graphic records of the chest contour, which are of interest in following the progress of a case can be obtained by cyrtometry.

PHYSICAL SIGNS OF ACUTE MILIARY TUBERCULOSIS.—If the condition develops acutely from breaking down of an infected bronchial gland or small lung focus, the physical signs are generally those of an acute generalised broncho-pneumonia, unless there is meningeal involvement as well, in which case the pulmonary symptoms and signs are masked or obscured by those of the cerebral involvement. When miliary tuberculosis occurs as a terminal event in a chronic case, marked dyspnoea, cyanosis and tachycardia are early symptoms. There may be crepitations or fine crepitant râles widely distributed over both lungs, and sometimes areas of tubular breath-sounds especially in the lower lobes. The original signs are often masked or less apparent. This is especially the case if meningeal involvement occurs also.

THE PHYSICAL SIGNS OF CONSOLIDATION.—Limitation of movement and flattening over the affected part of the lung, usually the apex, is now more noticeable.

The diminution of movement is confirmed on palpation, and vocal fremitus is found to be definitely increased.

The pulmonary resonance is diminished to definite dullness and the sense of resistance is correspondingly increased.

The breath-sounds are bronchial, or in acute caseous disease may even approximate to tubular.

Adventitious sounds may be absent, but usually fine or medium crackling râles are heard with inspiration, especially after coughing. When active softening is in progress the râles frequently become coarse and sticky. The voice conduction is much increased, bronchophony and whispering pectoriloquy being audible.

Mensuration may confirm the presence of flattening.

THE PHYSICAL SIGNS OF EXCAVATION.—Flattening of the chest-wall and diminished movement over the cavity are now more marked; if the cavity is apical there is in addition notable dropping of the shoulder, and wasting of the shoulder-girdle muscles.

The diminution of movement is confirmed by palpation. Vocal fremitus is generally increased owing to the surrounding consolidation, but if the cavity is full or there is much pleural thickening, it is diminished.

The percussion note is dull when the cavity is small or filled with secretion. A peculiar boxy or "cracked-pot" note, the "*bruit de pot fêlé*," is obtained over large superficial cavities, especially when communicating with an open bronchus. This is best heard on percussing with the mouth open, and Wintrich showed that the note may be altered in pitch over such cavities when percussing with the mouth open or closed, apart from the actual presence of the cracked-pot sound. Gerhardt's sign (alteration of note with the position of the body) is supposed to indicate a cavity of oval shape. It is rare, and of little value.

The breath-sounds are bronchial, broncho-cavernous, cavernous or amphoric, according to the size of the cavity, and to the amount of its contents. When it is full the breath-sounds may be distant, weak or even absent, and this is especially noticeable in basal bronchiectasis.

With a dry cavity there may be no adventitious sounds. Usually râles are audible; they may be medium or large, and bubbling or crackling in character. Over a large cavity a metallic tinkle and amphoric echo may be heard. With a very large cavity, extending through the whole of one lung, a typical *bruit d'airain* is at times obtainable. Voice conduction is increased, bronchophony and whispering pectoriloquy are present, and in some instances post-tussive suction is heard. Some cavities are only revealed by X-ray examination or by tomography.

Mensuration affords a graphic representation of the flattening of the chest-wall.

THE PHYSICAL SIGNS OF FIBROSIS.—The chest is asymmetrical, the affected side being flattened and moving little, while compensatory scoliosis or kypho-scoliosis is often present. The cardiac impulse is seen to be displaced towards the affected lung and may be higher or lower than normal. It may be drawn over to the right axilla, or on the left side as far back as to the posterior axillary line, or even to the angle of the scapula. The intercostal spaces may be retracted, and dilated venules are sometimes seen over the front of the chest as the result of obstruction, caused by displacement of the mediastinum and traction on the deeper veins.

Diminution of movement is confirmed by palpation, and the cardiac impulse can be more accurately localised. Vocal fremitus may be increased or diminished; the former occurs when the lung is consolidated and the large bronchi patent, the latter when there is much pleural thickening.

The percussion note over fibroid lung is dull and the sense of resistance increased, unless cavities are present. The opposite lung may be hyper-resonant, and its resonance extend across the mid-sternal line. The cardiac dullness is often continuous with that of the fibroid lung, and its area can only be determined by the cardiac pulsation.

The breath-sounds are weak and distant, unless modified by the presence of a cavity.

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Often there are no adventitious sounds, although fine or medium râles of a sticky or metallic nature may be heard. The voice conduction is usually diminished, and there is no pectoriloquy unless excavation has occurred, when bronchophony and pectoriloquy are audible.

It must be borne in mind that in actual disease the lesions are not so clear-cut and well-defined. In a case of some duration different stages of disease can be found in the same individual, thus infiltration, consolidation with softening, excavation and fibrosis may be present in different lobes of the lungs, and thus it may be possible to determine the site of origin and path of spread of the disease.

Certain other signs are occasionally seen in pulmonary tuberculosis.

Myoidema is an undue irritability of the muscles to direct mechanical stimulation, revealing itself by a flickering fibrillary contraction on tapping with the finger, and may occur in tuberculosis at all stages. It is best seen over the pectoralis major on the affected side. It may be present quite early, but is not pathognomonic, as it may occur in any cachectic state.

Clubbing of the fingers is commonly seen in chronic cases, the nails are curved and present a parrot-beak appearance, the thumb, index and middle fingers being most affected. Drum-stick clubbing is only seen in fibroid lesions with bronchiectasis.

CHRONIC MILIARY TUBERCULOSIS.—*Clinical features.*—Chronic miliary tuberculosis affects males and females equally. The age incidence is between 6 and 40 years, but usually the patient is between 11 and 30. The early symptoms include cough, shortness of breath, pain in the chest, expectoration and at times hæmoptysis. Constitutional disturbance is usually slight with low-grade fever. In some cases the spleen is palpable and in others tuberculous lesions are present in glands, bones or joints. Tubercle bacilli may be found in the sputum, gastric contents or pleural fluid. X-ray examination reveals small symmetrical, rather ill-defined opacities throughout the lung-fields. In some cases they may be limited to one portion of the lung. The course of the disease is more prolonged than that of miliary tuberculosis, being never less than three months and usually considerably longer. Death may occur in about six months, or eventually the disease may be arrested.

The differentiation of chronic miliary tuberculosis from the acute variety depends rather upon the clinical than the radiological findings. The fact that the pulmonary lesions are tuberculous is established in many cases by the finding of tubercle bacilli in the sputum or stomach contents. When tubercle bacilli cannot be detected, other conditions giving rise to scattered opacities throughout the lung-fields must be considered. Thus in secondary carcinomatosis, sarcomatosis or chorio-epitheliomatosis, the opacities are usually larger. Multiple opacities are also found in pneumokoniosis, congestive heart failure, sarcoidosis and more rarely with periarteritis nodosa, xanthomatosis, bilharziasis, leukæmia and disseminated broncho-pneumonia.

HILUM TUBERCULOSIS.—The existence of a special type of pulmonary tuberculosis commencing at the root of the lungs and extending thence in a fan-shaped manner along the bronchi has been postulated by some authorities, chiefly on X-ray evidence. While lesions in the middle zone of the lung are sometimes revealed by X-ray examination, it is more than doubtful whether this condition merits recognition as a separate variety of the disease.

EPITUBERCULOSIS.—This term was applied by Eliasberg and Neuland in 1920 to a condition of consolidation in tuberculous infants, often affecting a whole lobe. In spite of definite physical signs and characteristic radiological appearances, there are few symptoms, and recovery is the rule, with fairly rapid clearing of the X-ray shadows, from the periphery inwards. It is probably due to atelectasis of a lobe in whole or part, due to bronchial obstruction from enlarged hilar glands.

HILAR FLARE is the name given by Burton Wood to a similar condition affecting the right middle lobe, or the upper part of either lower lobe in childhood. The X-ray shadow is generally triangular in shape, the base being at the hilum or mediastinum. This, like epituberculosis, is probably due in many cases to localised bronchial obstruction, but in both conditions it is possible that there may be an allergic factor.

It is important to note that in both epituberculosis and hilar flare, the chief evidence is radiological, and that the prognosis is good.

PULMONARY OSTEO-ARTHROPATHY.—In cases with bronchiectasis the joints may be affected, swelling occurring especially in the wrist, ankles and knees, and rarely in the hips and shoulders. A serous effusion into the joints may be present. Pain is usually slight, but there is much deformity and functional impairment. X-ray examination reveals productive periosteal changes, which may also affect the long bones and the spine (see p. 1376).

RADIOGRAPHY OF THE CHEST.—If possible, the chest should be examined in every case with the fluorescent screen, and a photograph taken on a film. Certain important points can only be determined by a screen examination. The chief of these are the respiratory movements of the diaphragm, lighting up of the apex of the lung with inspiration, and the cardiac pulsation. Unilateral restriction of diaphragmatic movement not infrequently occurs in early apical tuberculosis, but as it may be observed under other conditions, notably with pleural adhesions, it is not diagnostic. The film will show the extent of the disease and in some cases it may suggest the existence of activity; thus areas of consolidation, caseation or excavation can be demonstrated, and thickening of the pleura, pleural effusion and pneumothorax give their characteristic appearances. In films taken in the erect position a fluid level is often apparent in cavities. A curious rounded shadow may be apparent in early disease called Assmann's focus, in which rapid changes, such as softening, may occur. The tomograph sometimes shows unsuspected cavities.

The significance of "root shadows" is still debatable. Although the presence of calcareous deposit in the glands at the roots of the lungs is usually obvious, the interpretation of the radiating shadows is a matter still under discussion. They may be due to peribronchial thickening, caused by the formation of fibrous tissue, or may merely represent the shadows cast by the branches of the pulmonary artery.

A film is often of crucial value in determining the presence or absence of early disease, but slight diminution of translucency of one apex may be due to an old arrested lesion, or, on the other hand, there may be definite physical signs of active disease, without abnormalities being found on X-ray examination. The heart shadow is often narrow and vertical in tuberculosis. Displacement of the heart due to pulmonary fibrosis or to affections of the pleura is clearly indicated. A good film may also give valuable information as to the

extent of lung involved and as to the presence of complications, such as effusion, pneumothorax or bronchiectasis. As the X-rays only cast shadows lacking in all pictorial details, tuberculous shadows cannot always be distinguished from those due to other pulmonary lesions. It is thus clear that the X-ray findings should always be interpreted in connection with the history, symptoms and physical signs of the case. The X-rays, although often of great help, do not afford a simple road to diagnosis or supply infallible evidence in determining the nature of an obscure case. On the other hand, they are absolutely essential in the determination of the suitability of a case for artificial pneumothorax or other collapse treatment and in controlling its application.

Complications and Sequelæ.—Compensatory emphysema is common in chronic fibroid disease, but bronchiectasis occurs less frequently. Gangrene of the lung is not often observed. Colds and catarrhal affections of the respiratory passages are frequent in sufferers from tuberculosis, and lobar pneumonia may develop as a complication. Bronchitis often occurs, due either to spread of the tuberculous process or to a secondary infection. In some instances asthma appears for the first time after tuberculosis has become manifest. A tuberculous abscess occasionally forms about a rib or costal cartilage.

Small areas of dry pleurisy are present at some stage in nearly every case; a serous pleural effusion is common, and an empyema may develop as the result of a mixed infection, or from the tubercle bacillus alone. Pneumothorax may occur as an early complication, or late in the disease, generally from rupture of a caseous focus just under the pleura; this frequently progresses to the formation of a pyo-pneumothorax. The implantation of tubercle bacilli from the expired air or sputum may lead to secondary foci in the larynx, trachea and epiglottis, or more rarely in the pharynx, tonsils, base of the tongue or nose. Swallowing of sputum containing tubercle bacilli gives rise to gastro-intestinal complications in many cases. The most common site of tuberculous ulcers, is the terminal portion of the small intestine, but the appendix may be affected, and the connective tissue around the cæcum is sometimes matted and thickened to form a palpable mass (hypertrophic tuberculoma). Tuberculous peritonitis is not common in adults and is usually secondary to intestinal lesions. The stomach is very rarely ulcerated, but an atrophic gastritis may occur in advanced cases. Fistula-in-ano and ischio-rectal abscess are comparatively common complications and tubercle bacilli may be found in the discharges.

Small vegetations may be found post mortem in the heart on the aortic and mitral valves, but these are usually due to some terminal infection. Fatty degeneration of the myocardium occurs as a result of toxæmia, and infection by direct spread along the lymphatics may lead to pericarditis. The peripheral circulation is not infrequently poor, chilblains are common, and cachectic purpura may be seen. Lardaceous degeneration as a consequence of chronic tuberculosis is not so common nowadays as formerly, but when present may affect the liver, spleen, intestines, lymph glands and kidneys.

The genito-urinary complications include lesions in the kidneys, bladder, epididymis and prostate. If the suprarenal body is affected Addison's disease will usually develop. Spinal caries is occasionally observed. A peripheral neuritis may form part of the lesions occurring with marked

cachexia. Generalised dissemination of the tubercle bacilli by the blood stream is followed by tuberculous meningitis.

Course.—The course pursued by pulmonary tuberculosis is variable, depending upon the clinical type of the disease. In acute miliary tuberculosis, death may occur in from 1 to 3 weeks from toxæmia or generalisation of the lesions. In acute caseous tuberculosis, death usually results in from 1 to 6 months. In chronic fibro-caseous tuberculosis, the disease may be completely arrested, or after a temporary arrest may become active at intervals and again become arrested under suitable treatment; in other instances it progresses steadily to a fatal termination. In fibroid tuberculosis the disease may become completely arrested or smoulder quietly for many years.

Apart from the question of the expectation of life, various stages of tuberculosis are described based upon anatomical lesions, toxæmia and functional disablement. The most important of these are as follows:

1. **THE TURBAN-GERHARDT CLASSIFICATION.**—An anatomical classification based upon the extent of lung tissue involved. Three stages are described. *Stage 1.* Early cases in which physical signs, if unilateral, only extend from the apex to the second rib, and, if bilateral, are limited to the supra-clavicular and supra-spinous regions. *Stage 2.* The signs, if unilateral, do not reach lower than the fourth rib, and, if bilateral, are situated above the second ribs. Excavation is not present in this stage. *Stage 3.* This includes more extensive lesions or localised ones in which excavation is present.

2. **SIR ROBERT PHILIP'S CLASSIFICATION.**—Both the extent of lung tissue involved and the degree of toxæmia present are taken into consideration. Twelve stages are described, which are indicated by the following signs:

L₁, L₁S, L₁S, and L₁S; L₂, L₂S, L₂S, and L₂S; L₃, L₃S, L₃S, and L₃S. L₁, L₂, and L₃ represent lung involvement to the extent of stages 1, 2 and 3 respectively, according to the Turban-Gerhardt scale. s applied to these letters indicates that there is only slight systemic disturbance, whereas S signifies marked systemic disturbance; and the signs L₁S, L₂S and L₃S show that the systemic disturbance is excessive in relation to the lung involvement.

3. **INMAN'S CLASSIFICATION.**—This is based solely on the temperature in relation to exertion.

Stage 1. The patient is febrile when resting. *Stage 2.* The patient is resting afebrile, but ambulant febrile. *Stage 3.* The patient is ambulant afebrile. *Stage 4.* The patient is working afebrile.

The course taken by tuberculosis of the lung may lead to several terminations. These are: (1) permanent arrest, either by fibrosis prior to caseation, or if the latter has occurred, by calcification and fibrosis; (2) incomplete arrest, as shown by the persistence of tubercle bacilli in the sputum, or by slight degrees of pyrexia on over-exertion; (3) rapid extension, here the disease spreads, and the toxæmia is out of all proportion to the extent of the lesions; (4) death, this may result from the pulmonary lesion or from complications. The former may prove fatal as the result of progressive asthenia or cardiac failure, from asphyxia due to acute miliary tuberculosis or hæmoptysis, or in a small proportion of cases directly from loss of blood in repeated hæmoptysis. The complications that most often prove fatal are meningitis, enteritis, laryngitis leading to dysphagia and starvation, or

pneumothorax. Intercurrent diseases, such as pneumonia, influenza or diabetes, are occasionally the cause of death.

Diagnosis.—This is easy when definite signs are present in the lungs, and when tubercle bacilli are found in the sputum. On the other hand, the diagnosis of early cases may present one of the most difficult problems in clinical medicine. Tuberculosis may be suspected on account of symptoms, although the physical signs are indefinite. The conditions which most frequently lead to doubt are dyspepsia, neurasthenia, debility, visceroptosis and intestinal stasis, oral sepsis, tachycardia associated with early Graves's disease or heart disorders, affections of the nose and throat, and in children enlargement of the bronchial glands or acidosis. The history and symptoms are of great importance in these cases, and a careful examination should be made of each system. A test meal, opaque meal, or blood examination may be required before the correct diagnosis is established.

On the other hand, there may be definite signs of disease in the lungs which have to be differentiated from those produced by other conditions simulating tuberculosis. The cases included in this group embrace the majority of pulmonary lesions, especially chronic bronchitis, fibrosis, bronchiectasis, asthma, emphysema, apical catarrhs and collapse, pleurisy, new-growths and cysts. Diagnosis depends upon the history and course of the disease, together with a careful record of the physical signs in the chest, investigation of the sputum for infecting organisms, X-ray examination and in some cases the determination of the Wassermann reaction.

A condition of special difficulty is that of the variety of sarcoidosis known as Boeck's sarcoid. The lesion is a benign lymphogranuloma or reticulosis. It affects the lymph glands, lungs, bones especially those of the fingers, and the skin (see pp. 1478-1480). The parotid and lacrimal glands are sometimes involved and irido-cyclitis has been recorded in 10 per cent. of the cases. The intestines, spleen and liver may be affected. The chief diagnostic points are the character of the skin lesions and the chronicity and tendency to spontaneous arrest. Tubercle bacilli are not found and the Mantoux reaction is often negative. The radiological appearances in the lungs are those of a diffuse mottling—coarser than that of miliary tuberculosis. The hilar glands are often markedly enlarged.

When the diagnosis still remains doubtful the patient should be placed under observation, and a series of examinations carried out, the object of which is to determine whether or not active tuberculosis is present. The temperature should be observed with the patient in bed, a daily rise to 99° F. or a swing of 1°·5 to 2° below normal being suspicious. The sputum should be examined repeatedly for tubercle bacilli by the ordinary method, and if not found, the antiformin process should be carried out.

Before applying any tuberculin tests the blood may be examined serologically. The use of the Arneft or von Bonsdorff blood count in diagnosis has been referred to on page 1196. The complement-fixation test has proved disappointing, and in the present form does not afford reliable criteria of activity or quiescence.

The sedimentation test of the blood (stability reaction), i.e. the rate of settling of the erythrocytes in blood diluted with sodium citrate solution, is affected in this disease. In active cases the sedimentation rate is increased, but this reaction is not specific. It is also increased in other conditions such

as pregnancy, carcinoma, syphilis, rheumatism and acute infections. The test is therefore of little or no value in diagnosis, but it affords valuable indications of the degree of activity, and may assist in determining the form of treatment.

It has also been used as a guide to prognosis, since it is affirmed that arrest should not be considered as firmly established until the sedimentation rate has returned to normal. This may not occur until some time after the usually accepted clinical symptoms and signs of activity have disappeared.

THE TUBERCULIN TESTS.—1. *Cutaneous (the Pirquet reaction).*—Scarifications are made on the skin of the forearm through a drop of Koch's old tuberculin, human and bovine, and through a drop of saline as a control. A positive reaction is shown by the formation of a slightly raised, reddened papule at the site of the scarification through one or other varieties of tuberculin, whereas the control is not affected. Unfortunately, except in the first two years of life, this affords no indication of active disease, but only reveals the presence of previous infection with resulting tuberculin sensitiveness. A positive reaction is therefore given by the majority of adults.

2. *Mantoux's intradermal test.*—An injection of 0.1 c.c. of a 1 in 10,000 dilution of old tuberculin (0.01 mgrm.) is injected intradermally. If no reaction occurs, the injection is repeated in a week, with 0.1 c.c. of 1 in 1000 dilution (0.1 mgrm.). A positive reaction is shown by a red areola, with some cedema and occasional vesiculation.

A modification of this test consists in the use of Purified Protein Derivative (P.P.D.). This is supplied in tablets of two strengths, which must be dissolved in a buffered solution immediately before use. The advantage of this preparation is its constant potency.

3. *Vollmer's patch test.*—A strip of adhesive plaster is applied over the sternum, previously cleaned with ether. To this strip are attached three small squares of filter paper, the central one is a control of untreated paper, the other two have been saturated with undiluted old tuberculin and allowed to dry. The plaster is removed in 48 hours and 12 or 24 hours later a positive reaction is shown by redness, infiltration and sometimes by papules or vesicles.

4. *The subcutaneous test.*—The patient must be apyrexial, and must be kept in bed— $\frac{1}{10}$ mgrm. of old tuberculin is injected subcutaneously, and its effect determined. The reactions that may develop are—(a) local, an inflammatory swelling at the side of the injection; (b) focal, an increase of the signs observed at the seat of disease in the lungs, such as the temporary appearance of a few râles at one apex. This is the most important; (c) general, as judged by a rise of temperature and sense of malaise. The temperature should be charted 4-hourly after the injection, and a rise to over 99° F. indicates a positive reaction. If no reaction follows this initial dose, larger injections are given at intervals of 2 or 3 days, in this sequence: $\frac{1}{2}$, $\frac{1}{3}$, 1, 5 and even 10 mgrms.

This test has the drawback that it does not indicate activity of disease, and it has the additional disadvantage that it may cause a quiescent pulmonary focus to light up and spread, and so cause irreparable damage.

Finally, the X-rays afford most valuable assistance in the diagnosis of early cases with doubtful signs, and also help in the differential diagnosis of tuberculosis from other lung diseases with well-marked signs.

Prognosis.—A number of factors must be critically considered in the determination of the prognosis in pulmonary tuberculosis.

A marked family incidence generally suggests an unfavourable course, though this rule is not invariable.

Personal history.—Chronic alcoholism is serious, chiefly because the régime of treatment is then peculiarly irksome, while the digestion and powers of resistance are often impaired in alcoholics. Tuberculosis in syphilitics frequently assumes a fibrotic type, and its course may be beneficially influenced by anti-syphilitic treatment. The outlook is grave when tuberculosis is conjoined with diabetes though less so since the use of insulin. Congenital heart disease and pulmonary stenosis are unfavourable factors; but hypertrophy of the heart and mitral stenosis are said to be beneficial.

The prognosis is very grave in infants and young children; but slightly less serious up to the age of 20. Between 20 and 50 age has little influence; but in later years the outlook becomes progressively less favourable.

Apart from the effects of pregnancy and exposure, sex plays no important part.

Freedom from financial embarrassment improves the prognosis, inasmuch as advice can be sought early, and treatment carried through thoroughly.

Marriage often leads to a breakdown in arrested cases especially in women, and induces more rapid spread of active lesions.

Persistence in an unfavourable occupation, or return to it after completion of institutional treatment, affects the prognosis adversely.

Poor chest development and the "habitus phthisicus" are usually bad prognostic signs, although tuberculosis may run a rapid course even in patients with good physique.

Patients with resolute and persistent personality are more likely to persevere with treatment and to recover, than those of weaker moral fibre.

The prognosis is greatly affected by the type of the disease—acute miliary tuberculosis is usually rapidly fatal, whereas in acute caseous tuberculosis, although the prognosis is very grave, recovery may occur. In fibro-caseous tuberculosis the prognosis is most uncertain and difficult to forecast. Every factor must be carefully considered, and the response to treatment noted. The best outlook is in fibroid disease, which often undergoes complete and permanent arrest.

SYMPTOMS IN THEIR RELATION TO PROGNOSIS.—Persistent cough, by exhausting the patient and disturbing sleep, is often unfavourable.

The amount of sputum is usually dependent upon the type of disease and upon the presence of secondary infection, and may therefore be of value in prognosis.

The significance of tubercle bacilli in the sputum.—The figures obtained at the Midhurst Sanatorium, over a period of 8 years, in which the after-history of the patients was traced for the ensuing 6 years, show that the prognosis is best in "closed" cases; but that it is nearly as good in those cases in which the tubercle bacilli disappear from the sputum during the sanatorium treatment. Persistence of bacilli in the sputum is an unfavourable sign. The actual number of bacilli in the sputum and the presence of "beading" have no definite prognostic significance.

Cases commencing with hæmoptysis progress more satisfactorily than

those with other modes of onset, chiefly because they are diagnosed earlier. *Hæmoptysis* occurring later may exert an unfavourable influence, either indirectly by spreading the disease into previously healthy portions of the lungs, or actually by the loss of blood.

If *dyspnoea* is not due to attacks of bronchial spasm, it has usually an unfavourable significance.

The temperature affords a clue to the type and activity of the disease, and is thus a valuable aid to prognosis. Profuse and persistent night sweats, or marked *anorexia*, especially when occurring early in the disease, are grave signs. *Tachycardia* due to *toxæmia*, signs of cardiac failure, *cedema* and *albuminuria* are of bad omen. The blood pressure is thought by some to be a useful guide, systolic figures below 100 mm. Hg being unfavourable, whereas a rise of pressure may be associated with amelioration of the disease. In fibroid lesions the pressure may be raised throughout.

THE EXTENT OF PHYSICAL SIGNS.—The activity of the disease rather than its extent is the important factor in determining prognosis. The development of compensatory *emphysema* is of value only as an indication of fibrosis in the tuberculous portion of lung, and therefore of chronicity.

THE INFLUENCE OF COMPLICATIONS ON PROGNOSIS.—Generally speaking, the presence of complications increases the gravity of the disease. Involvement of the larynx is a serious complication, especially when accompanied by *dysphagia*; but complete recovery may take place if the pulmonary lesion is quiescent. In early cases spontaneous *pneumothorax* occasionally acts favourably; but when it develops in association with extensive tuberculosis, and especially if it progresses to *pyo-pneumothorax*, it is almost invariably fatal though, if the disease is unilateral, surgical measures may prove successful.

Pleural effusion often has a beneficial influence by diminishing the movements of a lung in which there is an early tuberculous focus.

Secondary catarrhal affections tend to increase the cough and expectoration, and may lead to further spread of the disease.

Meningitis is almost invariably fatal. *Tuberculous peritonitis* or *enteritis* is a very grave complication, but *fistula-in-ano* often occurs in chronic cases, and exerts no marked deleterious effect. Involvement of the genito-urinary system increases the severity of the disease, especially if the kidneys or bladder are affected. If the *epididymis* alone is involved the prognosis is not materially affected, as the lesion can be dealt with surgically, although the administration of a general *anæsthetic* may cause spread of the pulmonary disease. For this reason when operations are urgently needed on these patients, gas and oxygen, basal *anæsthetics*, local or spinal *anæsthesia* should be insisted on.

As shown by the figures obtained at the Midhurst Sanatorium, a fairly accurate guide to prognosis is afforded by observing the condition of the patient on admission to the sanatorium, and his response to treatment. Even in the most favourable cases, which are diagnosed in an early stage, and progress satisfactorily under treatment, the mortality rate is six times greater after discharge from the sanatorium than it is for the remainder of the population of England and Wales for the same age periods; whereas in the cases of advanced disease the mortality rate is thirty-eight times greater than for the average population. As the most critical time is during the two

or three years succeeding discharge from the sanatorium, the prognosis is largely affected by the conditions of life during this period.

The rate of sedimentation of the erythrocytes (see p. 1204) has proved to be a valuable aid to prognosis. A persistently rapid rate is unfavourable.

Treatment.—**PROPHYLACTIC.**—The prophylaxis of tuberculosis involves a consideration of public health questions dealing with the purity of the milk supply, the infection of meat, sanitation and housing, the early diagnosis of tuberculosis, the examination of contacts, and the segregation of "open" cases. Inoculation with the B.C.G. vaccine (attenuated living bovine bacilli) has not met with favour in this country. All these questions are considered in the general article on Tuberculosis.

CURATIVE.—This varies with the type and stage of the disease. In all acute or febrile cases treatment should be commenced at home or in a nursing home or hospital, where the patient can be under careful observation in bed. The various forms of treatment which may be considered are—(1) sanatorium treatment; (2) home or institutional treatment; (3) dietetic treatment and personal hygiene; (4) climatic treatment; (5) graduated rest, exercise and labour; (6) medicinal treatment; (7) specific measures; (8) operative treatment; (9) symptomatic treatment.

1. SANATORIUM TREATMENT.—This constitutes the best mode of treatment for early and for certain types of chronic disease; but is totally unsuited for acute febrile or very active cases. The advantages obtained are: (a) the patient learns the most suitable mode of life, and the methods employed to check the spread of the disease; (b) the housing is specially designed and the climatic conditions are good; (c) the dietary is abundant and adapted to the patient's needs; (d) there is constant skilled medical supervision, and the daily routine is adapted to the actual physical condition of the patient.

On arrival a newcomer is kept in bed for a few days in order that his resting temperature may be observed, and the necessary examinations carried out. If there is pyrexia, rest in bed must be enforced until the temperature falls to normal. If the temperature rises above 99° F. when the patient is up, return to bed is usually necessary. The routine of sanatorium treatment varies in different institutions, the most important divergence being whether or not a system of "graduated exercise" is employed. In nearly all an hour's recumbent rest is enforced before lunch and dinner.

After three months' stay it is usually possible to decide whether the patient is responding to treatment, and, if so, it should, if possible, be prolonged for at least another three months, or until the sputum is free from tubercle bacilli.

2. HOME AND INSTITUTIONAL TREATMENT.—Treatment at home, in nursing homes or in special hospitals, is essential in early cases with fever, and in cases in which it is necessary to establish the diagnosis. Home treatment is also usually necessary on return from sanatorium or climatic treatment, if arrest is incomplete. An endeavour should always be made to carry out the principles inculcated at the sanatorium, and the patient should be under regular medical supervision. Advanced cases are best looked after in special institutions.

3. DIETETIC TREATMENT AND PERSONAL HYGIENE.—It is desirable to graduate the diet in each case so that the patient is restored to the previous maximum weight; but, in order to accomplish this, the food should be slowly

increased and all ideas of enforced overfeeding discountenanced. A total calorie value of 3000 to 3500 is usually ample ; but, if the patient is performing heavy work, as much as 4000 may be necessary. Meat, fish, eggs and fats are usually well tolerated. It is not often necessary to give large quantities of milk when the patient is on a full dietary. If extra food is required, the protein may be increased by raw meat sandwiches. Additional carbohydrates with small daily doses of insulin if they are not well tolerated are often helpful.

In all cases in which there is expectoration the patient should be clean-shaven. Great care must be taken in the disposal of sputum to ensure that it does not become dry, and that flies do not have access to it. All patients who are up should carry special sputum flasks, while those who are in bed should have sputum cups suitably covered and containing disinfectant. The sputum should be burnt, or, if this is impossible, it should be emptied into the water-closet after disinfection with carbolic acid or other simple or cheap disinfectant.

Smoking is best avoided in cases of active disease or laryngeal tuberculosis, and in no instance should inhaling be allowed. Woollen under-garments should be worn ; but all excess of clothing is harmful. Sun-bathing and injudicious uncontrolled sun exposure are dangerous and often activate quiescent lesions. Patients should be strongly warned of this danger.

4. CLIMATIC TREATMENT.—This is undoubtedly of value in carefully selected cases. The climatic resorts fall into three groups—mountain, marine and inland.

The mountain resorts.—In Europe the most suitable places are found in Switzerland. Among these are St. Moritz (6090 feet), Arosa (6000 feet), Davos (5150 feet), Montana (5000 feet) and Leysin (4690 feet). In America the most celebrated resorts are in the Rocky Mountains at Colorado Springs (5000 feet) and Denver (5000 feet), or in the Andes or Adirondack Mountains. The advantages of high altitudes consist in the stillness, purity and rarefaction of the air, and the greater diathermancy of the atmosphere to the sun's rays. Metabolism and the general circulation are thereby increased.

High altitudes are suitable for early cases which are afebrile, or for quiescent cases of more advanced type. Contra-indications are recent hæmoptysis, active disease with fever, extensive fibrotic lesions and complications such as emphysema, asthma, cardio-vascular lesions or nephritis.

Marine and coast resorts.—Among the important coast resorts in the British Isles are Hastings, Bournemouth, the Isle of Wight, Torquay, Falmouth, Llandudno, Penmaenmawr, Scarborough, Mundesley and the various seaside towns in Thanet. Farther afield are the French and Italian Riviera, Madeira, the Canary Isles, Morocco, Algiers and Egypt. The climate tends to be warm, moist and equable, and the amount of ozone is probably increased. These resorts are especially suitable for cases of more advanced and active disease, and for those complicated by hæmoptysis, bronchitis, emphysema and laryngitis. Residence by the sea actually at sea-level is undesirable.

Inland resorts.—These are to be found on the English and Scottish moorlands. The climate of California, the South African veldt, and parts of Australia and New Zealand are admirably suited to this disease, especially for arrested or early uncomplicated cases ; but the laws against the admission of tuberculous patients are strictly enforced at all of them.

Sea voyages.—These are contra-indicated for all except completely arrested

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cases owing to the lack of fresh air in cabins, the possibility of sea-sickness, and the difficulty of obtaining suitable treatment if the disease advances.

5. **GRADUATED REST AND EXERCISE.**—Treatment in bed is necessary so long as there is fever, and if the raised temperature is persistent “absolute rest” should be enforced. This consists in keeping the patient recumbent in bed, sufficiently well covered to prevent any muscular contraction from chill, while feeding and washing are attended to by the nurse, and the use of the bed-pan and slipper for evacuations is insisted upon. When the temperature becomes normal the patient is allowed up for varying periods, commencing with 1 hour daily, and increasing slowly to 6 or 8. If still apyrexial, walking exercise of 1 or 2 miles or more daily can be allowed.

The system of “graduated exercise” which Paterson instituted at the Frinley Sanatorium has proved of great value. There are six grades, the first and lightest consisting of walking up a slope carrying a light weight such as a basket of earth, while the sixth and heaviest involves hard manual labour with a pickaxe or shovel for 6 hours daily. This system is based on the principle that muscular exercise leads to the discharge of tubercle toxins from the pulmonary focus, and by liberating these in gradually increasing doses, a condition of active immunity is induced. A careful watch must be kept during this controlled process of auto-inoculation to prevent excessive doses of toxin being discharged, which are early indicated by rise of temperature and of pulse-rate, headache, increased cough and expectoration, lassitude and malaise. If such occur, the patient should be put back to bed for a few days, and when the condition has subsided the graduated exercise may be resumed at the grade which induced the over-inoculation or that immediately below it.

6. **MEDICINAL TREATMENT.**—No specific drug has yet been discovered for the treatment of tuberculosis. Amongst the medicines in most general use are :

(a) *Cod-liver oil.*—This may be administered by the mouth in doses up to 2 ounces daily. The cod-liver oil may be of value either on account of its fat-soluble A and D vitamins content, or, as suggested by Rogers, it may assist by dissolving the capsules of the tubercle bacilli and so facilitating their disintegration. Halibut liver oil is now sometimes used instead.

(b) *Creosote.*—This may be given in doses of min. 2 to 3 three times a day after food, either in combination with cod-liver oil, or in capsules. It should be discontinued if gastric disturbance or hæmoptysis ensue.

(c) *Hypophosphites.*—These are not so generally used as formerly and, beyond their “tonic” effect upon the nervous system, have no demonstrable influence upon the pulmonary lesion.

(d) “*Nascent iodine*”—With the idea of liberating free iodine in the tissues, potassium iodide grs. 30 is administered after breakfast in half a pint of water, and throughout the day 3 to 5 ounces of chlorine water are consumed with lemonade. This treatment is of value in certain chronic fibroid cases, but it often produces no appreciable results, and may cause dyspepsia.

(e) *Arsenic.*—Liquor arsenicalis min. 2 to 3 by mouth, or sodium cacodylate gr. $\frac{1}{4}$ to $\frac{1}{2}$ subcutaneously, are of value in some cases associated with anæmia. Neoarsphenamine, administered intravenously, is useful in chronic cases complicated by syphilis.

(f) *Inhalations*.—Disinfectant drugs when inhaled often check cough lessen expectoration and improve the general condition of the patient. Lees's inhalation is of value. It consists of creosote, parts 2; acid. carbol., 2; liq. iodi mitis, 1; sp. ætheris, 1; and sp. chlorof., 2. About 6 drops an hour are placed upon a Burney-Yeo mask, which should be worn almost continuously throughout the day. A modification now more frequently employed is: menthol, 4; olei cinnamomi, 3; olei limonis, 4; creosote, 10; olei pini, 10; sp. chlorof., 10.

(g) Calcium is often given by the mouth in the form of colloidal calcium in doses of 60 minims three times a day; or intramuscularly, as calcium gluconate, 5 c.c. of a 10 per cent. solution once or twice a week. Parathyroid extract is sometimes administered at the same time.

(h) There has been a revival of interest in preparations of gold in the treatment of this disease, notably by Møllgaard, who uses sodium aurothio-sulphate, to which the name of sancocrysin has been applied. It is now administered in smaller doses than when it was first introduced. The initial dose is usually 0.01 g. dissolved in sterile saline solution and injected intravenously. The second dose is given 5 days later, and is as a rule 0.025 g. If no reaction occur, the dose is increased to 0.1 g., and usually later to 0.25 g., the intervals being extended to a week. The total amount given in a course is usually 3 g., or sometimes up to 5. The course may have to be interrupted owing to reactions, chiefly fever, albuminuria, stomatitis, diarrhoea, peripheral neuritis and skin manifestations.

7. SPECIFIC MEASURES.—(a) *Active immunisation*.—Tuberculin and tubercle-vaccines.

The tuberculin treatment has not fulfilled the high hopes held out on its introduction by Koch. There are now numerous forms of tuberculin available, indicated by certain letters, and falling into three groups. (1) Those containing the exo-toxins only. These include Koch's old tuberculin T., O.T., and T.O.A., Denys' bouillon filtré (B.F.) and albumose-free tuberculin, T.A.F. (2) Those containing the endo-toxins chiefly, such as Koch's "new" tuberculin, T.R. (3) Mixtures of endo- and exo-toxins, the most important of which are Koch's bacillary emulsion, B.E., the sensitised bacillary emulsion, S.B.E. and Béranek's tuberculin, T.Bk.

The Therapeutic Substances Act, 1925, restricts the term "tuberculin" to the first of these groups, and recommends the name "tubercle vaccine" for any substance obtained directly from the bacterial bodies.

Tuberculins and tubercle-vaccines may be prepared from human or bovine bacilli; if from the latter the letter P. (perlsucht) placed before the letters indicating the variety of tuberculin, signifies its origin, e.g. P.T. In administration, some aim at producing reactions and establishing tuberculin tolerance by giving large doses at fairly frequent intervals; others believe in minute doses at longer periods, the chief object being to avoid the production of any reaction. The actual doses are either measured in milligrammes of dried tubercle bacilli, or in cubic centimetres or cubic millimetres of the fluid tuberculin. The usual method is to measure the doses in cubic centimetres or fractions thereof, and to make the necessary dilutions in a series of bottles. Smaller initial doses should be used with a strong tuberculin such as the B.E. than with the weaker ones such as the T. or T.A.F. Thus, if adopting minute doses, 800.000 mgrm. of B.E. would be a suitable initial dose, then for T.R.

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$\frac{1}{100,000}$ mgrm. would be used and $\frac{1}{1000}$ mgrm. of T., B.F., or T.A.F. As the different tuberculins are supplied by the makers in varying strengths, 0.001 c.mm. of the original fluid is equivalent to the above doses.

Those who believe in minute doses commence with 0.001 c.mm. and work up to 0.1 c.c., whereas others commence with 0.5 to 1 c.mm. and increase to 1 c.c. The tuberculin should be injected subcutaneously, and a careful observation kept for local, focal and general reactions. Tuberculin, if used injudiciously, can be productive of harm. At Midhurst Sanatorium it was tried for some years, and no better ultimate results were obtained than in the "non-tuberculin" years. It seems wise, therefore, to use tuberculin only in very carefully selected cases, thus T.R. does at times appear beneficial in chronic tuberculosis, promoting the formation of fibrous tissue and leading to the disappearance of tubercle bacilli from the sputum.

Various attempts have been made to remove as far as possible the fatty and waxy constituents from the tubercle bacilli before preparing a vaccine from it. Much and his collaborators have employed partial antigens or partigens derived from tubercle bacilli, singly or in varying combinations. Some good results are on record from both of these methods, but on the whole their use has so far been disappointing.

Grasset has recently recorded success with a preparation described as "tubercle endotoxoid," which he claims is freed from the toxic factor in ordinary tuberculins.

(b) *Passive immunisation*.—The various serums such as those prepared by Marmorek and Maragliano have not proved successful, and this applies to the "contra-toxin" of Mehnarto and the "I.K." (immune bodies) of Spengler.

8. OPERATIVE TREATMENT.—(a) *Artificial pneumothorax*.—This mode of treatment is now becoming more generally adopted in selected cases. It is increasingly employed in cases of early disease, where there are indications of incipient softening or of recent cavitation. In early cases without softening it is not as a rule used, unless there are indications of rapid spread or of pleural involvement when it is wise to start before adhesions have formed. In extensive bilateral disease it may be dangerous. If old and dense pleural adhesions are present, it is impracticable. If there is much emphysema or cardiac embarrassment, it involves risk. It is also of value in certain cases of repeated severe hæmoptysis. Tuberculous laryngitis or enteritis are not contra-indications, providing that other conditions are suitable. Sterile air is introduced into the pleural cavity, and the lung allowed to collapse. The method of induction of artificial pneumothorax is as follows: A preliminary subcutaneous injection of $\frac{1}{2}$ grain of papaveretum (omnopon) is given half an hour before the start. The patient lies on the sound side with the head low and supported on a pillow. A second pillow is placed under the chest to expand the intercostal spaces. The skin and the tissues down to the pleura are anaesthetised with 2 per cent. procaine hydrochloride solution after the application of iodine. The site usually chosen is in the sixth intercostal space in the mid-axillary region. A special pneumothorax needle, attached by a rubber tube to the pneumothorax apparatus, which is carefully examined to see that it is in proper working order, is then pushed through the intercostal space until the pleural cavity is reached. The apparatus is then adjusted so that the intrapleural pressure can be observed. No air should be introduced until the manometer shows a normal negative

pressure range with inspiration, of 5 to 10 or more cm. of water. This is the test of entry into the pleural space, and when this is established 200 to 300 c.c. of sterile air may be allowed to enter the pleural cavity. The final pressures are then recorded and the needle is withdrawn. A refill is given next day and another after two more days, the quantities of air introduced being determined by the final pressures, which should be kept slightly negative. Subsequent refills are gradually spaced out to a week, then ten days and later to two, three and four weeks' intervals. The usual custom now is to maintain the collapse for three years or even longer. If the condition of the patient is satisfactory, re-expansion may then be permitted cautiously. It should be remembered that after expansion pleural adhesion almost invariably occurs and the treatment by artificial pneumothorax cannot be repeated. In some cases of bilateral disease, which is active but not very extensive in either lung, a cautious use of bilateral artificial pneumothorax has proved practicable and helpful, but very great care is necessary in adjusting the pressures.

There are certain dangers in the procedure. These are now rare, and they can usually be prevented by careful attention to the technique. Death has occurred from pleural shock when the needle has reached the pleura and before any air has been introduced. Adequate anæsthesia of the pleura is the only known method of eliminating or minimising this risk. If the lung is adherent to the chest-wall, owing to pleural adhesions, or if the needle is pushed in too far, it may be inserted into the lung or into a pulmonary cavity; the manometer will then show a swing above and below the zero line instead of entirely below it. Under these circumstances, no air should be allowed to enter. The needle may be inserted into a blood vessel. In this case the manometer pressure will rise above zero, and blood may appear in the glass section inserted in the rubber tube leading from the manometer to the needle. The needle should be withdrawn immediately, lest air should enter the vessel.

If the pleura is found to be adherent at the site of the first puncture, another attempt may be made elsewhere, *e.g.* just below the inferior angle of the scapula. This spot may be selected for the initial puncture in left-sided cases where there is marked cardiac displacement. In cases in which localised band or cord adhesions prevent adequate collapse, it is often possible to cut them by electrocautery or diathermy through an operating thoracoscope, thus ensuring completely effective collapse. This is called internal pneumolysis.

(b) *Oleothonax*.—Sterilised olive oil or liquid paraffin with a varying percentage of gomenol is sometimes used to maintain the collapse started by artificial pneumothorax. Paraffin is used when the mediastinum is unduly mobile, since it tends to thicken the pleura. The oil or paraffin is introduced by means of a Dieulafoy syringe, and air is withdrawn at the same time by a reversed artificial pneumothorax apparatus. Whichever is used, it is important to test the sensitiveness of the pleura by small injections of 2 to 5 c.c. to start with. In later injections the amount introduced may be gradually increased to 200 c.c. or more. Oleothonax is now for the most part restricted to cases of therapeutic pneumothorax tending to obliterate from spreading adhesion, and those with mobile mediastinum.

(c) *Evulsion or crushing of the phrenic nerve*.—This is now often performed

in order to produce basal collapse, but it helps to produce relaxation at the apex and may aid in the contraction and closure of a cavity. Division alone is not sufficient; it is desirable to remove as long a stretch of the nerve as possible. In recent years temporary paralysis has been induced by crushing the nerve, the effects lasting about 6 months. This is often carried out instead of evulsion, since it does not prejudice further surgical treatment subsequently.

(d) *Thoracoplasty*.—If owing to adhesions it is impossible to collapse the lung temporarily by an artificial pneumothorax, permanent collapse by thoracoplasty may be considered. In the Sauerbruch operation, the posterior 3 or 4 inches of the ribs from the first downwards are removed, even to the tenth or eleventh, the lower level being determined by the extent of the compression of the lung it is desired to produce and therefore by the amount and situation of the diseased area. This operation is now performed in two or three stages, and generally under local anaesthesia. Good lateral collapse is usually obtained by this operation, but antero-posterior compression is less complete. Semb's operation is more satisfactory and is now more frequently employed. The first, second and third ribs are completely removed and the fascial supports of the apex of the lung are divided, which is thus allowed to sink down, producing a concentric collapse. The second and if necessary a third stage are similar to those in the Sauerbruch operation.

(e) *Apicolysis (extrapleural pneumolysis)*.—Successful local collapse can sometimes be attained by introducing some extraneous material like paraffin between the chest wall and the parietal pleura over a local area of disease, or a cavity which is not too near the pleura.

(f) *Extrapleural pneumothorax*.—In some cases where artificial pneumothorax fails owing to extensive apical adhesions, an extrapleural pneumothorax may be induced. A portion of the fourth rib is removed near the spine and the parietal pleura with the adherent lung is stripped away from the chest wall through the endothoracic fascia. The separation is carried down to the hilum on the mediastinal aspect, to the eighth rib posteriorly and to the fourth costal cartilage in front. The wound is then carefully closed and sutured. The extrapleural space thus produced is maintained by repeated refills of air, at first very frequent, subsequently at longer intervals. After about a month high positive pressures up to +18 and +24 must be maintained, otherwise the space obliterates. This procedure, though sometimes successful, seems less generally satisfactory than apical thoracoplasty. On the other hand it involves less shock than the operation of thoracoplasty, and is therefore practicable in some cases where thoracoplasty cannot be considered. The existence of an intrapleural pneumothorax below where it is necessary to keep the lower lobe under control does not contra-indicate it.

9. SYMPTOMATIC TREATMENT.—When cough is ineffective it may be relieved by a sedative lozenge or linctus containing diamorphine or codeine, or by the well-known liquorice lozenge. If there is difficulty in bringing up the sputum, a simple saline mixture is of value, such as sodii bicarb. grs. 10, sodii chlorid. grs. 3, sp. chlorof. min. 10, and aq. anisi ad fl. oz. 1.

Pain in the chest is usually alleviated by local application of pigmentum iodi, liniments, mustard leaves or other counter-irritants.

Night sweats.—The windows should be widely opened at night. A pill containing zinc. oxid. grs. 3 and ext. belladonn. succ. gr. $\frac{1}{4}$ is often of value.

Picrotoxin, agaricin and strychnine have also been used. A rush mattress as used in the tropics has been recommended.

Fever.—Rest in bed up to the extent of “absolute rest” is the best means of lowering the temperature. Antipyretic drugs have no effect upon the course of the disease, but may alleviate malaise. Amongst these may be mentioned aspirin and cryogenin.

Slight hæmoptysis, in which the sputum is only streaked, calls for no special treatment. Moderate hæmoptysis, with expectoration of 3 or 4 ounces of blood, requires more active measures. The patient should be put to bed, a saline aperient administered, and if there is anxiety or alarm a sedative drug should be given, such as heroin or morphine. In profuse or persistent hæmoptysis the patient should be confined strictly to bed, and if it is known from which side the bleeding has occurred, it is best to lie on this side. If the cough is troublesome, or if the patient is alarmed, morphine gr. $\frac{1}{4}$ to $\frac{1}{2}$, or diamorphine, hydrochloride gr. $\frac{1}{2\pi}$ to $\frac{1}{\pi}$ should be injected subcutaneously. The food is best given cold, and may be iced; no alcohol must be taken. A course of calcium lactate grs. 10, t.d.s., may be commenced; but its action is somewhat uncertain. If the bleeding persists, various other remedies should be tried, these include the inhalation of amyl nitrite, or the injection of hæmoplastin, coagulen ciba, horse serum, or emetine hydrochloride subcutaneously. Congo red, given intravenously, has proved of value in some cases. Ergot and adrenaline are both contra-indicated. If the hæmorrhage is still unchecked, or is frequently repeated, the advisability of establishing an artificial pneumothorax must be considered.

Gastro-intestinal symptoms.—Anorexia or dyspepsia can often be relieved by changes in diet, or by the administration of suitable drugs. Alkalis and gentian are especially valuable, and when hypochlorhydria is present, dilute hydrochloric acid (min. 10–30) should be given well diluted after meals. Digestive ferments, such as taka-diastase or papain, may be required at times. All tendency to constipation should be checked by laxatives, and if diarrhœa develops, avoidance of diet leaving bulky or irritating residues should first be tried, before administering drugs containing lead, opium, bismuth or tannic acid.

Insomnia is often a troublesome symptom, and every endeavour should be made to obtain a good night's rest by administration of mild hypnotics, and by relieving distressing cough and pain.

The treatment of the complications of pulmonary tuberculosis is described under their respective headings. The after-care of patients discharged from sanatoria is an important subject, to which considerable attention is being devoted, and involves a consideration of the advisability of establishing training centres or industrial colonies for consumptives. These are proving of very great value.

THE PULMONARY MYCOSES (PNEUMONOMYCOSES)

A number of fungi produce pulmonary lesions. Considerable confusion exists in regard to their nomenclatures, and at the present time it is difficult to give accurate accounts of them. The pulmonary mycoses have one feature in common, in that they produce chronic pulmonary lesions

practically indistinguishable clinically from those of the chronic forms of pulmonary tuberculosis.

Among the varieties of mycotic infection at present separated clinically may be mentioned—(1) Actinomycosis and other streptothrix infections; (2) Aspergillosis; (3) Blastomycosis; (4) Sporotrichosis; (5) Moniliasis; (6) Mucormycosis.

STREPTOTRICHOSIS (ACTINOMYCOSIS)

Ætiology.—The general characters of the streptothrix group of organisms are described in the section on Actinomycosis. It is now recognised that more than one of these may be pathogenic for man, and some authors give separate descriptions of the forms due to the various streptothrix organisms. At the present time there seems little advantage in so doing, since the important point in regard to treatment is to recognise that the morbid process is due to some form of streptothrix infection, the identification of the variety being a pathological refinement. The manner of infection is at present obscure. The organism is now believed to be present not infrequently in the alimentary tract, but the conditions favouring its invasion of the tissues are not known. A large proportion of cases show the first lesions in the head and neck regions, but primary pulmonary cases occur, and are probably more frequent than is generally recognised.

Pathology.—The streptothrix group of organisms produces an inflammatory reaction which leads to the formation of granulomatous tissue. This, like the granuloma of tubercle, is very liable to undergo secondary changes producing small areas of pus formation or leading to fibrosis. Unlike tuberculosis, however, streptotrichosis tends frequently to transgress anatomical limitations and spreads by contiguity. In the primary pulmonary cases the distribution of the lesions is at first very similar to that of tuberculosis, and the disease may extend in an identical manner. In the forms due to spread from other organs such as the liver, the base of the lung may be first involved, while in cases extending down from the neck the path of the infection is apparent.

Owing to the tendency of the lesions to spread by contiguity, subcutaneous abscesses may form and simulate caries of the ribs. Pleural adhesion is the rule, but occasionally empyema results. When a subcutaneous abscess ruptures or is opened, the characteristic “sulphur granules” may be found, although this is not invariable. The skin around the sinuses which result is often puckered in a somewhat characteristic fashion.

Symptoms.—These are in general identical with those of the chronic forms of pulmonary tuberculosis, such as cough, expectoration, which may be offensive, dyspnoea, fever and night sweats. The occurrence of local abscesses under the skin or the presence of the organism elsewhere may give rise to special features; but these are late developments in primary pulmonary cases.

Complications and Sequelæ.—These are usually due to the other localisations of the organism; but, in addition, empyema and bronchiectasis may be mentioned.

Course.—This is progressive, and leads eventually to asthenia, emaciation and death.

Diagnosis.—This can only be established by the discovery and identification of the organism in the sputum and the discharge. The characteristic "sulphur grains" are not invariably present, and may escape notice unless looked for carefully. In any obscure case of pulmonary disease in which tubercle bacilli are not found after repeated search, the possibility of streptotrichosis should be considered, and direct films should be specially examined.

Prognosis.—This is serious, although some cases respond well to treatment.

Treatment.—Large doses of potassium iodide should be administered, commencing with 5 or 10 grains three times a day, and increasing until the dose reaches a drachm or even more thrice daily. In addition, collosol iodine (Crookes) may be given intravenously in doses of 5 c.c. at least once a week. If the organism grows well in culture, a vaccine may be prepared and employed cautiously, especially if the iodides do not act satisfactorily. A stock vaccine may be helpful in other cases. Surgical treatment of local abscesses or of empyema may be required. External application of a radium pack is sometimes useful.

PULMONARY ASPERGILLOSIS

Ætiology.—Infection of the bronchi and lungs sometimes occurs by the *Aspergillus fumigatus*, more rarely by the *A. nidulans*. The disease has been most frequently observed in France. It occurs among pigeon breeders and hair sorters and combers. The former acquire the disease from the process of artificial feeding, from grains in the mouth to the beak of the bird; the latter from the use of rye flour in cleaning the hair. Millers and farm labourers have also been the subjects of the disease.

Pathology.—The fungus induces nodular formations in the lung tissue somewhat resembling aggregated tubercles. Bronchitis, patchy lobular consolidation and fibrosis result. Emphysema, bronchiectasis and cavity formation may follow. A secondary aspergillosis may occur in chronic cases of bronchitis or lung disease, but is of little clinical importance.

Symptoms.—Primary aspergillosis produces symptoms similar to those of bronchitis, broncho-pneumonia or pulmonary tuberculosis, according to the localisation and degree of the lesions. The sputum may be blood-stained, or definite hæmoptysis may occur. There is generally wasting with irregular fever.

Course.—Acute broncho-pneumonic forms may be fatal in a few weeks or months. The chronic lesions may extend to years, and arrest with fibrosis is not uncommon.

Diagnosis.—The condition has to be differentiated from pulmonary tuberculosis, and from other varieties of pneumonomycosis. This depends upon the recognition of the fungus by microscopical and cultural examination of the sputum.

Treatment.—This consists in avoiding further infection, and giving large doses of potassium iodide as in streptotrichosis. Open-air measures and general tonic treatment are also to be recommended.

OTHER MYCOTIC INFECTIONS

Fungi of the genera *Blastomyces* (*Oidium*, *Coccidioides*) and *Sporotrichum* are well known to produce cutaneous affections simulating chronic gummatous or tuberculous lesions. They may also give rise to pulmonary disease producing symptoms like those of tuberculosis.

Castellani has described various broncho-pulmonary conditions due to species of the genus *Monilia*, including the "tea-tasters' cough" and "tea-factory cough." Another fungus, *Mucor mucedo*, has been found in the sputum, and is regarded as pathogenic to man.

All these moulds produce bronchitic symptoms and mild infections, while more severe forms simulate pulmonary tuberculosis. The diagnosis in each case depends upon the recognition of the fungus, and the treatment recommended is large doses of potassium iodide.

SYPHILIS OF THE LUNGS

Ætiology.—Clinically recognisable pulmonary syphilis is a rarity; but syphilitic lesions occur in the lungs in both the congenital and acquired forms of the disease.

Pathology.—Even post mortem it is often difficult to establish the syphilitic nature of the pulmonary lesions found in cases of syphilis, owing to the fact that they tend to the formation of scars presenting no characteristic features.

Congenital syphilis.—The essential changes are—(1) Round-celled infiltration with eventual fibrosis, starting round the bronchi and spreading to the inter-alveolar framework; (2) periarteritis of the smaller arteries; and (3) desquamation and degeneration of the epithelium of the alveoli and bronchi. Gummata may be present, but are rare. Spirochætes can be demonstrated in the lesions by Levaditi's method. The microscopic appearances comprise the "white pneumonia" of Virchow and an interstitial pneumonia, which is commoner, although both conditions are frequently associated. White pneumonia is found in premature or still-born infants, and in those dying soon after birth. The condition may be widespread or localised. The affected areas are firm, consolidated, smooth and greyish-white in colour. There are no interstitial changes, and the consolidation is due to the filling of the alveoli with desquamated, degenerating epithelial cells.

In the commoner interstitial form the lung is firmer, harder and darker grey in colour, and the connective tissue is mainly involved. To this condition the term "pancreatisation of the lung" has been applied by Rogers.

Acquired syphilis.—Syphilitic lesions of the bronchi have already been described in the section on diseases of the bronchi. Gummata may occur in or around the intra-pulmonary bronchi or in the lung tissue. They may be single or multiple, and vary in size from that of miliary granules to a hen's egg. They are said to be more common in the deeper parts of the lung near the roots and in the lower lobe. They undergo changes similar to those occurring in gummata elsewhere, but tend more to fibrosis and contraction than to softening. Owing to these secondary changes, the following

conditions may result: broncho-pneumonic processes, widespread fibrosis and contraction with pleural adhesion, bronchiectasis and occasionally excavation.

Symptoms.—Small gummata may be latent and give rise to no symptoms or signs. When fibrosis occurs, they are similar to those of pulmonary fibrosis from other causes. It is generally recognised that in rare cases a destructive process occurs, formerly called "syphilitic phthisis," and almost exactly similar in its clinical manifestations to those of caseous or fibro-caseous tuberculosis.

Complications and Sequelæ.—Syphilitic lesions in the larynx, trachea or bronchi may complicate the course. Bronchiectasis has already been mentioned, and tuberculosis may occur as a complication.

Diagnosis.—This is often difficult and sometimes inconclusive. Obscure pulmonary signs in a syphilitic subject should arouse suspicion. The Wassermann reaction should be determined, and other indications of syphilis looked for in all fibrosing and destructive lung conditions when no tubercle bacilli are found in the sputum. The difficulty of diagnosis is increased by the association of syphilis and tuberculosis mentioned above.

Course and Prognosis.—Where the lesions are localised and can be recognised early, the course is favourable if anti-syphilitic treatment is applied. Where fibrotic changes occur, leading to bronchiectasis, the course is less favourable, and in the destructive form it is serious. An inter-current tuberculous infection increases the gravity of pulmonary syphilis.

Treatment.—When a diagnosis of pulmonary syphilis has been established, vigorous anti-syphilitic treatment should be carried out. Its beneficial effect is undoubtedly promoted by open-air treatment. In cases where tuberculosis coexists with syphilis, anti-syphilitic treatment is strongly recommended, especially by French physicians.

NEW-GROWTHS IN THE LUNGS

Both simple and malignant tumours may occur in the lungs, the latter being the more common.

Ætiology.—Malignant tumours occur more frequently in the male sex in the ratio of five to one; carcinoma is rare before the age of 40, but sarcoma may develop in earlier years. Simple tumours may arise at any age, but are found chiefly in adult life. The exciting cause is unknown. In some cases of malignant growth there is a history of thoracic trauma or disease.

Pathology.—Simple tumours found in the lungs usually arise in the bronchial mucous glands or in the bronchi. They include adenoma, fibroma, lipoma and chondroma (see p. 1141).

Malignant tumours may be primary or secondary. The primary growths are carcinoma, sarcoma or endothelioma. Carcinoma arises in the bronchi, usually as a columnar-celled growth. A variety of bronchial new-growth formerly regarded as a lympho-sarcoma is now called an oat-celled tumour. It is probably derived from the basal cells of the bronchial mucous membrane. Squamous-celled growths of the bronchi are rare and are now said to be derived from basal cells. Round-celled and spindle-celled sarcomata growing from the pulmonary connective tissue are met with, while endotheliomata are

usually derived from the endothelium of blood vessels and lymphatics, or from the pleura. A primary carcinoma of the breast, cesophagus or mediastinum may directly invade the lungs. Secondary carcinoma may have its primary focus in the breast, stomach, intestines, liver, pancreas or prostate, whereas a secondary sarcoma most often results from metastasis of a primary bony growth. Chorion-epithelioma and hypernephroma also give rise to secondary deposits in the lungs.

Primary malignant tumours are unilateral; but secondary growths are often multiple and diffuse. Dissemination in the lungs may occur by spread along the bronchi or vessels, and a condition of miliary carcinomatosis is at times produced. The pleura is often affected by direct extension. Infiltration of, or pressure upon, the mediastinal structures frequently occurs.

Symptoms.—Simple tumours except adenomata are pathological curiosities and, as a rule, only produce symptoms when they cause obstruction of a bronchus or press on mediastinal structures (see pp. 1046 and 1280).

The early symptoms of malignant growths are slight, and consist of malaise with, perhaps, cough and expectoration. Later, when the growth becomes more extensive and exerts pressure on, or involves the larger bronchi, mediastinum or pleura, they are more noticeable. Pain, dyspnoea and loss of weight with cachexia usually develop, and the cough and expectoration are more marked. The latter is often of the typical "currant jelly" or "prune juice" appearance due to altered blood. Microscopically, groups of large fatty cells, or irregular epithelial cells may be seen. Malignant cells may be found in 60 per cent. of cases by Dudgeon's wet method. There are usually no definite physical signs until the tumour causes pressure upon the bronchi, mediastinum or deep thoracic veins or nerves. The chest-wall may bulge locally, owing to the presence of a growth near the surface, or it may be retracted if a main bronchus is obstructed. An actual subcutaneous swelling caused by the tumour eroding through the chest-wall may be visible. Enlarged veins often run across the chest, and one or other arm may be swollen or oedematous if there is mediastinal obstruction. Vocal fremitus is often unaffected; but is increased when the growth is near the surface, and diminished if pleural effusion has occurred. The percussion note over a moderate-sized tumour is impaired and may be extremely dull; more often the dullness is due to collapse of the lung. The breath-sounds vary with the size and position of the growth, and with the displacement or pressure effects produced. They may be weak, or loud and stridorous. The stridor is usually unilateral. Adventitious sounds depend upon the presence of complications such as bronchitis. Some degree of fever often occurs. The supra-clavicular and axillary glands are not infrequently enlarged, and evidence of malignant disease may be present in other parts of the body such as the abdomen.

One special variety of apical carcinoma is the superior pulmonary sulcus or Pancoast tumour, which gives rise to a somewhat characteristic or suggestive clinical picture. The chief symptoms are pain in the shoulder, inner side of the arm and forearm together with weakness and wasting of the small muscles of the hand. Paralysis of the cervical sympathetic on the same side develops. There is usually localised dullness at the extreme apex. Radiological investigation reveals a sharply defined apical shadow with destruction

of the posterior part of the first three ribs and sometimes localised vertebral erosion. Pancoast suggests that these tumours may arise from remnants of the fifth branchial cleft.

Complications and Sequelæ.—Bronchitis is nearly always present in some degree. Pulmonary collapse, fibrosis, bronchiectasis, emphysema, gangrene, hæmoptysis, pleural effusion, abscess and empyema are sometimes observed. The effusion is frequently bloodstained. In cases of primary malignant disease of the lungs, secondary deposits may occur in other parts of the body such as glands, brain, suprarenals, heart and bones.

Course.—This is progressive, the patient gradually losing strength and dying from cachexia or some intercurrent affection.

Diagnosis.—This is difficult in early cases, and not easy in some advanced ones. It not infrequently happens that metastases, especially in brain or bone, afford the earliest manifestations to be recognised. Difficulties may arise in connection with pulmonary tuberculosis, fibrosis and gumma of lung, aneurysm, pericardial and pleural effusion and enlargement of the mediastinal glands due to Hodgkin's disease or tuberculosis. The whole body should be searched for evidence of malignant disease elsewhere. The sputum should be examined repeatedly for tubercle bacilli and for cellular elements, and an X-ray examination made of the chest. By the stereoscopic method excellent evidence of pulmonary neoplasms is often obtainable. Lipiodol injection and X-ray examination or tomography may demonstrate the obstruction of a bronchus by the growth which often presents a tapering or "rat-tail" appearance. Bronchoscopy may also serve to establish the diagnosis especially in bronchial carcinoma. Temporary artificial pneumothorax may be helpful in diagnosis, particularly in differentiating simple tumours in the periphery of the lung, growths in the mediastinum and in the chest-wall.

The Pancoast tumour may give rise to special difficulty. It has to be differentiated from syringomyelia, cervical rib, apical pulmonary tuberculosis and secondary sarcoma.

Prognosis.—Apart from those cases in which early recognition may in suitable conditions render lobectomy, with removal of the growth, possible, this is hopeless, death occurring in a few weeks, or being delayed for two or three years.

Treatment.—Simple tumours are often capable of complete removal with gratifying success.

In malignant growths lobectomy or dissection pneumonectomy with complete removal of the growth is only practicable for cases recognised early in which there are no secondary deposits.

Radon seeds are sometimes used; when practicable they are inserted into the growth through a bronchoscope. In other cases they may be introduced directly into the growth by thoracotomy. Treatment by deep X-ray application may be useful by diminishing local pressure and relieving symptoms chiefly in sarcoma or oat-celled carcinoma. Cure by these methods is rare.

In cases unsuitable for lobectomy and radiation treatment this can only be palliative and symptomatic. Useless cough should be checked by sedative lozenges or a linctus. Dyspnoea due to pleural effusion may be relieved by tapping with or without air replacement; but the fluid often reaccumulates

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rapidly. Pain should be relieved by analgesic drugs, and in the later stages those containing opium or its alkaloids may be required.

PARAGONIMIASIS

Synonyms.—Pulmonary Distomatosis; Lung Fluke Disease; Endemic Hæmoptysis; Parasitic Hæmoptysis.

Ætiology.—(See p. 306).

Pathology.—The flukes settle down in the lungs and form burrows. These burrows may coalesce and give rise to cystic swellings, varying from $\frac{1}{2}$ to $1\frac{1}{2}$ inches in diameter. These in turn develop fibrous sheaths and may give rise to abscess formation or pleurisy. The adult fluke is hermaphrodite, and lays numerous eggs which measure $100 \times 70 \mu$. These are coughed up in the sputum, and are easily recognisable owing to their large size. The adult parasites are also occasionally found in the brain, liver, lymph glands and peritoneal cavity.

Symptoms and Complications.—The onset of symptoms, after infection has taken place, is insidious, with cough and expectoration. The latter is very constantly blood-stained, and there may be profuse hæmoptysis. Secondary pleurisy occurs when the cysts reach the surface of the lungs, causing pain. Examination may reveal no abnormal signs, at most there are a few scattered râles, together with signs of dry pleurisy at one point. Later in the disease the characteristic signs of the various complications may appear.

For general, abdominal and cerebral symptoms and complications, see pp. 306, 307.

Course.—This is chronic: the disease often persists for years, without giving rise to any acute disturbance apart from periodic hæmoptysis.

Diagnosis.—Distinction from other forms of hæmoptysis is accomplished by discovering the ova in the sputum. To facilitate the examination a little 0.1 per cent. sulphuric acid should be added to it.

Prognosis.—The immediate prognosis is good, the ultimate unfavourable, as there is considerable difficulty in eliminating the parasites, and permanent damage is wrought in the lungs.

Treatment.—Prophylaxis is important where the disease is endemic. No bathing should be allowed in infected rivers, and all water used for drinking or washing should be boiled or filtered. Crabs should not be eaten. When the disease has developed the patient should move from the infected area. Potassium iodide (grs. 10–20, t.d.s.) is recommended, but other treatment is symptomatic.

CONGENITAL CYSTIC DISEASE OF THE LUNG

Ætiology.—Congenital cysts of the lung may be met with in infants, children or adults. There is no infective or parasitic cause, and as in some instances cysts have been found in the fœtus they are considered to be due to developmental errors.

Pathology.—The following varieties are described: 1. The large balloon

cyst. This may completely compress a lung in an infant or young child. 2. The solitary cyst. This may occupy half the lung-field. 3. Multiple medium-sized cysts, often situated near the root of a lung. 4. Multiple small cysts. These cause a honeycomb appearance of the lung resembling bronchiectasis.

It is probable that in all cases the cysts are of bronchial origin. The lining membrane of the cyst is uniform, the cells having the characters of bronchial epithelium. Microscopically, the irregular distribution of the cartilage, muscle, elastic tissue and mucous glands in the supporting tissues differentiates congenital cysts from bronchiectatic cavities. The cysts may contain air only, or the contents may be watery, mucoid, or purulent if they become infected.

Symptoms.—These vary with the variety of cyst present. The large balloon cyst, met with in infants or young children, may result in severe respiratory and cardiac distress. In such cases there is cyanosis, dyspnoea and displacement of the trachea, mediastinum and heart to the opposite side of the chest. The percussion note over the cyst is hyper-resonant and the breath-sounds are absent. Solitary cysts often give rise to no symptoms and are only discovered on routine X-ray examination. When infected the clinical features may resemble those of lung abscess or bronchiectasis. With multiple medium-sized or small cysts no symptoms usually appear until infection occurs, though hæmoptysis may occur early. When infected, toxæmic symptoms develop, such as loss of weight, irregular fever, cough and expectoration which is sometimes offensive. Clubbing of the fingers may then soon be noted. On examination scattered areas of slight dullness, weak air entry with a few persistent râles may be detected.

Course, Complications and Sequelæ.—The onset of complications usually leads to the development of symptoms which call for investigation. Thus spontaneous pneumothorax may result from rupture of a cyst. In other cases suppuration occurs in the cyst with the formation of lung abscess, bronchiectasis or empyema. Cerebral abscess may be a late sequel.

Diagnosis.—This is suggested by X-ray and by lipiodol or neo-hydriol examinations and possibly tomography. If the space in the cyst is free from fluid the X-ray appearances must be differentiated from those of pneumothorax, an emphysematous bulla, a thin-walled tuberculous cavity or, in some cases, a diaphragmatic hernia. If the cyst contains fluid, further investigations are required to exclude the presence of such conditions as lung abscess, encysted pleural effusion or empyema, hydatid cyst, dermoid cyst or a blood cyst. A definite diagnosis can sometimes only be made after operation by microscopical examination of a portion of the cyst.

Prognosis.—This varies with the type of cyst present, the development of complications, and the treatment adopted. In many cases the prognosis is good, apart from rupture or infection. In the large balloon cyst there is risk of sudden death during an attack of distension.

Treatment.—The large balloon cysts which are causing respiratory and cardiac embarrassment call for immediate treatment by the insertion of a needle. Subsequently the only hope of recovery lies in pneumonectomy.

When the cysts are infected, treatment by postural drainage should first be adopted. Failure usually follows attempts at surgical drainage or collapse operations. If the cysts are unilateral and infected, the only hope of cure lies

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in radical removal of the portion of lung involved, either by lobectomy or by pneumonectomy; and so in bilateral cases it necessarily follows that no radical cure is possible.

HYDATID DISEASE OF THE LUNG

Hydatid cysts may develop in the lung in patients infected by the ova of the *Tænia echinococcus*.

Ætiology.—Man is the intermediate host of this parasite, and becomes infected directly or indirectly from the dog. The modes of infection and the life-history of the parasite are elsewhere considered (p. 314). Males are more often affected, and the condition is commoner among the poor than the well-to-do. It is rarely seen in this country except in patients from abroad, especially from Australia.

Pathology.—Hydatid cysts have been described in the lungs in from 5.6 to 16.8 per cent, of cases of hydatid disease in different parts of the world. The right lung is more often the site of the disease than the left, and the cyst is usually basic, though it may occur in the upper parts of the lung. It is generally supposed that infection of the lung is usually secondary to the liver, the ova reaching the lung through the diaphragm; but the occurrence of primary lung hydatid suggests the possibility of the embryo gaining access to the general blood stream, and thus reaching the lung by the pulmonary artery. There is, as a rule, a single cyst in the lung, but multiple or multilocular cysts are occasionally observed. The cyst may become as large as a cricket ball, but usually ruptures before it reaches this size. It has the same structure as hydatid cysts of other organs, with ectocyst and endocyst. It may develop brood capsules and daughter cysts, but is often sterile in this situation.

The reactive changes in the lungs are at first irritative and congestive, but eventually some fibroid changes occur, producing a more or less definite fibroid capsule around the ectocyst. The overlying pleura may become inflamed, thickened and adherent when the cyst grows near the surface. Rupture may occur into a bronchus, into the pleura, pericardium or peritoneum, or occasionally into the aorta or pulmonary vein. Rarely the contents of a small cyst may become inspissated, thus producing spontaneous cure.

Symptoms.—Until the cyst becomes large enough to cause irritation, there may be no symptoms, but sooner or later cough and expectoration develop. The latter is generally mucoid, and frequently bloodstained. Dyspnoea becomes apparent and pain results if the pleura is involved. The signs may be: diminished vocal fremitus, localised dullness and weak or absent breath-sounds and voice-sounds over a limited area, generally in the lower lobe. A few râles may be audible round the dull area. Occasionally with a large cyst there may be some bulging on the affected side, and "hydatid fremitus" has been described. The heart may be displaced in rare cases. Examination by the X-rays generally shows a suggestive rounded shadow with very little change in the surrounding lung, except in chronic cases where some fibrosis may be observed.

Some degree of eosinophilia is common but not invariable. When rupture into a bronchus occurs, there is usually sudden copious expectoration of watery

fluid containing hooklets. Daughter cysts and parts of the ectocyst may be coughed up and lead to dyspnoea and even suffocation from laryngeal obstruction.

After rupture, spontaneous cure may result if the ectocyst is expectorated. More commonly the cavity becomes infected and the symptoms and signs become those of chronic abscess (see p. 1179). Rupture into the pleural cavity produces great pain, dyspnoea, cyanosis and shock, similar to the condition induced by pneumothorax. Rupture into the pericardium or into a vein is usually quickly fatal. When rupture occurs into a serous cavity, urticaria and severe toxic symptoms sometimes develop.

Course.—This is generally progressive, though occasionally spontaneous cure occurs either before or after rupture. More commonly the cyst causes increasing pressure or irritative symptoms, and eventually rupture or suppuration produces acute manifestations.

Diagnosis.—The clinical features of pulmonary hydatid may be suggestive of pulmonary tuberculosis, pleural effusion or new-growth. Diagnosis may be difficult before rupture occurs; after this the discovery of hydatid hooklets or pieces of cyst-wall may establish the diagnosis. In suspicious cases the X-ray findings may be of great assistance, and confirmatory evidence may be obtained from cytological and serological examination. The former frequently shows eosinophilia, and the latter gives complement deviation when a suitable antigen, such as extract of hydatid cyst-wall, is used. A precipitin reaction may also be obtained with the fluid from another cyst. The Casoni intradermal test with the appropriate antigen has established itself as having special diagnostic value.

Prognosis.—The prognosis is serious owing to the risks of rupture and suppuration. Spontaneous cure is rare, but can occur. After rupture into a bronchus, recovery may ensue, but more commonly abscess formation results. Rupture into a serous cavity is frequently fatal. Early surgical treatment either before or after rupture improves the outlook.

Treatment.—Aspiration of the cyst, either exploratory or therapeutic, is to be avoided. If the cyst can be diagnosed or localised before rupture, the lung should be exposed by thoracotomy, the pleura stitched together and the cyst incised, the endocyst removed, and the cavity drained. Suppuration of a pulmonary hydatid must be treated as a pulmonary abscess.

THE PNEUMONIAS

The term pneumonia has been somewhat loosely applied to any inflammatory condition of the lung producing consolidation. When the consolidation affects large areas of lung uniformly it is described as lobar pneumonia, and when it is patchy or lobular in distribution it is called lobular or broncho-pneumonia.

1. LOBAR PNEUMONIA

Synonyms.—Croupous or Pleuro-Pneumonia.

Definition.—This is an acute infectious disease characterised by an inflammatory lobar consolidation.

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Ætiology.—*Predisposing causes.*—Pneumonia may occur at any age. It is common in children up to the sixth year, the incidence being about equal in the two sexes. It is commonest between the ages of 15 and 40, when there is a preponderance in the male sex of two or three to one. It is also a frequent terminal malady in the aged of both sexes. It may be doubted whether race has much influence, although in America and in the Rand mines the incidence and mortality among the black races are both high. Pneumonia is met with all over the world, but it is more rife in localities with changeable climate and cold winds. Its seasonal incidence is well marked; it is uncommon in the summer and autumn, and is most prevalent from November to March in this country. Although pneumonia is as a rule endemic and sporadic in its incidence, it is generally admitted that localised epidemics occur. Urban conditions, defective sanitation, overcrowding and insufficient ventilation all conduce to the incidence of pneumonia. It is not uncommon to obtain a history of several previous attacks. Although the disease often attacks those in normal robust health, there can be no doubt that debilitating conditions and diseases predispose to it, among them being chronic nephritis, diabetes, over-fatigue, exposure and alcoholic excess.

Exciting causes.—The exciting cause in most cases is the presence of the pneumococcus of Fränkel. It may be the only pathogenic organism found in the lung lesions and in the sputum, but not infrequently others, such as streptococci, staphylococci or Pfeiffer's bacillus are also present. Occasionally these organisms, and others, such as Friedländer's pneumobacillus, the *Bacillus typhosus*, the gonococcus and the *B. pertussis*, cause lobar consolidation; but these conditions should be regarded as varieties of secondary pneumonia, and differentiated from the acute primary condition now under consideration.

The pneumococcus.—The pathogenicity of the pneumococcus has been the subject of an interesting study by Cole, Dochez, Avery and Gillespie and more recently by Georgia Cooper and her co-workers. Originally three types were described; Types I, II, III., which together account for more than 50 per cent. of all cases. The remainder were included in a group referred to as Group IV. This has now been separated into 29 other types, making 32 in all, by means of serological reactions.

The American observers have shown that 40 per cent. of contacts with cases of pneumonia due to types I. and II. may harbour the corresponding organism for an average period of 23 days, and that they may develop pneumonia from it. They have further demonstrated that a convalescent patient may carry pathogenic pneumococci in his mouth for as long as 90 days from the onset of the disease. They have also found pathogenic pneumococci in the dust of rooms in which patients suffering from pneumonia have been nursed. The significance of this work is obvious. It confirms the view that pneumonia is an infectious disease, capable of being spread by carriers, by the convalescent patient, and by the dust of rooms.

Although the pneumococcus is the specific exciting cause, its activities are often determined by some other factor, such as chill, exposure, over-exertion or injury. The presumption is that these conditions lower the general resistance of the individual, and thus impair the defensive mechanisms. Post-operative pneumonia may be a further instance of this, but doubtless some supposed cases are in reality due to lobar collapse.

Pathology.—The pneumococcus is found in the pulmonary lesions and elsewhere when complications occur. In some patients it is found in the blood. These are referred to as bacteriæmic cases and are usually more severe and often associated with complications. Experimental investigations on animals indicate that the avenue of infection is to the lungs by way of the trachea and bronchi, the blood infection being secondary to the pulmonary lesion. Four stages are commonly described in the process by which the lung becomes consolidated and returns to normal, namely, engorgement, red hepatisation, grey hepatisation and resolution.

In the stage of engorgement the affected part of the lung is slightly enlarged, deep red in colour, and heavier than normal, although it still crepitates and floats in water. The pleura over it may be injected and lustreless and may even show early fibrinous exudate. On section, the hyperemia is obvious and there may be some cedema. On squeezing, frothy, bloodstained fluid exudes. Microscopically, the engorgement of the capillaries, the swelling and partial desquamation of the alveolar epithelium are the chief changes to be noted. In the stage of red hepatisation the affected area becomes completely consolidated, the general aspect on section being remotely similar to liver, hence the name hepatisation. The pleura is now notably inflamed and may be obscured by yellow fibrinous exudate. The hepatised area of lung is larger and much heavier than normal and bears the impress of the ribs upon it. On section, it is seen to be red in colour, solid and completely airless. It does not crepitate and it sinks in water. The lung tissue is found to be more friable than normal. On scraping the cut surface, which has a granular appearance, a reddish fluid is collected, containing small fibrinous plugs, which are practically alveolar casts. Microscopically, the alveoli are occupied by a coagulated exudate rich in fibrin and red blood corpuscles, with scanty leucocytes and a few larger cells derived from the alveolar epithelium. In the stage of grey hepatisation the lung tissue, although still solid, airless and non-crepitant, is greyish in colour, softer in consistence and still more friable. The surface of the section is less granular, and on scraping, a pale yellowish, almost puriform fluid is obtained. Microscopically, the blood vessels are found to be relatively empty, the alveoli are now incompletely filled, the fibrin and red corpuscles have largely disappeared, and the alveoli are occupied by leucocytes and desquamated alveolar cells. In the stage of resolution, the exudate becomes more liquid and its cellular constituents undergo fatty degeneration. The liquefied exudate is largely absorbed, although expectoration may possibly assist in its removal. The lung returns to its normal spongy state and the alveolar epithelium is replaced. Some pleural thickening or adhesion may, however, result. In very severe and fatal cases, the stage of resolution may be replaced by one of purulent infiltration, in which the lung becomes paler, softer and in places almost diffuent. The scrapings are practically purulent.

Although these four stages are described, it should be remembered that they are not sharply defined from one another, and that they only represent special appearances in a continuous process. Consequently, although the major part of the affected area of lung may be characteristic of any one of them, all four stages may be recognisable, especially in cases of a spreading type. The base is more often affected than other parts, and the right side more than the left, in the ratio of 3 to 2. The unaffected parts of the lung

may show some catarrhal bronchitis, or some degree of collateral hyperæmia or œdema. Pleurisy is an integral part of the affection, but it may proceed to serous or purulent effusion. Pericarditis and less frequently acute endocarditis may be found in fatal cases. Pneumococcal meningitis, arthritis and otitis are very occasionally observed. The liver and kidneys may show cloudy swelling, and the spleen is often slightly enlarged and soft. Jaundice may be observed, especially in right-sided cases. The right side of the heart may be engorged and dilated.

Symptoms.—The exact incubation period is not yet established, but it is short, being probably from 1 or 2 days up to a week. The onset is sudden and acute, with chill, shivering or rigor in the majority of cases. In children convulsions take the place of rigors. Vomiting at the onset is not infrequent, occurring in about one-third of the cases. Less commonly the onset is insidious, or is preceded by malaise and catarrhal symptoms. The temperature rises with the rigor, and as a rule a short, dry, irritating cough develops quickly, accompanied by a severe cutting pain on the affected side. The pain often becomes intense, and coughing may cause the patient great distress. The cough is frequently restrained as much as possible, and the breathing is rapid and shallow. By the second or third day the pain becomes less and the cough easier and more effective. Sputum, which at first is scanty, extremely viscid, tenacious and difficult to expectorate, now becomes more abundant, although remaining viscid. In typical cases it is characteristically rusty at this stage, containing mucus, altered red blood corpuscles, alveolar epithelium and large numbers of pneumococci. In a few instances a small but definite hæmoptysis occurs. Occasionally the sputum is thinner and of "prune juice" type.

Sleeplessness is often a distressing symptom, especially in the early and late stages. In some cases there are marked cerebral symptoms. Headache at the onset is common. Delirium is frequent, particularly in the asthenic type, in apical cases, and in alcoholics. In the latter it may be violent and is often like delirium tremens. The temperature is usually of high continuous type throughout, reaching 103°, 104° and even 105° F. or more on occasions, especially in the sthenic type. In the asthenic it is often of lower range. Defervescence is by crisis in about 60 per cent. of the cases. Crisis is commoner in sthenic patients in types I., IV. and VII., and occurs more often on the odd than on the even-numbered days of the disease. The most common day for the crisis is the seventh. It is rare before the third or after the ninth day. At the crisis the temperature falls to normal or subnormal in about 12 hours. The patient often sleeps soundly at this time and may sweat profusely; respiration is slower and easier and the pulse-rate falls. On waking, a dramatic change in the condition is usually noticeable. Pain and distress are ameliorated, cough is loose and easy, and the patient feels better, although weak. Looseness of the bowels and free diuresis are not infrequent, constituting the "critical evacuations." The crisis is sometimes preceded by a pseudo-crisis, in which a considerable fall of temperature occurs, with little or no improvement in the general condition. A slight post-critical rise of temperature of 1° or 2° F. is sometimes seen, but as a rule the temperature remains subnormal for a few days and slowly returns to normal. The pulse-rate may be slow for a time. Convalescence is generally rapid, although in cases which have had marked delirium, some mental confusion may be present for a day

or two. Defervescence by lysis is more common in asthenic patients. The temperature remits and may take from 2 to 4 days to reach normal or sub-normal levels.

The physical signs vary with the stage of the disease. At first there is some restlessness, but soon the patient assumes a dorsal decubitus, or lies more on the affected side. The cheeks are flushed, often markedly so on the side of the lesion. The eyes are bright, but the expression is one of pain or anxiety. A crop of herpes on the lips is very common. The tongue is thickly coated and white, becoming dry and cracked in bad cases at a later stage. The skin feels dry and pungently hot. The *alæ nasi* are in action, and in children a puff or grunt accompanies each expiration, while the pause follows inspiration, instead of expiration. The respiration and pulse-rate are increased, the former disproportionately, so that the pulse respiration ratio becomes 3 or even 2 to 1, instead of the normal 4 or 5 to 1.

In the early stage the pulmonary signs are slight. At the most there is lessened movement and diminished vocal fremitus over the affected area, with dubious impairment of note, weak air entry and possibly a few crepitations (*indux*), or pleural friction sounds, vocal resonance being unaltered. Of these, lessened air entry is probably the most common. Slight hyper-resonance of the opposite lung, with harsh breathing, may lead to error in diagnosis as to the side affected.

The signs of consolidation (*hepatisation*) are generally apparent on the second or third day, except in cases where the disease starts deeply (*central pneumonia*). There is definite limitation of movement on the affected side, which is, however, slightly increased in size, as can be demonstrated by mensuration. Vocal fremitus is markedly accentuated over the affected area, except in massive pneumonia, and friction fremitus may be palpable. The note on percussion is dull, but has not the resistant stony character of that over an effusion. The note above or below the consolidated area is sometimes *skodaic*. The breath-sounds are tubular, and a few crepitations may be heard, but frequently adventitious sounds are absent. In some cases a friction rub is audible. *Bronchophony* and *pectoriloquy* are usually very marked over the consolidated area. The breath-sounds in other parts may be vesicular or harsh, and a few rhonchi may be present. The heart is usually in its normal situation, but is sometimes slightly displaced away from the affected side. In later stages the signs of dilatation of the right heart may become apparent.

During resolution, which begins after the crisis or during lysis, the tubular character of the breath-sounds disappears, and they become at first bronchial and later harsh or vesicular. Coarse moist sounds, known as *redux* crepitations, are heard both with inspiration and with expiration. The dullness gradually diminishes, and the voice-sounds return to normal. In basal cases, in which the diaphragmatic pleura is involved early, there may be pain, tenderness and abdominal rigidity simulating peritonitis, perforation or appendicitis. Graham Hodgson has shown by X-ray examination that the diaphragm rises in pneumonia, and in types I. and II. the consolidation begins near the hilum and spreads peripherally. In type III. the appearances are less characteristic, and may begin peripherally and spread centrally. Resolution reverses the order of appearance. It is rare for the spleen to be sufficiently enlarged to be palpable. The blood shows a leucocytosis up to

20,000, occasionally up to 50,000 in young or sthenic patients. Blood culture may yield pneumococci, although this was successful in only 30 per cent. of cases at the Rockefeller Institute. The urine is diminished in quantity, and there is a great reduction in the sodium chloride excretion until the crisis. Albumin and albumose are frequently found in small quantities in the urine during the febrile stage, and a few granular casts may be present. The uric acid excretion is increased to two or three times the normal, commencing the day before the crisis and generally falling to normal during the ensuing week. This is probably due to disintegration of the exudate in the alveoli, and so forms a measure of resolution, although some authorities maintain that it runs parallel with leucocytosis and not with cell destruction. Pneumococci can sometimes be obtained from the urine at the height of the disease. The blood pressure usually falls during the course of pneumonia, and according to G. A. Gibson a sudden rise indicates the imminence of some complication, such as delirium, whereas a sudden fall suggests the onset of cardio-vascular paralysis.

The disease does not always follow the typical clinical course, and certain varieties are described :

Apical pneumonia.—The consolidation may be limited to the apex or upper lobe of one lung. This is more common in children, the aged and alcoholics, and is often associated with marked cerebral symptoms.

Creeping pneumonia (Migratory or wandering pneumonia).—The consolidation spreads irregularly in one or both lungs. Partial resolution occurs, but there is no true crisis, and as successive portions of the lungs become involved the temperature exacerbates, eventually falling by lysis in cases that recover.

Central pneumonia.—The symptoms and appearance of the patient may suggest lobar pneumonia, and yet no abnormal signs can be detected in the lungs. In some of these cases there may be a deep-seated consolidation, which can usually be revealed by X-rays. A typical crisis may occur.

Massive pneumonia.—The bronchi, as well as the alveoli, may be filled with a fibrinous exudate. It is a rare condition and leads to difficulty in diagnosis, as the physical signs resemble those of pleurisy with effusion, vocal fremitus being diminished and breath-sounds weak or absent. The heart, however, is not displaced, or only slightly so.

Post-operative pneumonia.—It is probable that some cases that were formerly described as post-operative pneumonia were in reality instances of massive lobar collapse (see p. 1166). At times a pneumococcal pneumonia follows the administration of a general anæsthetic, but it does not present any peculiar features.

Traumatic pneumonia.—The fact that an injury to the chest may be followed after a short interval by a pneumonic process in the lungs has long been recognised. The condition was called "contusional pneumonia" by Litten in 1881. Külbs showed later that the changes in the lungs in dogs following local trauma were mainly hæmorrhagic, and that the lung opposite to the side injured may be affected by "contre-coup." In the recorded cases of traumatic pneumonia two types can be differentiated—(1) those with hæmorrhagic lesions only, and (2) those showing hæmorrhagic foci with a superimposed bacterial infection. The former recover rapidly, the latter often lead to a fatal issue.

Pneumonia in children.—This often presents certain characteristic features. There is rarely any sputum, the expectoration being swallowed. Convulsions at the onset are common. The lesion is often at the apex of the lung. Cerebral symptoms are frequent, and empyema or otitis media often occurs as a complication.

Pneumonia in the aged.—This occurs frequently as a terminal infection, often leading to a rapid and comparatively painless death. The onset may be insidious and the physical signs slight.

In *pneumonia in the insane*, lobar consolidation is often observed, without marked constitutional disturbance other than fever.

Secondary pneumonia.—Lobar pneumonia may develop during the course of certain acute specific fevers, notably enteric, typhus and plague. It is doubtful whether a true lobar pneumonia occurs in influenza, the condition to which the name *influenzal pneumonia* is applied being due to coalescing lobular pneumonia with hæmorrhagic extravasations.

Complications.—Delayed resolution not infrequently occurs, the signs of consolidation persisting for weeks instead of days. Frequent careful examinations should be made and possible errors in diagnosis considered, such as the presence of tuberculosis or empyema. Gangrene and abscess are rare but recognised complications.

Dry pleurisy is an invariable accompaniment when the consolidation reaches the surface, and in a considerable proportion of cases slight serous effusion occurs. This occasionally becomes frankly purulent and an empyema results. Bronchitis is common and may be due to a complicating secondary infection. Cardiac failure is a grave occurrence and can be recognised by increasing cyanosis, lividity and dyspnoea, with signs of enlargement of the right heart and with enfeeblement of the heart-sounds. Pericarditis is not very uncommon and is a serious complication. It may be dry or proceed to serous or purulent effusion. Acute endocarditis, sometimes of infective type, occurs. Abdominal complications are comparatively rare. They include pneumococcal peritonitis, colitis and nephritis. Acute dilatation of the stomach occurs in rare cases, and is usually rapidly fatal. Meteorism is more common and, although serious, is more amenable to treatment. Jaundice, due to catarrh of the bile-ducts, or to hæmolysis, is sometimes present.

Pneumococcal meningitis supervenes in rare cases, and is invariably fatal. Delirium has already been referred to, and is especially serious when occurring in alcoholics. *Peripheral neuritis* has been described, but is very uncommon. Otitis media and arthritis, proceeding sometimes to suppuration, occur as complications, both being commoner in children. A parotitis, sometimes going on to suppuration, is an occasional and serious complication, especially in old people. During convalescence, *thrombosis of the veins of the legs* may occur in rare instances.

Sequelæ of lobar pneumonia are uncommon. Perhaps the most remarkable is the liability to subsequent attacks possibly due to infection by different types of pneumococcus. Some permanent pleural thickening or adhesion may occur, and after an empyema the usual sequelæ may result. *Pulmonary fibrosis* (chronic interstitial pneumonia) is rare, especially in comparison with its frequency after broncho-pneumonia; this may lead to bronchiectasis.

Course.—The course depends on the type and virulence of the infection and on the resistance of the patient. In a typical *sthenic* case, consolidation

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is well established by the second or third day, defervescence by crisis occurs usually on the seventh day, signs of resolution become apparent a day later, and all signs clear up within 14 days of the onset. In asthenic cases the course is less typical and often prolonged to 9 or 10 days, defervescence occurring by lysis. In fatal cases, death commonly occurs between the fourth and tenth days, although severe cases may prove fatal as early as the first or second day. After the tenth day a fatal result is generally due to complications. An abortive course is described, in which typical symptoms occur with slight or indefinite signs, the temperature falling by crisis within 36 hours, followed by rapid recovery. This group includes the "*maladie de Woillez*." It is difficult in many instances to establish the true causation of such cases.

Diagnosis.—When the disease is well established and the history is available, diagnosis is as a rule easy. To prove the pneumococcal origin, sputum examination, lung puncture, or blood or urine culture is necessary. The investigation of serum reactions is necessary to establish the type of pneumococcus concerned if specific treatment is employed.

At the onset, especially before the signs of consolidation develop, difficulties in diagnosis often occur. The initial rigor or convulsion with vomiting may suggest scarlet fever. In children, especially those with early apical pneumonia, headache, vomiting, convulsions, head retraction, squint and even slight Kernig's sign may lead to an erroneous diagnosis of meningitis. Pain in the side and cough, the altered pulse respiration ratio, and the presence even of slight abnormal physical signs in the chest, usually suffice in both instances to suggest the correct explanation.

Occasionally the onset of pneumonia may simulate an acute abdominal condition, such as appendicitis or perforation of a gastric ulcer, owing to referred abdominal pain, sometimes with rigidity. The diagnosis may be very difficult, and laparotomy has not infrequently been carried out in error. The history, the pulse respiration ratio, the absence of tenderness on rectal examination, and the presence of pulmonary signs usually enable a correct decision to be made.

Influenza may start acutely and simulate pneumonia, but the distribution of the signs and the examination of the sputum generally serve to distinguish between them. Typhoid fever less often gives rise to difficulty, but some cases of pneumonia pass quickly into a typhoid state, while some cases of typhoid fever develop consolidation in the first week.

When consolidation is well established, the chief conditions to be differentiated are—(1) Broncho-pneumonia. The slower onset, the more prolonged course, the bilateral patchy physical signs, and the marked predominance of the bronchitic manifestations usually suffice to differentiate this group of conditions. (2) Secondary pneumonias, such as those in plague, typhoid fever, and influenza, can be diagnosed only from the history, the associated symptoms and signs, and from the bacteriological examinations. (3) Friedländer's pneumonia is rare. Its course is short, its prognosis grave, and it can only be recognised by bacteriological investigation. (4) Massive collapse. The diagnosis of this condition and its differentiation from pneumonia are discussed on p. 1167. (5) Acute pneumonic tuberculosis. The onset and early signs may be identical with those of pneumonia. The persistence of the fever, its tendency to become remittent or intermittent, and the

occurrence of night sweats should suggest looking for tubercle bacilli in the sputum. (6) Pleural effusion and empyema. Differentiation is generally easy, except in cases of massive pneumonia. Investigation of the position of the cardiac impulse, and of vocal fremitus and resonance, affords the most valuable aid. Grocco's triangle may also assist. In some cases the diagnosis can only be established by the exploring needle. (7) Infarction of the lung in cardiac disease, causing pain, cough, blood-stained expectoration, and dyspnoea, may simulate pneumonia. The absence of fever, the presence of the cardiac condition and the localised physical signs are generally characteristic. (8) Acute oedema of the lung, especially in mitral stenosis, may suggest pneumonia. Fever is generally absent, the sputum is typical, and the primary cause may be apparent. An attack of paroxysmal tachycardia may give rise to difficulty, when it leads to dullness and crepitations at the bases, but careful examination should establish the very rapid action of the heart and the evidence of venous engorgement in other parts.

Prognosis.—Lobar pneumonia is a serious disease, with a high mortality rate. This is profoundly influenced by age and by recent methods of treatment. It is but little fatal in childhood, except in the first years of life. After the age of 60, the mortality until the new chemo-therapeutic measures were employed was from 60–80 per cent. The New York investigations at the Rockefeller Institute demonstrated the importance of the type of pneumococcus in prognosis; thus, it was found that the mortality of cases with types I. and II. was about 25 to 30 per cent., of those with type III. 50 per cent., and of other types collectively only 12 per cent. With M. and B. 693 (Dagenan, sulphapyridine) the average mortality of all types has been reduced to about 8 per cent.

The previous habits and history of the patient influence prognosis considerably; chronic alcoholism doubles the risk of a fatal issue, and the outlook is grave in patients who are the subjects of diabetes, chronic cardio-vascular disease, nephritis, marked debility or obesity. Unfavourable indications during the course of the disease are profound toxæmia, a pulse-rate persistently 130 or more, a blood pressure in millimetres of mercury lower than the pulse-rate, and a temperature remaining at 105° F. or over for several days. Absence of the usual leucocytosis is generally of sinister import. Dilatation of the right heart, with cyanosis progressing to lividity, is most grave. Modern statistics confirm the traditional view that labial herpes is a favourable prognostic sign.

Of complications, meningitis is invariably fatal, unless it responds to treatment by sulphapyridine while septic endocarditis is extremely grave. Cases with abscess or gangrene, although serious, sometimes recover, especially if operative treatment is practicable. The prognosis of those with pericarditis is serious, but not uniformly unfavourable. Cases with bilateral empyemata show a high mortality. Late delirium is a very serious indication.

Treatment.—**PROPHYLACTIC.**—Prophylactic vaccination has been used with success by Lister in South Africa. He employed a triple vaccine, made from three types, and gave 6000 millions of each. Three injections were made at weekly intervals.

When a case has occurred, all contacts should have a throat examination, and if virulent pneumococci are found a suitable antiseptic mouth wash

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should be used. The room in which the patient has developed the disease should be disinfected afterwards. If possible, no case of pneumonia should be nursed in a general ward of a hospital, and the doctor and nurse in attendance should wear gauze masks. All sputum should be disinfected. The patient should lie in a narrow bed away from a wall to facilitate nursing. The room should be well ventilated, and the temperature maintained at 60° to 65° F. Treatment in the open air is not advisable except in very mild weather. Two important factors are rest and sleep. The patient should, therefore, be disturbed as little as possible by the examination of the physician and by the attentions of the nurse. He should not, however, be allowed to lie flat all the time, to avoid basal congestion. The diet should be restricted to fluids and semi-solids, eggs, milk, meat extracts and the various invalid foods being given up to 2 or 3 pints in the 24 hours. Dextrose, in the proportion of 2 to 4 ounces to the pint of lemonade or orangeade, is useful. Too much milk should be avoided, as it is liable to cause indigestion and flatulence. The irritating cough, which induces such intense pain, should be checked by a sedative linctus, or by lozenges, but it may be necessary to inject $\frac{1}{2}$ th grain diamorphine hydrochloride (heroin), or even $\frac{1}{4}$ th grain morphine to relieve pain and to induce sleep in the early stage. Local applications to the chest help to relieve pain. Hot linseed poultices to the back and side may be employed, but cataplasma kaolini or antiphlogistine applied on lint does not require such frequent changing and disturbs the patient less. Care should be taken to avoid too hot application, which may injure the skin. A pneumonia jacket is preferred by some, by others the ice poultice or ice-bag is found very soothing. A dose of calomel should be given at the onset, and the bowels should be opened daily, either by a laxative or by a small soap enema unless at any stage the patient is profoundly exhausted.

Sulphapyridine (Dagenan, M. & B. 693) has proved of great value in the treatment of lobar pneumonia due to all types of pneumococcus. It is put up in tablets containing 0.5 grm. for adults, and 0.125 grm. for infants and young children. The usual dosage for adults is 4 tablets repeated in 4 hours, followed by 2 tablets every 4 hours for 2½ days. Subsequently 1 tablet is given every 4 hours for 24 hours, then 1 tablet every 8 hours for 36 hours, making a total of 23 grms. in 5 days. In infants 0.125 to 0.25 grm. is given four-hourly. The dose for children is based on the body weight, but they require proportionately up to 50 per cent. bigger doses than adults, e.g. up to the age of 3 years 0.375 grm., and at 5 years 0.5 grm. is given four-hourly, until the temperature falls to normal, when a smaller dose may be given eight-hourly for two further days. Children tolerate the drug well. The tablets are best administered crushed and suspended in water, milk or fruit juices. The patient should not be given sulphur-containing substances such as eggs, onions, Epsom salts or Glauber salts during the course of the treatment. A sufficient quantity of fluid should be taken to result in the passage of at least 50 ounces of urine in the 24 hours. At whatever stage in the illness the treatment is instituted the temperature usually falls to normal by lysis in 24 to 36 hours when the drug is effective, although the actual process of resolution in the lung is not accelerated. In some cases vomiting prevents an adequate concentration of the drug in the blood and the treatment has to be abandoned, but frequently the difficulty can be overcome by varying the method of administration. The most important toxic effects

which may occur include nausea and vomiting probably central in origin, headache, cyanosis due to methæmoglobinæmia, and hæmaturia. The latter may occur if the urine is too concentrated and is thought to be due to the formation of crystals of acetylated sulphapyridine. If the drug is administered for longer than 5 days there is a possibility of further complications ensuing, such as drug fever and granulocytopenia. In the former the temperature may rise to 104° or 105° F. and morbilliform, scarlatiniform or urticarial rashes appear. A preparation of soluble sulphapyridine (Dagenan—sodium) may also be given intramuscularly or intravenously if the patient cannot tolerate the drug by mouth. It is put up in ampoules containing 1 grm. in 3 c.c. The intramuscular injections are given undiluted and are painful. For intravenous injection the contents of the ampoule are diluted to 10 c.c. with normal saline. The usual dose is 1 grm. every 4 hours. In the early stage, a simple saline diaphoretic mixture may give comfort by promoting the action of the skin and by rendering the sputum less viscid. For this purpose liq. ammon. acetat. min. 120, potass. citrat. grs. 20, syrup. aurantii min. 60 and water to the ounce, may be given every 4 to 6 hours. The use of depressant drugs, such as tartar emetic, aconite, or pilocarpine, although formerly recommended, is now generally discarded. Expectorants such as ammonium carbonate or iodide of potassium in doses of 3 to 5 grains are often recommended after the second day, but are of doubtful utility.

Cardiac embarrassment and failure are the conditions requiring the most active treatment in this disease. A careful watch should be kept upon the colour of the patient, the condition of the pulse and the size of the heart. Digitalis, in doses of 5 to 15 minims of the tincture, may be added to the mixture, or given with brandy. Nikethamide (coramine), either by the mouth or hypodermically, is often of value and has largely replaced the use of camphor, though the latter dissolved in sterile oil may be given in 3-grain doses twice daily. If signs of acute heart failure occur, strophanthin gr. $\frac{1}{200}$ may be given intravenously, or digitalin gr. $\frac{1}{100}$ with strychnine sulphate or hydrochloride gr. $\frac{1}{80}$ to $\frac{1}{40}$ hypodermically. The latter may be repeated in from 4 to 6 hours if necessary. Strychnine alone may be given in doses of gr. $\frac{1}{80}$ every 4 hours, and is often very useful. Other circulatory tonics which may be employed hypodermically are pituitary (posterior lobe) extract $\frac{1}{2}$ to 1 c.c., or adrenaline 5 to 10 minims of 1 in 1000 solution. Alcohol is often useful; it should not be given too early in the attack, but where there are indications of incipient cardiac weakness 4 to 6 ounces daily may be given, and this even to alcoholics.

Oxygen inhalations may be helpful in any case where there is distress or cyanosis. It should be warmed, and may be bubbled through alcohol. It may be administered continuously by means of a double nasal catheter with flow-meter and humidifier if available, or the B.L.B. mask by which with various adjustments an alveolar concentration of oxygen of more than 90 per cent. can be obtained. An oxygen tent is now rarely used except for infants and children. These methods may prove of the greatest value where there is marked anoxæmia. Venesection to the extent of 10 or 12 ounces is of some value if there is lividity from right-sided engorgement, especially in sthenic cases. As a rule it is best not to interfere with the temperature by antipyretic drugs and measures unless it remains over 104° F., when sponging, either tepid or cold, should be tried.

Sleeplessness is a frequent and distressing symptom and requires treatment. In the early stages 10 grs. of Dover's powder or an injection of morphine or diamorphine hydrochloride (heroin) are usually effective. In the later stages, morphine should only be given with care, and then in association with atropine gr. $\frac{1}{200}$ to $\frac{1}{100}$ and strychnine gr. $\frac{1}{80}$ to $\frac{1}{40}$. Paraldehyde min. 120 with syrup of orange in 2 ounces of water, is safe and often effective. Chloralamide grs. 20 to 30, with bromides may be tried. In cases with delirium an ice-cap should be applied to the head, and the patient sponged with tepid water. Morphine may be necessary, and in severe cases hyoscyne, gr. $\frac{1}{100}$, may be injected; but the latter is a dangerous drug and the patient's condition should be watched, and strychnine administered if necessary. Tympanites, when present, is distressing and exhausting, and should be treated by passing a rectal tube or by the administration of an enema of asafoetida or a turpentine wash-out. If these measures fail, carbachol (doryl) or acetylcholine may be employed cautiously.

Specific.—Specific antisera are now available for many of the types, and have been used with some success. Horse serums, highly concentrated and refined by Felton's method, are now available for types I., II., IV., V., VII. and VIII. Rabbit antipneumococcus serum is available for type III., and many others. It has the advantage of ease of preparation and of considerable concentration. If it is proposed to employ serum treatment it is necessary first to determine the type of pneumococcus concerned by serological tests. A rapid method involving the testing of the organisms in the fresh sputum against test serums has been introduced by Armstrong and simplified by Neufeld. The specific serum causes swelling of the capsules in the corresponding type. The patient's sensitiveness to the serum must then be determined by an intradermic injection of 0.02 c.c. of diluted serum (diluted $\frac{1}{10}$ with saline), and if he shows a reaction he must be desensitised by small injections to prevent anaphylaxis. The serum is then diluted with an equal volume of warm sterile saline and 10 to 15 c.c. injected intravenously at the rate of 1 c.c. a minute, followed by 90 c.c. more during the next quarter of an hour. This dose is repeated every 8 hours until improvement occurs. Felton's serum (2000 units in 1 c.c.) is administered intravenously undiluted, after being warmed to body temperature and after careful preliminary tests as to sensitiveness. The initial dose is 10,000 units, slowly injected intravenously. Amounts up to 40,000 or 50,000 units are given in the first 24 hours, though as much as 100,000 units may be necessary in severe cases. The earlier it is given, the greater is the likelihood of success. It is as a rule unnecessary in young children and adolescents, and contra-indicated in advanced age and in patients known to be allergic. Rabbit serums are given undiluted intravenously after being warmed to body temperature. At least 5 minutes should be taken to inject each dose. If anaphylactic manifestations occur adrenaline should be given subcutaneously at once.

Vaccine treatment is recommended by some, but the results are generally disappointing during the acute stage. A common method is to give 20 millions of a stock pneumococcus vaccine, and then to use an autogenous one as soon as it can be made. Sensitised and detoxicated vaccines have also been prepared. Vaccines seem to be more valuable in cases of delayed resolution.

Artificial pneumothorax has been suggested as a method of treatment.

There is as yet no convincing evidence of its value, but in cases with severe pain, the introduction of sufficient air to separate the inflamed surfaces of the parietal and visceral pleura is worth considering.

2. BRONCHO-PNEUMONIA

Synonyms.—Lobular Pneumonia; Catarrhal Pneumonia; Capillary Bronchitis.

Pulmonary consolidation of lobular distribution occurs in a variety of conditions which have little else in common. A satisfactory classification is at present difficult. The term capillary bronchitis is misleading and should be regarded as obsolete, since any inflammatory condition affecting the finer bronchi is invariably associated with alveolar changes. For convenience the following varieties of broncho-pneumonia may be described: (1) Primary. (2) Secondary. (3) Aspiration or deglutition. (4) Tuberculous.

a. PRIMARY BRONCHO-PNEUMONIA

Ætiology.—This form almost invariably affects infants under 2 years of age, in whom a lobular pneumonia seems sometimes to occur under conditions which would induce lobar pneumonia in older children or adults. It occurs equally in the two sexes, and is commoner in the winter and the spring. Rickets, malnutrition and debility are predisposing conditions, but it sometimes develops in healthy robust infants after exposure or chill. The pneumococcus is the organism usually found, either alone or in association with others, such as streptococci, staphylococci, the *Micrococcus catarrhalis* or Friedländer's pneumo-bacillus.

Pathology.—Widely scattered patches of consolidation are found in one or both lungs. These may be small and separated by areas of collapse or emphysema. Occasionally they are almost confluent, and at first sight appear like lobar pneumonia, constituting the pseudo-lobar form; but careful observation shows that the distribution is lobular and that zones of incomplete consolidation or of normal lung tissue separate the solid areas. If the process reaches the surface some degree of pleurisy is present, although this is less than in lobar pneumonia.

Microscopically, the appearances approximate to those of the lobar form; the alveoli are found to be filled with exudate, in which leucocytes and desquamated epithelial cells are present, together with some fibrin and red blood corpuscles. Catarrhal changes are also present in the bronchi.

Symptoms.—The onset is acute, with vomiting and chill, or convulsion, as in lobar pneumonia, but may be more gradual. Cough, cyanosis and dyspnoea develop rapidly. There is no expectoration, since infants and young children swallow the sputum. Cerebral symptoms simulating meningitis are common. The temperature rises quickly to 103°, 104° F., or higher, and the range is of the same character as in lobar pneumonia. Deferrescence by lysis is the rule.

The physical signs are variable. In cases with widespread consolidation they are very similar to those of lobar pneumonia, with dullness, tubular breathing, increased voice-sounds and crepitations. In other cases, although

the aspect of the infant appears characteristic of pneumonia, with rapid breathing, cyanosis, reversed rhythm of inspiration and expiration, sucking in of the lower ribs and dilation of the *alæ nasi*, the signs are more scattered. Tubular breathing and increased voice-sounds may only be heard in localised patches, especially in the lower lobes. Crepitations are commonly present, and rhonchi may be audible over both lungs.

Complications and Sequelæ.—These are similar to those of lobar pneumonia.

Course.—This is usually short, the temperature falling in from 3 to 7 days, but it may be more prolonged and be suggestive of tuberculosis, or some other form of secondary bronchitis.

Diagnosis.—Primary broncho-pneumonia has to be distinguished from the lobar form to which ætiologically and pathologically it is so closely related. The acute onset without previous respiratory symptoms will suggest its primary character, while the patchy distribution of the signs generally suffices to establish its lobular distribution. In pseudo-lobar forms, this differentiation may be almost impossible during life. The cerebral symptoms at the onset, and the early absence of pulmonary signs may give rise to difficulty, as in the first stage of lobar pneumonia.

Prognosis.—The prognosis of primary broncho-pneumonia is generally unfavourable, especially in very young or debilitated infants.

Treatment.—This is practically identical with that of secondary broncho-pneumonia in children.

b. SECONDARY BRONCHO-PNEUMONIA

In this condition there is inflammation of the bronchi, spreading down to and involving the alveoli. It is generally a catarrhal process, but may go on to septic or suppurative manifestations.

Ætiology.—A secondary broncho-pneumonia may occur at any age, but is much more common in early and advanced life. It is equal in its incidence in the two sexes. It frequently occurs as a complication of measles, whooping-cough, psittacosis and influenza, less commonly in cases of diphtheria, scarlet fever, plague and the enteric group. A bronchitis starting in the larger tubes may spread downwards to the alveoli. Broncho-pneumonia may develop during the course of acute gastro-enteritis. A secondary broncho-pneumonia occurs as a terminal infection in many old and debilitated persons and in those with chronic wasting or cachectic diseases, and also in chronic cardio-vascular conditions, chronic renal disease and in many progressive nerve degenerations. Any septic process may produce a metastatic broncho-pneumonia. This occurs in association with otitis media, suppurative processes about the appendix or Fallopian tubes, and cerebral abscess.

Bacteriology.—This is, as might be expected, very varied. Streptococci are frequently present, especially the hæmolytic variety, generally associated with other organisms, such as the pneumococcus, Pfeiffer's *H. influenza*, staphylococci and those found in catarrhal conditions of the upper air-passages. The *B. pertussis* may be found in cases associated with whooping-cough, the *B. pestis* in plague, and occasionally the *B. diphtheriæ* in diphtheritic broncho-pneumonia. The importance of Friedländer's *B. pneumonia* was formerly overestimated in this connection.

Pathology.—When, from any of the above-mentioned causes, an inflammatory process reaches the finer bronchi, the alveoli become affected in three different ways. Owing to the blocking of the bronchi by secretion or exudate, small areas of collapse of lobular distribution are produced. The inflammatory process extends into some or all of these, and areas of lobular consolidation result. Not infrequently the adjacent groups of alveoli become distended and are thus in a condition of acute emphysema. The lungs are normal in size or slightly enlarged. The surface presents a somewhat uneven, mottled appearance. There are small projecting patches of firmer consistence and reddish-grey colour, due to the consolidated lobules. Adjacent areas may be depressed and slaty blue, from lobular collapse, while the intervening lung tissue is normal or pinkish and emphysematous. There may be dimness or slight roughening of the pleura where the consolidated areas reach the surface, but serous or purulent effusion is uncommon. On section, the lung is found to be congested and sometimes œdematous, especially at the bases, while the bronchi exude pus or muco-pus from their cut ends. The reddish-grey areas of consolidation are found to vary in size from a pin's head to a hazel nut. They are generally more abundant in the lower lobes, especially posteriorly. The consolidated and collapsed areas both sink in water, and do not crepitate. There is often some peribronchitis, and the bronchial glands are usually enlarged. Microscopically, the finer bronchi and the consolidated alveoli are found to be filled with an exudate containing large numbers of leucocytes and desquamated, proliferating epithelial cells, but in which few red blood corpuscles and little or no fibrin are found.

In the very acute condition to which the name capillary bronchitis was formerly applied, consolidation may not be apparent, but microscopical examination invariably demonstrates the involvement of the alveoli. In influenzal broncho-pneumonia the pathological changes probably commence as an exudative bronchiolitis, associated with capillary hæmorrhages. Secondary infections are probably responsible for the consecutive broncho-pneumonic process, which results in flooding of the alveoli with an exudate containing red cells, but little or no fibrin.

Symptoms.—In the cases ensuing on bronchitis in infants or old people (formerly called capillary bronchitis), initial symptoms may be slight, and simply those of ordinary bronchitis, namely, malaise, slight fever and cough, with or without expectoration. The implication of the finer tubes and alveoli is usually marked by a rapid rise of temperature, great prostration, quick breathing and an irritating, persistent and often ineffective cough. In children, the *alæ nasi* work, the lower ribs are sucked in, and the pneumonic type of breathing develops. The patient becomes cyanosed, the pulse is rapid, 120 or more, and the respirations 50 or 60 per minute. The physical signs are in general indistinguishable from those of primary broncho-pneumonia, but breath sounds are often harsh and puerile, while tubular breathing is not heard, or only in very localised areas. In old people, cyanosis, restlessness and delirium may occur, and later the cough become less frequent, the patient being drowsy and tending to sink down in the bed, whereas previously there was orthopnoea. These symptoms are ominous and indicate failure of the respiratory centre.

The physical signs are often those of bronchitis, harsh or weak inspira-

tion and prolonged expiration, sibilant and sonorous rhonchi and crepitations or crepitant râles especially at the bases. Patches of tubular breathing with increased voice sounds may develop but are not always present.

In other forms of secondary broncho-pneumonia similar symptoms and signs develop more insidiously in the course of the primary disease. Broncho-pneumonia should be suspected when cough, expectoration and dyspnoea, together with a remittent type of temperature, develop in the course of an acute specific fever or other severe illness. In all forms, anorexia is common, the mouth and tongue become dry, and thirst is complained of. The urine presents the usual high-coloured, concentrated character of febrile conditions. It is often diminished in quantity, may contain a small quantity of albumin, and not infrequently deposits urates.

Complications and Sequelæ.—These are relatively infrequent. Pleurisy may proceed to effusion, and when this occurs it is often purulent. Abscess and gangrene are rare, but develop rather more frequently than after lobar pneumonia. Other complications, such as pericarditis, endocarditis, meningitis and nephritis, are probably due to blood-borne metastasis.

The most important sequel is pulmonary fibrosis, which is often the origin of bronchiectasis later in life. Pulmonary tuberculosis is frequently described as a sequel, especially after measles, and may be due to inflammatory changes in the bronchial glands activating a quiescent tuberculous deposit there. In many cases of tuberculosis described as following on broncho-pneumonia, it is more probable that the original lung affection was tuberculous.

Course.—Secondary broncho-pneumonia generally has a longer course than either the primary form or the lobar variety of pneumonia. The fever often persists in remittent type for two or three weeks, and sometimes even for two or three months, although in this case tuberculosis should be suspected. The decline is almost always by lysis. Convalescence is often slow, the patient being left thin, weak, anæmic and debilitated.

Diagnosis.—The development of pulmonary symptoms, and of more or less characteristic physical signs in the course of measles, whooping-cough or one of the other diseases mentioned above, usually renders the diagnosis easy. Difficulty may arise in regard to tuberculosis, which in one form produces lobular pneumonic lesions with symptoms and signs indistinguishable from other varieties of secondary broncho-pneumonia. In any case where the fever lasts more than three weeks, or where the signs show no tendency to resolve or are chiefly apical, tuberculosis should be suspected. Unfortunately in children sputum is rarely available. An attempt is sometimes made to obtain it on gauze held in forceps, after exciting cough by touching the fauces. The mucus in the fauces may also be examined for tubercle bacilli. The diagnosis may, however, remain doubtful, until signs of softening become established.

Bronchitis rarely gives rise to difficulty. The fever is usually less high, and of shorter duration, while the physical signs are different, signs of consolidation being entirely absent. Hypostatic pneumonia may have to be considered. There is usually some obvious cause for this, such as cardiac disease and failure, or prolonged confinement to bed. The temperature is generally lower and the distribution is lobar.

Pleural effusion and empyema can generally be differentiated by the alteration of vocal fremitus and the displacement of the cardiac impulse.

In case of difficulty the exploring syringe enables a distinction to be made.

Prognosis.—The prognosis in secondary pneumonia is serious. Many deaths occur from this complication in the acute specific fevers, particularly with measles and influenza. Even the form following on severe bronchitis is frequently fatal, especially in old people and in wrongly fed or debilitated infants. The development of delirium, of a pulse-rate over 150, of marked cyanosis and dyspnoea is unfavourable. In old people, drowsiness, sinking down in the bed, and cessation of cough are very grave indications.

Treatment.—The treatment is very similar to that of lobar pneumonia, except that stimulant and expectorant drugs may be necessary from the first. At the present time there is practically no difference in the methods of treatment applicable to the primary and secondary forms. In cases due to pneumococcal infection sulphapyridine should be employed (see p. 1234). If streptococci are established as the infecting agent, one of the sulphanilimide preparations should be given at once.

The patient must be in bed, and the position should often be changed so as to prevent hypostatic congestion. The room should be well ventilated, but without draughts, and the temperature kept at 65° F. both night and day. In the early stages the air may be moistened by a steam kettle, but the use of a tent is generally to be avoided. Poultices are now less generally employed than formerly, especially for children, and a light pneumonia jacket of Gamgee tissue is usually preferred. The diet should be restricted to fluids and semi-solids, as in pneumonia. Stimulants may be given early if the pulse becomes weak, in doses of 10 drops of brandy every 2 hours to infants, and quantities up to 4 or 6 ounces in the 24 hours to old people. The dry, distressing cough at the onset may be loosened by giving a simple alkaline febrifuge mixture, such as liq. ammon. acetat. min. 120, pot. citrat. grs. 10, sod. bicarb. grs. 10, with flavouring agents, such as syrup of tolu and chloroform water. Later, ammon. carb. and tinct. ipecac. may be given, but large doses of expectorants are to be avoided because of their irritant effect on the stomach. Opiates should not be administered except as tinct. opii camphorata or possibly Dover's powder in the early stages. In infants they should not be given at all.

When in infants or children, the bronchi are becoming blocked by the secretion within them, as evidenced by increasing dyspnoea, an emetic should be given. For this purpose tinct. ipecac. or ammon. carb. in emetic doses is the most effective. In old people, ammon. carb. may be given in milk in doses of grs. 10 two or three times a day, and energetic counter-irritation applied to the bases by means of turpentine stupes, dry cupping or strong liniments.

Strychnine either by the mouth or hypodermically is strongly recommended in cases in which the respiratory centre shows signs of failure. It may be pushed, if necessary, to the point of producing slight muscular twitchings. Nikethamide (coramine) or camphor injections and cardiac tonics may be given under the same conditions as in lobar pneumonia. The administration of warmed oxygen may give relief to dyspnoea and distress.

In cases in which resolution is delayed the question of vaccine therapy may be considered. It seems sometimes to be of distinct value.

c. INHALATION, ASPIRATION AND DEGLUTITION BRONCHO-PNEUMONIA

Acute broncho-pneumonic processes may be caused by the inhalation or aspiration of fluid or solid particles, derived from the upper air-passages or from other parts of the lung. To this form the name of aspiration, or inhalation pneumonia is applied. When from any cause food particles are drawn into the bronchi and broncho-pneumonia results, the condition is referred to as deglutition pneumonia. The resultant processes are similar, and are in effect analogous to those caused by other septic or infected foreign bodies inhaled into the bronchi.

Ætiology.—These conditions may occur at any age, but are more common in adult life. They result from septic processes in the mouth, naso-pharynx, larynx or trachea, and from any morbid state leading to anæsthesia of the pharynx, or to difficulty in deglutition. They occur in association with ulcerating growths of the mouth, tongue, tonsil, pharynx or larynx, and after operations for these conditions or upon the nose and throat, including tracheotomy. Aspiration broncho-pneumonia may also result from vomiting during or after the administration of an anæsthetic. Carcinoma of the œsophagus eroding the trachea may be a cause. Diphtheritic or other forms of paralysis, coma from any cause, especially cerebral vascular lesions and uræmia, may lead to the passage of food particles into the air-passages. Other cerebral lesions, such as abscess or tumour and bulbar paralysis, can also produce the same condition. Infected material may be aspirated from diseased to healthy parts of the lung, as in hæmoptysis, abscess, gangrene and bronchiectasis, or after rupture of an empyema into a bronchus.

Pathology.—Any material reaching the air-passages in this manner is certain to be laden with infective micro-organisms, which may induce bronchitis and broncho-pneumonia. Since pyogenic organisms are often present, suppuration is frequent and single or multiple abscesses result, or even gangrene. If the pleura becomes involved, empyema may develop.

Symptoms.—These are in general similar to those of secondary broncho-pneumonia and are superadded to those of the primary condition. There is generally high temperature, sometimes with rigors, cough and expectoration which is occasionally offensive. It may be mixed with food material and with blood. The physical signs are those of bronchitis and widespread broncho-pneumonia.

Complications and Sequelæ.—These are somewhat similar to those of other inhaled foreign bodies, and comprise abscess, gangrene and empyema.

Course.—The course is generally short, owing to the severity of the process and the gravity of the primary cause. In the comparatively rare cases that recover the course may be severe and protracted.

Prognosis.—From the nature of the primary condition and the intensity of the resulting broncho-pneumonia, this is usually grave.

Treatment.—**PROPHYLACTIC.**—The utmost care should be paid to the toilet of the mouth and pharynx in disease of, or operations upon, these parts. In paralysed or unconscious patients it may be necessary to resort to nasal feeding. In hæmoptysis or bronchiectasis the patient should lie rather on the affected side.

The treatment of the developed condition can be only palliative or

symptomatic in many cases. In most instances the general treatment is similar to that of secondary broncho-pneumonia.

d. TUBERCULOUS BRONCHO-PNEUMONIA

This constitutes one form of pulmonary tuberculosis (see Acute Caseous Tuberculosis, p. 1191).

PNEUMONITIS

Definition.—A localised or disseminated inflammatory process involving the whole texture of the lung and bronchial structures in the areas affected.

Ætiology.—Any severe septic infection of the bronchi or lungs may proceed to pneumonitis. The commonest infecting agents are streptococci, especially the hæmolytic and anærobic varieties. The fusi-spirochætal organisms may also cause it. Pneumococci alone seldom lead to pneumonitis, though they may be found in association with other organisms. Pneumonitis is chiefly met with in later adult life, and more in the male sex. It may be produced by the aspiration of foreign bodies. It sometimes occurs in association with bronchiectasis and may be one of the conditions associated with the febrile attacks occurring in that condition.

Pathology.—The affected area is deeply congested, solid and airless. The bronchi may exude pus. Softening is frequent, leading to the formation of one or more abscesses. Empyema may occur as a sequel or complication.

Symptoms.—The onset is usually acute and the general clinical features are identical with those of severe broncho-pneumonia. There is usually troublesome cough, with more or less copious mucopurulent expectoration. Dyspnoea and cyanosis may be marked. There is marked prostration and the patient is almost always gravely ill. The physical signs are usually those of localised pneumonia or broncho-pneumonia. The blood picture is similar to that of abscess of the lung—a leucocytosis with polymorphonuclear preponderance.

Diagnosis is generally established by X-ray examination, showing diffuse dense areas of consolidation, often progressing to abscess formation. The areas may be multiple and the process may spread widely.

Prognosis.—The prognosis is in the main serious, and depends to some extent upon the cause. Many cases, however, recover under treatment either with or without abscess formation.

Treatment is in the first instance that of broncho-pneumonia. When abscess formation occurs, postural drainage, adapted to the situation of the abscess or abscesses, should at once be adopted.

A *simple pneumonitis* has been described in children by Gill. The symptoms are cough, anorexia and loss of weight. On physical examination one or more areas of impaired percussion note with a few râles are found. Radiological examination reveals opacities in the corresponding situations. These symptoms and signs usually clear up in a few days without treatment.

R. A. YOUNG.

G. E. BEAUMONT.

DISEASES OF THE PLEURA

PLEURISY

Pleurisy or pleuritis is an inflammation of the pleural membrane covering the lung, or of its parietal reflexions.

An ætiological classification, based on the bacteriological findings, would be the most satisfactory one, but is at present impracticable, chiefly owing to the difficulty of establishing the bacteria concerned in many cases. The classification usually adopted depends upon the effects produced. If the process leads only to fibrinous deposit it is described as *dry pleurisy*. If, in addition, much serous fluid is poured out, the condition of *pleurisy with effusion* results, while if pus-formation occurs, the affection is described as *purulent pleurisy* or *empyema*.

It is, however, important to recognise that, although such a classification is convenient from a clinical standpoint, the three conditions are in reality only stages or degrees in the pleural response to irritative or noxious agents. The form occurring in any given case depends upon the nature of the cause, the extent of the infection and the degree of resistance possessed by the individual affected. Further, pleural inflammations may be primary or secondary to local disease or to blood infection, and they may be acute or chronic in course.

A.—ACUTE DRY PLEURISY (ACUTE FIBRINOUS OR PLASTIC PLEURISY)

Ætiology.—This affection may be primary or secondary, the latter being much more common. Even in many cases of so-called idiopathic or primary pleurisy, the condition is in reality secondary to latent or unrecognised disease of the lung or adjacent structures.

Primary dry pleurisy.—Predisposing causes include occupation and climate. Exposure to sudden changes of weather or cold winds, and the necessity of remaining in wet or damp clothing, favour its onset. It is commoner in men, particularly in those of poor physique. It may occur at any age, but is most frequently seen between the ages of 20 and 40 years. Chill seems to be common as a determining cause. It is now certain that the great majority of cases are due to the tubercle bacillus, and that chill or injury is simply concerned in lowering resistance and thus promoting activity of the bacillus. It is possible that some cases may be due to acute rheumatism.

Secondary dry pleurisy.—Dry pleurisy is a frequent complication or concomitant of many diseases of the lungs, notably of pulmonary tuberculosis in any form. It is almost invariably present in lobar pneumonia. It occurs in association with pulmonary collapse, interstitial pulmonary fibrosis, bronchiectasis, abscess, gangrene, infarcts and new growths of the lung. Injuries of the chest-wall, disease of the ribs, chronic nephritis, septicæmia or pyæmia may all be complicated by acute dry pleurisy.

Pathology.—The inflamed area is often localised, but the process may be

widespread or even involve the whole pleural surface. Either the visceral or parietal layer may be first affected, but as a rule both become involved. There is at first hyperæmia with exudation of serum into the subpleural connective tissue. The pleura then appears slightly dull or matt, instead of shiny. Further exudation leads to the deposit of fibrin on the roughened pleural surfaces in the form of a thin false membrane, which often presents a rough or even shaggy appearance. This membrane consists of fibrin entangling leucocytes, a few red blood corpuscles and desquamated epithelial cells.

During the process of resolution, localised adhesions commonly form, but this is not invariable, and a patch of thickening without adhesion may be the ultimate result.

Symptoms.—The onset is usually sudden with acute pain in the side, often described by the patient as a "stitch." Occasionally a sense of malaise may precede the development of the pain by a few hours or even days, but this is not the rule. The pain is aggravated by deep inspiration, by coughing or even by movement. Cough is generally an early symptom, and it is characteristically short, dry, ineffective and distressing. The temperature is usually raised, but, as a rule, only to 100° or 101° F., and some cases are practically apyrexial. In secondary pleurisy these symptoms are added to those of the primary condition.

The decubitus is variable. The patient may lie on the affected side, but in some cases this aggravates the pain, and it is more comfortable to lie on the back or slightly turned towards the sound side. There is diminished movement on the affected side, and breathing may be rapid, although not dyspnoëic. On palpation, vocal fremitus is unaffected, but local tenderness is sometimes elicited, and occasionally a friction fremitus may be felt. The breath-sounds are generally unaltered, but they may be short or jerky in the neighbourhood of the lesion. The characteristic sign of dry pleurisy is the friction rub. This is typically a creaking, rubbing or leathery sound heard towards the end of inspiration and sometimes at the beginning of expiration. In the early stages there may be fine crepitant friction sounds only at the end of inspiration. These are very similar in character to intrapulmonary crepitations and can only be distinguished by their association with local pain, and by being unaltered by cough. Pleural friction sounds may be localised to a small area, and may not be present with every respiration. They may sometimes be brought out again after disappearance by moving the arm, or by taking a deep breath. The voice-sounds are not altered.

Complications and Sequelæ.—Dry pleurisy may proceed to effusion, or may lead to pleural adhesion, and this in turn may result in interstitial pulmonary fibrosis. The most common sequel is pulmonary tuberculosis, sometimes after an interval of years, the explanation being that the original pleurisy is frequently tuberculous. Aching pain in the side with some dyspnoëa may be a temporary sequel of dry pleurisy.

Course.—The temperature usually subsides in 2 or 3 days, the pain in the side and cough disappear, and convalescence is rapid, unless effusion occurs.

Diagnosis.—The differentiation of dry pleurisy from the other causes of pain produced in, or referred to, the chest-wall is not always easy and

requires careful observation of the case. The distinction is important, since an erroneous diagnosis of pleurisy may arouse a suspicion of tuberculosis in subsequent febrile diseases. In the conditions comprised in the term pleurodynia, which include fibrositis of the intercostal muscles and membranes, the pain is increased by deep inspiration, by other muscular movements, and by local pressure, but there is no rise of temperature and pleural friction is not present. In intercostal neuralgia, the pain follows the course of the nerve and is often periodic in character. It may be influenced by movement, but is less affected by respiration than that of pleurisy. There may be tenderness and hyperalgesia over the points of exit of the posterior primary, lateral or anterior cutaneous branches of the nerve affected. Similar manifestations may occur at the onset of acute posterior ganglionitis or herpes zoster. Other conditions inducing pain referred to the chest-wall are tumours or aneurysm pressing on the intercostal nerves, malignant disease of the spinal cord or of its membranes, and caries of the vertebræ. Where the pain lasts more than a few days, and no friction is heard, these conditions should be borne in mind.

Occasionally adventitious sounds of extra-pleural origin may give rise to some difficulty. Contraction of the muscles of the chest may cause a muscular "susurrus"; grating sounds may be produced in the shoulder-joint or in the fascial planes of the back muscles. The origin of these sounds can usually be determined by causing the patient to cease breathing while carrying out movements of the shoulder or back muscles. Occasionally true friction sounds may have a cardiac rhythm as well as a respiratory one, when the area of pleura involved is near the pericardium. It is then referred to as pleuro-pericardial friction.

Having established the evidence of dry pleurisy, a careful search should be made for some primary condition before regarding the case as one of simple primary dry pleurisy. Pulmonary tuberculosis, pneumonia, bronchiectasis and the other causes mentioned above should be considered and excluded.

Prognosis.—The immediate prognosis is good, but as has been mentioned already, the condition may be of tuberculous origin, and eventually be followed by active disease of the lung.

Treatment.—The patient should be kept in bed, no matter how mild the attack. The diet should be fluid or semi-fluid, especially if more than a moderate degree of fever occurs. The pain can often be relieved by strapping the affected side. Strips of adhesive plaster are applied from the sternum to the vertebræ, beginning from below and working upwards. Occasionally this fails to afford relief and may even induce dyspnoea. As alternatives, a local application of tincture or ointment of iodine, a mustard leaf, capsicum ointment or small flying blisters may be employed. Leeches may also give relief in severe cases. Sometimes the pain is so intense that a small injection of heroin or morphine is necessary. A small artificial pneumothorax has been suggested as a means of separating the inflamed surfaces and giving relief to the pain in severe cases. A dose of Dover's powder is useful in the early stage to ensure a night's rest. The irritative cough is often relieved by strapping, and a sedative linctus or lozenge may be a comfort to the patient. An aperient is usually advisable. As a rule no other drugs are necessary, but in cases suspected to be due to rheumatism, salicylates and alkalis should

be administered. Convalescence is usually rapid, but the patient should not be allowed to resume work until fully restored to health, and if a tuberculous origin is suspected prolonged treatment on sanatorium lines should be advised.

Certain localisations of dry pleurisy require separate notice. These are the diaphragmatic and interlobar forms.

DIAPHRAGMATIC ACUTE DRY PLEURISY

Ætiology.—This affection may occur primarily under conditions similar to those causing dry pleurisy in other parts; not infrequently it is secondary to pathological changes in the abdomen. Thus hepatic cirrhosis, perihepatitis, perisplenitis, perinephric suppuration or peritonitis may lead to a spread of infection through the diaphragm to the adjacent pleura. It may also occur as a localised variety of secondary dry pleurisy, when the primary lesion is situated near the base of the lungs.

Symptoms.—Pain is usually very severe and may be referred to the shoulder or to the abdomen. The former is caused by nociceptive impulses ascending the phrenic nerve to its origin in the third to the fifth cervical segments of the spinal cord, leading to pain and hyperæsthesia referred to the cutaneous area of distribution of the fourth cervical root, at the summit of the shoulder. The abdominal pain is in the epigastric and hypochondriac regions, and in addition there is a localised tender spot, known as the “bouton diaphragmatique” of Guéneau de Mussy. This is situated in the subcostal plane, about 2 inches from the mid-line. The diaphragm is nearly motionless on the affected side, and there is often some rigidity of the corresponding upper abdominal muscles. Hiccough may be a noticeable and troublesome symptom. The diaphragm, being nearly fixed in the inspiratory position, may cause a slight downward displacement of the liver if the pleurisy is on the right side. A pleural friction rub is rarely heard, the only abnormal signs commonly present being diminution of air entry, and possibly slight dullness over the corresponding lower lobe of the lung.

Diagnosis.—This is often difficult, owing to the fact that the severity of the symptoms and their localisation frequently suggest the occurrence of some acute abdominal catastrophe such as perforation of a hollow viscus. The abdomen should be most carefully examined in every case. The history, the collapsed state of the patient and the evidence of free gas in the peritoneal cavity in perforation may assist in distinguishing between these conditions.

Treatment.—This is similar to that of simple dry pleurisy elsewhere, save that morphine should be withheld until the diagnosis is conclusively established.

INTERLOBAR DRY PLEURISY

Just as inflammation may be limited to the diaphragmatic portion of the pleura, so the membrane in the cleft between two lobes of the lung may be alone affected. This does not give rise to definite symptoms and signs by which it can be diagnosed during life, though its effects are not infrequently seen in X-ray films. It is frequently discovered on autopsy, but is generally secondary to pulmonary tuberculosis or pneumonia, and there is usually

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evidence of pleurisy elsewhere. It only assumes clinical importance when followed by effusion, and this condition is considered later.

B.—CHRONIC DRY PLEURISY

Under this heading a variety of conditions are included. Strictly it should be restricted to those rare cases, probably usually tuberculous in origin, in which the signs of dry pleurisy persist for long periods, or recur at frequent intervals. In such cases coarse dry friction may be heard over large areas of one lung, often with little or no accompanying pain.

Pleural adhesion and thickening are usually included in the group of chronic dry pleurisies. There may be no symptoms, or at most slight dyspnoea on exertion, with aching or pain on straining, or on lifting weights. Signs suggesting adhesion are local flattening and limitation of movement of the chest-wall. Litten's sign is also absent or diminished when the adhesion is basic, that is, the shadow cast by the movement of the diaphragm, best seen in the region of the seventh and eighth ribs in the anterior and mid-axillary lines, is not present or is much restricted. The vocal fremitus may be diminished and the percussion note impaired. The breath-sounds are often slightly weaker, and the voice-sounds may be diminished over the area where the thickening or adhesion exists.

Chronic diaphragmatic pleurisy or adhesion may give rise to a group of symptoms simulating chronic gastric ulcer. There is pain in the hypochondrium extending through to the back and aggravated by food. Radiographic examination may be of value in demonstrating limitation of movement of one cupola of the diaphragm, together with an angularity due to alteration of its normal contour. Investigation of the gastric functions may also prove of value in diagnosis.

The treatment of chronic dry pleurisy is mainly symptomatic.

C.—PLEURISY WITH EFFUSION

Many cases of pleurisy, possibly the majority, proceed to effusion. The effusion is usually serous in character, but may be hæmorrhagic. Inflammatory effusions must be distinguished from passive transudates, which will be considered separately under the heading of hydrothorax.

SERO-FIBRINOUS PLEURISY

Ætiology.—This is in the main identical with that of dry pleurisy, of which it is, in effect, a later stage. It has now been established that the majority of cases of sero-fibrinous pleurisy are due to the tubercle bacillus. The evidence on which this conclusion has been arrived at is—(1) the subsequent history of the cases shows that a considerable proportion develop active lung signs within 5 years; (2) the cytological and bacteriological examination of the exudate; (3) post-mortem examination of fatal cases; (4) the results of tuberculin reactions.

Other conditions which may give rise to serous effusions are lobar and lobular pneumonia, pulmonary infarcts and new growth. It may also occur

in the course of generalised infections such as the enteric group, acute rheumatism, and septicæmia due to streptococci or staphylococci. In most of these conditions the exudate often becomes purulent. Inflammatory serous effusion may also occur as a complication of severe anæmias, leukæmia, chronic nephritis, injury to the chest-wall and inflammatory conditions below the diaphragm or in the pericardium. It is also a common feature of polyorrhœnitis.

Pathology.—The affection commences with dry pleurisy, spreading over the visceral and parietal pleura, the fibrinous exudate soon forming a thick rough layer on the surface. Further exudation of fluid occurs and accumulates in the pleural cavity, the lung collapsing *pari passu* to accommodate it. Owing to the hilar attachment of the lung, it retracts upwards and inwards, allowing the fluid to accumulate at the bases and in the axillary region, where it reaches its highest level, unless previously existing adhesions prevent it. The lung retracts in this way owing to its elasticity, until the pleural negative pressure is completely abolished. In like manner the mediastinal contents, including the heart, are displaced away from the affected side. If fluid continues to be effused after the lung has retracted to the full extent, and after the negative pressure has become abolished, a positive pressure is produced. The lung is now compressed, and the diaphragm with the liver and spleen are pushed down, while the mediastinal structures are now displaced further towards the sound side. In long-standing cases, the lung may undergo the change known as carnification, as the result of the compression apneumatoxis. The lung appears dark red or slaty grey in colour, is firm, airless and heavier than water. If old adhesions are present, the effused fluid may be loculated and the collapse of the lung may be only partial.

If there is much positive pressure collateral hyperæmia of the sound lung may result and progress to œdema. The fluid in the pleural cavity is pale and clear; it often coagulates after withdrawal. Its characters are further described on page 1252. The quantity may amount to as much as 5 or 6 pints.

Symptoms.—The onset is usually similar to that of dry pleurisy, but the constitutional symptoms are often more marked. There may be an initial rigor, but as a rule pain and dry cough are the earliest symptoms. The fever is of moderate degree, although it may reach 103° F. or more. When effusion develops the pain is often relieved owing to the separation of the inflamed pleural surfaces. If a large quantity of fluid is poured out rapidly, distress of another kind becomes apparent, namely dyspnoea caused by the mechanical effects of the fluid, collapsing the lung and dislocating the mediastinum. In more slowly developing effusions there may be little or no dyspnoea, except on exertion. Expectoration is not common, unless there is co-existent pulmonary disease, or unless œdema of the sound lung develops.

The patient often lies on the affected side or may be propped up in bed. Cyanosis is not a marked feature even in large effusions, unless there is collateral hyperæmia of the sound side. There is generally some prominence on the side of the effusion, but the intercostal spaces are rarely bulged. Movement is restricted or absent in the lower part of the chest on the affected side, although with a moderate effusion the apical region may still expand. The cardiac pulsations may be seen in an abnormal position, the impulse being displaced away from the side of the fluid. In left-sided effusions, the

pulsation may be most marked in the fourth space on the right side as far out as the nipple line. On palpation, the position of the impulse should be verified, and then the amount of chest movement and the character of the vocal fremitus determined. The latter is diminished or completely absent over an effusion of moderate or large size, although it may be obtained over the area where the collapsed or relaxed lung is in contact with the chest-wall. The percussion note over the fluid is one of stony dullness, and the sense of resistance is greatly increased. The exact limits of this area of dullness should be determined with the patient sitting up and recumbent. With moderate effusions the upper level is usually found to assume a curved line, with the summit in the mid-axilla; this is known as the S-shaped curve of Ellis or Damoiseau's line. In the recumbent position, a change in the level of this line may be observed, particularly in the front of the chest and in the axilla. This shifting dullness forms one of the pathognomonic signs of fluid, but it is not always easy to demonstrate. It is much more apparent in cases of pyo-pneumothorax. In large effusions, the dullness may extend up to the level of the clavicle and reach across the mid-line of the sternum; moreover, in left-sided effusions it blends with the cardiac dullness, and the area of gastric resonance, known as Traube's space, may be encroached on or obliterated. The relaxed lung above the effusion in front often yields a skodaic note, which becomes dull if the quantity of fluid increases. At the back there is a triangular area of relative or moderate dullness above the stony dull area of fluid. This is known as Garland's dull triangle. It also corresponds with the relaxed or collapsed lung. At the extreme base on the contralateral side there is often a small area of dullness known as Grocco's triangle. The apex is usually at the vertebral column, about the upper level of the effusion, the base extends outwards at the lower margin of the lung for 1 or 2 inches. This paravertebral dull area is believed to be due to mediastinal displacement by the effusion. Elsewhere over the sound lung the note may be slightly hyper-resonant. The area of deep cardiac dullness should be carefully marked out. In left-sided effusions it is displaced to the right and extends beyond the sternum in the third and fourth spaces, even to the nipple line or beyond it. In right-sided effusions, the displacement may be very obvious, the left margin of the dullness extending as far out as the left mid-axillary line. The auscultatory signs are very variable, and much less characteristic than those obtained by palpation and percussion. In some cases, the breath-sounds over the dull area are distant and weak or even absent, in others they are loud and bronchial or tubular. This inconstancy probably depends upon the extent of pulmonary collapse and the degree of patency of the bronchi. With marked collapse and patent bronchi, bronchial breathing is heard; with partial collapse and obstructed bronchi, the breath-sounds are almost or quite abolished. As a rule, no adventitious sounds are heard, but râles may be audible in the lung above the effusion. Conduction of spoken voice is diminished or abolished, but towards the upper part of the effusion and just above it, the sound produced is heard distantly and with a peculiar nasal or bleating twang, a condition known as ægophony. Baccelli stated that the whispered voice is conducted through a serous but not through a purulent effusion, and called this sign "pectoriloquie aphonique," but no reliance can be placed upon this as a diagnostic sign. The breath-sounds heard under the clavicle over the relaxed

lung above the effusion are frequently harsh or puerile. In the contralateral lung the breath-sounds may be vesicular or exaggerated, and in cases of large effusions, where there is marked circulatory obstruction, there are frequently signs of congestion or oedema at the base. Similarly pressure on the descending thoracic aorta may cause lowering of the blood-pressure in the leg as compared with that in the arm (O. K. Williamson). There may be a systolic murmur over the cardiac region (displacement murmur). The abdomen should be examined to determine any downward displacement of the liver or spleen. The blood count in sero-fibrinous pleurisy rarely shows any leucocytosis, apart from complications.

Complications and Sequelæ.—Acute oedema with albuminous ex-
pectoration is rare, but is a dangerous condition unless treatment is prompt. Permanent collapse and carnification of the lung may remain after absorption in prolonged cases, and may progress to diffuse interstitial fibrosis. More commonly some degree of pleural thickening and adhesion persists, and expansion of the lower lobe may never be completely restored. Sero-fibrinous effusion due to tuberculosis rarely becomes purulent, but this sequence is common in other forms. Tracking of the fluid externally through the chest-wall and rupture through the lung occur but rarely. An infrequent complication is hemiplegia, probably due to an embolus derived from a thrombus originating in a pulmonary vein. Miliary tuberculosis occasionally follows rapidly on an effusion; more commonly active tuberculosis of the lungs occurs after a lapse of some years.

Course.—In effusions of moderate size the temperature usually subsides in from 7 to 10 days, and spontaneous absorption is complete in 2, 3 or 4 weeks. In large effusions reaching up to the second rib or higher, the course may be less favourable. The fever may persist even for weeks, and absorption of the fluid may be slow or wanting entirely. Aspiration may accelerate the resolution, and usually only one tapping is necessary, the fluid left behind being absorbed rapidly. In rare cases fluid reaccumulates quickly after repeated tapplings, and a so-called inexhaustible effusion occurs. In some such patients fluid may remain in the pleura for the rest of life.

Diagnosis.—The recognition of the presence of fluid in the pleural cavity is generally easy, but with small or localised effusions it may be difficult. The most valuable signs are the displacement of the heart, the absence of vocal fremitus, and the stony resistant dullness. The auscultatory signs are of less value, and may even be misleading. The chief conditions which may simulate effusion are fibroid lung with thickened pleura and bronchiectasis, pneumonia, particularly the massive form, malignant disease of the lung, pleura or mediastinum, massive collapse, a large pericardial effusion, and an aneurysm pressing on one or other main bronchus. Subphrenic abscess may also give rise to difficulty (see Empyema). Fibroid disease can usually be recognised, since there is generally flattening and sinking-in of the affected side instead of bulging. The heart, if displaced, is drawn towards instead of away from the affected side, vocal fremitus is present although possibly diminished, and the dullness is rarely of the stony character obtained over fluid. The breath-sounds are generally weak, and if bronchiectasis is also present, the characteristically variable signs of that condition should be helpful in diagnosis. In massive pneumonia the differentiation may be difficult, since breath-sounds and voice-sounds are sometimes completely

absent, but the position of the cardiac impulse is generally of decisive importance. In malignant disease and aneurysm, careful observation should afford diagnostic indications, such as glandular enlargement or abnormal pulsation, and in both instances the X-rays may establish the diagnosis. Malignant disease of the pleura may first show itself as a pleural effusion; the tendency to recur after tapping, the presence of blood in the effusion, and the onset of emaciation may help to suggest the cause. In massive collapse there is, as a rule, but little difficulty, owing to the displacement of the cardiac impulse to the affected side. In pericardial effusion the shape of the cardiac dullness may be suggestive, and the dislocation of the impulse may indicate the real condition; moreover, the dullness over the lung behind is rarely of extreme degree unless pleural effusion co-exists. In any doubtful case, examination by the X-rays is desirable, since it may give valuable aid in diagnosis. The shadow of serous fluid is generally dense, but does not obscure the rib shadows completely. The upper level is curved and shifts to some extent with the position of the patient. It merges into the shadow of the collapsed lung above. The diaphragm is immobile on the affected side. A further aid to diagnosis consists in exploratory puncture, which has the advantage of establishing the nature of the fluid as well as its presence. The technique of puncture is similar to that of paracentesis described on pages 1253, 1254, save that a 5 or 10 c.c. syringe with a needle long enough to enter the pleura is used instead of an aspirator. The preliminary local anaesthesia by novocain or some similar preparation, with or without adrenaline, should be employed in every case, not only to avoid pain but also to obviate the risk, remote though it be, of pleural shock. Serous pleural fluid of inflammatory origin varies in colour from pale greenish yellow to brown. The specific gravity is usually 1018 or over. Protein is present as serum albumin, serum globulin and fibrinogen, the total quantity being, as a rule, over 4 per cent. The fluid generally clots spontaneously after withdrawal. The cytology of the fluid is varied, showing lymphocytes, polymorphonuclear cells, erythrocytes and altered endothelial cells in varying proportions. A marked preponderance of lymphocytes is very suggestive of a tuberculous origin, while the presence of large numbers of polymorphonuclear cells is usually an indication of some other infection, generally by a pyogenic organism. In rare cases large numbers of eosinophils have been found. The origin of these cases of so-called "eosinophil pleurisy" is at present doubtful. Cultural examination of tuberculous fluid usually proves sterile unless Loewenstein's medium is used, but in fluid from other causes the infecting organism can often be grown. To establish the tuberculous nature of a pleural fluid, inoculation of 15 c.c. of the fluid into a guinea-pig may be tried. Other methods formerly employed were examination of the centrifugate from the fluid, and Jousset's "inoscopy," which consists in examination of the clots derived from the fluid after they have been submitted to artificial gastric digestion. These two methods, however, fail in many cases. The methods of differentiation of an inflammatory exudate from a passive transudate are given on page 1259.

Prognosis.—The immediate prognosis is good, although with large effusions of 4 pints or more, sudden death sometimes occurs from acute oedema of the lungs, cardiac failure or embolism. The ultimate result depends on the cause. In non-tuberculous effusions, recovery may be complete, save for

pleural adhesion, or they may progress to empyema. In tuberculous effusions arrest may remain complete, but, as already stated, a considerable proportion of the cases develop active pulmonary disease in after years.

Treatment.—The patient should be kept in bed in an airy and well-ventilated room until the temperature is normal. Fluid should be restricted, and the diet may be salt-free with advantage. The administration of diuretics, diaphoretics and saline or mercurial aperients may assist in the disappearance of the exudate. The use of iodide of potassium has been recommended, but it is of doubtful value in these cases. The application of counter-irritants to the chest-wall in the form of iodine or of fly blisters is often helpful. A sedative lozenge or linctus may be given for the irritating cough present in the early stage. Exploratory puncture is generally advisable to permit the examination of the fluid. Opinions differ somewhat as to the indications for paracentesis, which, however, is nowadays performed earlier and more frequently than was formerly the case. It is unnecessary in cases in which absorption of the fluid is apparent within 10 days. The following conditions may be considered to suggest its employment: (1) if the effusion is large and causing positive pressure, as shown by dullness up to the clavicle, marked dyspnoea, downward displacement of the liver or spleen, and collateral hyperæmia of the sound lung; (2) if absorption is slow, the fluid remaining at the same level for a fortnight or more; (3) if acute oedema with albuminous expectoration occurs; (4) in cases of bilateral effusion with increasing dyspnoea, the side with the larger effusion may be aspirated. Paracentesis can be performed in various ways. The simplest method is that of siphonage; a long rubber tube filled with sterile saline solution is attached to a trocar and cannula, which are passed into the pleural cavity and the fluid is siphoned into a receptacle at a lower level. This method has the great advantages that the degree of suction employed is under control, and the lung expands gradually as the fluid is withdrawn. It is often difficult to remove a large quantity of fluid by this means, and it fails in loculated effusion. Aspiration is more generally effective, and may be carried out either by Dieulafoy's pump and two-way tap, Martin's syringe, or by Potain's apparatus. With these methods it is impossible to withdraw all the fluid, and removal with air replacement is now often practised with advantage. For this purpose an aspirator and an apparatus like that used in the induction of artificial pneumothorax are required. This method permits of almost complete removal of the fluid, prevents cough and discomfort, lessens the tendency to recurrence of the effusion and promotes expansion of the lung. In performing aspiration the patient should sit up in bed, or lie slightly turned on the unaffected side, and the area for operation should be painted with iodine. The skin and muscles down to the pleura should be anæsthetised with procaine (novocain) or other local anæsthetic, and a small incision made through the skin, though this is not essential in the case of a small instrument. The trocar and cannula are then pushed carefully into the pleural cavity just above a rib to avoid puncturing the intercostal artery. The sites chosen depend on the situation of the fluid, but the most convenient are in the sixth space in the mid-axilla, the seventh space in the posterior axillary line, or the eighth space just below the angle of the scapula. Aspiration should be stopped if cough occurs, if pain is severe, or if albuminous expectoration with signs of oedema supervenes. In rare cases sudden death from pleural

shock has occurred. The risk of this may be obviated by careful local anaesthesia down to the pleural level. Other risks are due to faulty technique, and comprise entrance of air into the pleural cavity from wrong connection of the apparatus or from wounding the lung, and infection of the pleural cavity from failure in the aseptic preparations leading to empyema. Air replacement seems preferable, since it allows of almost complete removal of the fluid.

After absorption or removal of the fluid, re-expansion of the lung may be promoted by the use of Wolff's bottles, or by appropriate breathing exercises. With the former, fluid is forced from one bottle to another by blowing. In the latter, the patient takes deep inspirations while seated in a chair with the sound side partly fixed. In all cases in which a tuberculous origin is proved or suspected, prolonged convalescent treatment on sanatorium principles is advisable.

ANOMALOUS PLEURAL EFFUSIONS

Two unusual forms of pleural effusion require brief mention—they are encysted interlobar and encysted diaphragmatic sero-fibrinous pleurisy. The former of these can only be recognised by X-ray examination followed by exploratory puncture. Encysted diaphragmatic sero-fibrinous pleurisy is rare, but a case has been erroneously recorded as acute serous mediastinitis. This condition is simply one of pleural effusion localised to the space between the mediastinal pleura, the diaphragm and the lung. Both of these conditions, if diagnosed, should be treated on general principles. The effusion may absorb spontaneously, but if not, aspiration may be necessary.

PURULENT PLEURISY (EMPHYEMA)

In this condition the pleural exudate becomes purulent. The fluid may be turbid and the presence of pus be apparent only on microscopical examination, or it may consist of typical pus.

Ætiology.—**PREDISPOSING CAUSES.**—Empyema is common in children under 10 years of age, and the younger the child the greater the probability that any effusion will be purulent. In adults it is commonest between the ages of 20 and 40 years, probably owing to the heavy incidence of pneumonia in this age period. Debility, exposure and alcoholism may promote its occurrence. Purulent pleurisy is but rarely primary, except in the form due to the pneumococcus. It is most commonly due to extension from the lungs, especially from lobar pneumonia and from bronchopneumonia. Other pulmonary causes are tuberculosis, bronchiectasis, abscess, gangrene, new-growth, or septic infarcts in infective endocarditis. It may develop in association with mediastinal lesions, such as suppurating glands, ulcerating carcinoma of the œsophagus, or from suppuration in the neck tracking downwards. Infection of the pleura may occur through the chest-wall as a result of gunshot wounds, stabs, fractured ribs, and faulty technique in aspiration of a serous effusion. The primary source of pleural infection may be in the abdomen, the organisms passing through the diaphragm from a perinephric, subphrenic, or hepatic abscess, or from localised or generalised peritonitis consequent on rupture of a gastric or duodenal ulcer. The involve-

ment of the pleura may take place through the blood in septicæmia, suppurating gunshot wounds, compound fracture of the femur, and in otitis media with lateral sinus thrombosis.

Empyema may develop during the course of many of the acute specific fevers, such as scarlet fever, variola, measles and the enteric group; but since in these conditions it is usually secondary to broncho-pneumonia, it belongs strictly to the pulmonary group.

EXCITING CAUSES.—The organisms most frequently found in purulent effusions are the pneumococcus and the streptococcus, the former accounting for more than half of the cases. Occasionally the pus proves to be sterile on culture; such cases are generally the result of the tubercle bacillus or of a pneumococcus which has died out. Other organisms less commonly found are staphylococci, Pfeiffer's *H. influenzae*, the *B. typhosus*, and Friedländer's pneumo-bacillus. Streptothrix organisms are occasionally found (see Actinomycosis), also various saprophytes and anaerobic organisms, especially in fetid empyema.

Pathology.—The initial stages are similar to those of dry and sero-fibrinous pleurisy, but when the effusion occurs, it proves to be rich in leucocytes undergoing disintegration and to contain the infecting organism. It varies from a slightly turbid semi-translucent fluid to typical thick, opaque, creamy pus. Its colour ranges from pale amber to green or greenish grey. It may be odourless or extremely offensive. In cases secondary to gangrene, it may be thin and horribly fetid, while in pneumococcal cases it may be curdy and of slightly sweetish odour. The pleura is covered with a more or less thick layer of sodden fibrinous exudate. In cases due to the pneumococcus this false membrane may be very thick. Adhesions form quickly, leading to encystment or loculation of the pus. Such adhesions also prevent the lung from expanding after evacuation of the pus, with the result that the lung becomes carnified and interstitial fibrosis results. There is usually some enlargement of the bronchial glands. In long-standing cases there may be lardaceous disease of the liver, spleen, kidneys and intestines.

Symptoms.—Since empyema usually develops in the course of, or as a sequel of, some other disease, its symptoms are often masked by those of the primary disease and may easily be overlooked. In primary cases due to the pneumococcus the onset may be like that of pneumonia; in the more common secondary cases a rise of temperature and increase of signs develop after the crisis. In general it may be stated that the symptoms are similar to those of sero-fibrinous pleurisy, but more severe. There is more malaise, and the patient may appear profoundly ill, with rigors, sweats and dyspnoea. The temperature ranges higher, up to 103° F. or more, and may be of septic type with marked daily remissions, but some cases are almost if not completely apyrexial. The signs are usually exactly similar to those of sero-fibrinous effusion, but in some instances special features may be noticed. In neglected or prolonged cases, wasting, pallor and cachexia become marked. The intercostal spaces may be found to bulge, and œdema of the chest-wall is sometimes apparent. The pus may track through an intercostal space, generally the fifth near the nipple, producing a fluctuating swelling known as a pointing empyema or *empyema necessitatis*. This may infiltrate the skin and simulate a superficial abscess. The swelling so induced may pulsate, especially if it be on the left side—a condition known as pulsating empyema.

Pulsation communicated to the chest-wall may also be observed in some large left-sided purulent effusions without local swelling. The displacement of the liver or spleen may be greater than with serous effusions, probably owing to the higher specific gravity of the pus, which is usually 1030 or more, and to the associated inflammation of the diaphragm. In fetid empyema, the breath and sputum may be offensive, even before rupture into a bronchus occurs. Clubbing of the fingers and toes occurs in empyema of long standing, but may develop in a few weeks. Blood examination reveals a moderate leucocytosis in the majority of cases. Counts of 15,000 leucocytes per cubic millimetre are usual, and in some instances figures up to 100,000 per cubic millimetre are obtained.

Complications and Sequelæ.—In neglected or untreated empyema the pus may track and become discharged in various directions. The commonest is rupture through the visceral pleura into the lung and discharge through a bronchus. This may lead to sudden death from suffocation; on the other hand, in small empyemata spontaneous cure may follow this evacuation of the pus. In other instances pyo-pneumothorax results, and occasionally gangrene of the lung. A second method of discharge is through the chest-wall, as an *empyema necessitatis*. Perforation may occur into the pericardium, or into the œsophagus with the formation of a pleuro-œsophageal fistula. The diaphragm may be perforated with the production of a subphrenic, lumbar or psoas abscess, while in other cases general peritonitis may ensue.

The pericardium or the mediastinum may become infected without perforation; similarly costal periostitis may be induced. After spontaneous or operative evacuation the cavity may fail to close and a chronic empyema or sinus result. This is generally due to the lung being permanently collapsed and adherent, and therefore failing to expand. It subsequently undergoes fibrosis with development of bronchiectasis. Sometimes the failure to close may be due to the nature of the infection, particularly when it is due to tuberculosis or actinomycosis (streptotricosis). In other cases it may be due to a bronchial fistula, or to a foreign body in the pleura. Generalised infection is rare, but cerebral abscess, probably of embolic origin, is not very uncommon, especially in cases due to streptococci. Chronic pulmonary osteo-arthritis is an occasional complication, and lardaceous disease sometimes occurs in cases of long duration. Diphtheritic infection of the wound, with subsequent paralysis, has been recorded after operation, more especially in cases secondary to influenzal broncho-pneumonia.

The sequelæ in untreated cases may be fistulæ, such as pleuro-bronchial, pleuro-œsophageal or external, and various deformities. The sequelæ after operation may be a small amount of pleural thickening, or if operation were delayed, and re-expansion incomplete, there is falling-in of the chest, with flattening, dropping of the shoulder and secondary scoliosis. In other cases, as mentioned above, a chronic sinus may result.

Course.—Apart from spontaneous cure of small empyemata by inspissation of the pus, or discharge through a bronchus or through the chest-wall, death generally occurs in untreated cases within a month or two. As in sero-fibrinous pleurisy, sudden death may occur. Death may occur after operation, from exhaustion or from cerebral abscess.

Diagnosis.—The diagnosis of empyema involves two distinct problems—one, the recognition of the presence of fluid in the pleura, which is considered

under sero-fibrinous pleurisy; the other, the demonstration of its purulent character. In spite of the more severe symptoms, empyema is frequently overlooked even by physicians of experience. This is partly due to the fact that its development may be insidious, with signs increasing but little from day to day, and partly to its secondary character, its onset being obscured by the clinical features of the primary condition. It is wise, therefore, to suspect its existence in any case of obscure lung signs, especially those with dullness, cardiac displacement and fever, consequent on pneumonia of any variety.

There are a few special difficulties as compared with sero-fibrinous effusion which merit separate mention. The first of these is subphrenic abscess. This may lead to immobilisation of the diaphragm on one side, more commonly the right, and cause collapse of the lung and even pleural effusion. The difficulty is the greater when the subphrenic abscess contains gas as well as pus. The history, the absence of displacement of the heart's impulse, and examination by X-rays may all assist, but the differentiation is often extremely difficult.

Empyema necessitatis may simulate a tuberculous or other abscess about a rib, and empyema should always be suspected in any case of local fluctuating swelling about the chest-wall. Pulsating empyema requires to be distinguished from aortic aneurysm; the pulsation is less forcible and little, if at all, expansile in the former. The cardiac displacement, the X-rays and cautious exploratory puncture, enable the nature of the condition to be recognised.

In any case in which empyema is suspected three examinations may be undertaken—a blood count, radiographic methods, and exploratory puncture. A polymorphonuclear leucocytosis of 15,000 per cubic millimetre and over, a dense shadow in the radiogram obscuring the ribs, together with cardiac displacement may be very suggestive, while puncture may prove the presence of pus. Sometimes, however, puncture may fail, although pus is present. This may be due to the pus being too thick to pass through the needle, to loculation of the pus, or to wrong choice of the site for puncture. In this case, if the other signs indicate pus, repeated punctures with a larger needle under anæsthesia are called for, but it is well to be prepared to proceed to operation if pus is found.

Prognosis.—This depends upon the primary cause, the method of treatment adopted, and the duration of the effusion before the operation. The most favourable forms are those due to the pneumococcus, which are recognised and treated at an early stage. In neglected cases, with profound toxæmia, with gangrene of the lung or lardaceous disease, the outlook is extremely grave. Empyemata due to streptococcal infection are serious, unless recognised early; similarly with cases of fetid empyema due to anaerobic infections. Infected hæmothorax consequent on gunshot wounds of the chest is of grave prognosis. The outlook is serious in cases of bilateral empyema, but recovery may follow successive evacuation of the pus on the two sides.

Treatment.—This consists in the evacuation of the pus by operation as soon as the diagnosis is established in pneumococcal cases. In those of streptococcal origin, operation should not be resorted to while the fluid is of thin sero-purulent character, but should be postponed until it is definitely purulent. Premature operation in streptococcal cases has been shown by

the American Empyema Commission to be a very dangerous procedure, since the fluid is not shut off by adhesions and operation may lead to open pneumothorax, with flapping mediastinum. At this stage, the condition is described as pyothorax. A preliminary aspiration is of advantage in large effusions, and may be repeated in streptococcal effusions until they are ready for operation. The operation consists in drainage by removal of a piece of rib subperiosteally and incision of the parietal pleura. For the operation a general anæsthetic may be given, but it is now almost always carried out under local anæsthesia; but if the patient's condition renders this undesirable, an incision under local anæsthesia may be made through an intercostal space and a drainage tube inserted, a piece of rib being removed later under general or local anæsthesia when improvement has occurred. The wound is dressed at least daily and the drainage tube sterilised, every endeavour being made to prevent secondary infections. To this end the pleural cavity may be irrigated daily by the Carrel-Dakin method, or washed out with some antiseptic such as flavine or brilliant green. To avoid pleural shock, free exit for the wash fluids must be ensured. By some authorities pneumococcal empyemata, particularly in young children, are treated by repeated aspirations or by siphon drainage. If the pus is thick and difficult to evacuate, incision of the pleura with immediate suture is performed, any reaccumulation being treated by aspiration with or without oxygen replacement. If, however, toxic symptoms persist, drainage should be effectively established at once.

In cases of chronic empyema, or of sinus failing to close, the question of some plastic operation must be considered. Various forms of operation have been devised, involving removal of portions of many ribs, and the decortication operation of Fowler and Delorme. The general condition of the patient must be carefully considered before these operations are advised. In some cases an autogenous vaccine seems to be of value, if drainage is satisfactory.

SPECIAL VARIETIES OF EMPYEMA.—Certain special localisations of purulent pleurisy require separate consideration, notably apical, interlobar and diaphragmatic empyemata.

Apical empyema.—This condition is usually secondary to apical pneumonia, less commonly to pulmonary tuberculosis. It is one variety of encysted empyema, the pus being shut off from the rest of the pleural cavity by adhesions. The symptoms and signs are not characteristic, but may be suggestive. There is very marked dullness below the clavicle, not transgressing the middle line, with weak or absent breath-sounds, and possibly some indications of mediastinal displacement. Diagnosis can, as a rule, be established only by the X-rays and exploratory puncture, the latter being carried out in the second space near the mid-clavicular line. The treatment consists in drainage by incision as near the lower limit of the effusion as possible.

Interlobar empyema.—Pus collecting between two of the lobes may be difficult to differentiate from pulmonary abscess, gangrene and bronchiectasis. It is often not diagnosed until rupture into a bronchus draws attention to it. The signs are generally most marked in the axilla or near the angle of the scapula. They are often slight until rupture occurs, and even then there may be only a small area of dullness in the line of an interlobar fissure, with distant or weak bronchial breathing and a few râles. The pus expectorated may be fetid, and the patient's breath may be offensive a few days

before rupture occurs. The condition simulates abscess of the lung, and may be almost impossible to differentiate from that affection. Examination by the X-rays gives the greatest help in the diagnosis. Recent observations suggest that interlobar empyema is much less common than abscess. The treatment is identical with that for pulmonary abscess.

Diaphragmatic empyema.—The pus is usually encysted, and may be so deeply situated as to give but few signs. The initial symptoms are generally severe, being those of diaphragmatic pleurisy, but hiccough is often a troublesome feature. When pus forms, there may be marked constitutional symptoms, and obscure signs may develop, such as dullness, at a point just above the base behind, with weak or distant bronchial breathing. With such a history and obscure basic signs, especially when they occur after an attack of pneumonia, the use of the X-rays and of the exploring needle should not be neglected. In cases not recognised and treated, rupture into a bronchus or through the diaphragm may occur. The treatment is similar to that for ordinary empyema.

HYDROTHORAX (DROPSY OR HYDROPS OF THE PLEURA)

Hydrothorax is the name applied to a collection of clear fluid in the pleural cavity, the result of passive transudation from the capillaries.

Ætiology.—The commonest cause of hydrothorax is cardiac failure from chronic valvular disease, or from myocardial weakness or degeneration. It occurs in acute and chronic renal disease, under conditions similar to those leading to dropsy in these affections. It is sometimes found in severe anæmias, especially pernicious anæmia. Obstruction to the azygos veins may lead to transudation into one pleural cavity or into both. This obstruction may be induced by pressure from without by a mediastinal or pulmonary new-growth, or by internal causes such as thrombosis.

Pathology.—The pathology of hydrothorax is that of dropsy elsewhere. It is produced by mechanical or chemical conditions affecting the blood flow through the capillaries, and it must be distinguished carefully from inflammatory effusion. There is a difference in the composition as well as in the origin of the two kinds of pleural fluid. The characters of inflammatory effusions have been described under pleurisy with effusion. The fluid in hydrothorax is pale yellow in colour, and the specific gravity is 1015 to 1010 or less. It is clear and does not clot after removal. There is little protein, often not more than 1 per cent., but transudates due to local obstruction may contain as much as 3 per cent. The cellular elements are scanty, although some endothelial cells may be present, often united together in plaques. The fluid may be definitely bloodstained, when it is described as hæmo-hydrothorax.

Hydrothorax is usually bilateral in cases due to cardiac or renal disease, but in the former there is often more fluid on the right side, or the fluid may be confined to that side. The explanation of this is somewhat obscure. It has been suggested that it is due to pressure or traction on the vena azygos major by the enlarged right heart, but according to Fetterolf and Landis, a more likely explanation is pressure of the distended right auricle upon the pulmonary veins. Fluid may also collect in greater quantity on the side

upon which the patient lies most constantly. In cases with unilateral pleural adhesion, œdema of the lung may occur on that side, while hydrothorax occurs upon the other.

Symptoms.—The symptoms of hydrothorax are generally overshadowed by those of the condition causing it, but the occurrence of dyspnoea and cyanosis in any case of cardiac or renal disease should suggest careful examination of the bases of the lungs. In the absence of inflammatory complications the condition is afebrile. The signs are identical with those of sero-fibrinous pleurisy, save that no friction sounds are audible at any stage. It is, however, more difficult to assess the significance of displacement of the cardiac impulse, owing to the increased size of the heart in the cases of cardiac origin.

Diagnosis.—This depends upon the presence of signs of fluid in the pleura in association with cardiac or renal disease, with absence of fever, and also upon the characters of the fluid withdrawn by puncture or aspiration.

Treatment.—Removal of the fluid may give great relief. It may be necessary to repeat the operation, since the fluid often reaccumulates. The treatment of the primary condition should also be carried out.

HÆMORRHAGIC PLEURAL EFFUSIONS

All fluids poured out into the pleura contain a certain number of red blood corpuscles. It is only when a number sufficient to give a definite red colour are present, that the fluid is regarded as hæmorrhagic.

For convenience of description three forms may be differentiated—

- (1) Hæmorrhagic pleurisy or hæmo-serothorax ; (2) hæmo-hydrothorax ; and (3) hæmothorax.

1. HÆMORRHAGIC PLEURISY.

This is simply a pleurisy with effusion, in which the exudate is blood-stained.

Ætiology.—The usual causes are malignant disease of the lungs, pleura or mediastinum, and rarely tuberculosis of the lung and pleura. Hæmorrhagic pleurisy may occur in association with hepatic cirrhosis, but in this case it is often the result of a terminal tuberculosis. It occurs less frequently in association with blood diseases, such as purpura, and with the malignant or hæmorrhagic varieties of acute infectious fevers such as scarlet fever and small-pox, and occasionally with lobar pneumonia. Sometimes in tapping a sero-fibrinous effusion for the second-time, it is found that the fluid, which was originally clear, is now blood-stained. This is not necessarily an indication of increase in the severity of the process, but may be due to injury of a blood vessel at the first operation.

Symptoms.—The symptoms and signs are identical with those of serous effusion, and the hæmorrhagic character can only be recognised by withdrawal of the fluid. An interesting point is the frequency of excess of eosinophils in these effusions. Diagnosis and treatment are the same as for sero-fibrinous pleurisy.

2. HÆMO-HYDROTHORAX.

This condition has been referred to under hydrothorax. It consists simply in blood-staining of a passive transudate into the pleura.

3. HÆMOTHORAX.

Hæmorrhage into the pleural cavity is the result of injury or disease of the vessels of the lung, mediastinum or chest-wall.

Ætiology.—The chief causes are injury, such as penetrating chest wounds or fracture of the ribs, rupture of an aneurysm, and erosion by new-growth. Experience of the traumatic group has been largely increased during the War of 1914–1918. Hæmothorax was noted in about 70 per cent. of chest wounds.

Pathology.—The effused blood generally comes from the lung vessels, less commonly from the intercostals. It is “whipped” by the movements of the heart and lungs, with the result that fibrin is deposited in layers upon the diaphragmatic pleura, and the parts of the visceral and parietal pleura in contact with the blood. The fluid remaining in the pleura or withdrawn by aspiration is largely defibrinated and therefore does not clot, unless a secondary pleurisy develops.

The lower lobe of the lung on the affected side becomes collapsed and eventually carnified, unless absorption occurs or unless the blood is aspirated. The upper lobe may show some compensatory emphysema, and adhesions may form in the pleura, separating it from the hæmothorax below. When secondary infections of the bronchi or lungs occur, such as bronchitis or broncho-pneumonia, the collapsed lower lobe is not affected.

Symptoms.—The symptoms of hæmorrhage into the pleura from medical causes, such as rupture of an aneurysm or erosion of a large vessel, are collapse and rapid death. When due to disease or injury of an intercostal vessel, they may be insidious and slowly ingravescent until dyspnoea, restlessness and the other indications of internal hæmorrhage develop. When due to injury, similar symptoms occur, but may be masked or overshadowed by the shock, hæmoptysis and cough, induced by the wound of the lung or chest-wall. The signs are those of pleural effusion, but in traumatic cases certain special features may be mentioned. There is a great tendency to retraction of the chest-wall on the affected side, and the cupola of the diaphragm on this side is displaced upwards. This is thought to be due to an active lobar collapse of the lung, the lung contracting, not as the result of the pressure of the effusion, but in consequence of a nervous protective reflex initiated by the trauma. Vocal fremitus is usually diminished or absent. The breath-sounds over the effusion are frequently bronchial, and well-marked bronchophony and pectoriloquy may be present.

Complications and Sequelæ.—The most serious complication is infection of the effusion. This is generally due to organisms introduced at the time of the wound, either by the missile or by portions of the clothing or skin carried in with it. Aerobic organisms, such as a streptococcus, or anaerobic ones, as the *B. aerogenes encapsulatus* or the *B. sporogenes*, may be present. A hæmo-pneumothorax may develop, the gas entering the pleural cavity from the wound in the lung or through the chest-wall. Gas may also be formed by gas-producing infecting organisms in the effusion. Massive collapse may occur in the contralateral lung, or other complications may arise, such as bronchitis, broncho-pneumonia, lobar pneumonia or œdema of the lungs. If the effusion is small and not infected, there are usually no permanent after-effects. In severe cases sequelæ, similar to those of sero-fibrinous pleurisy and empyema, may result.

Course.—This depends upon the cause and size of the hæmothorax, and upon the mode of treatment adopted. It is profoundly and gravely influenced by infection of the effused blood. A small sterile hæmothorax is generally absorbed spontaneously. Medium-sized and large effusions may not disappear unless aspirated. An infected hæmothorax will inevitably prove fatal, if untreated.

Diagnosis.—Hæmothorax should be suspected when basic dullness develops shortly after a gunshot wound of the chest. The mistake that is most likely to be made in such cases is to confuse hæmothorax with lobar pneumonia. The cardiac displacement and the diminution of vocal fremitus over the dull area are the most valuable diagnostic signs. An active lobar collapse is distinguished by the fact that the heart is displaced towards the affected side. The X-rays afford valuable confirmatory evidence in most cases. When air and blood are present, the upper border of the dark area in the radiogram has a sharply defined edge, while the pleural cavity above is very translucent. The use of the exploring syringe generally settles the diagnosis, except in certain cases in which, although a considerable quantity of blood may be present, none is removed by aspiration owing to the needle entering the clot.

Prognosis.—In a sterile hæmothorax due to a chest wound the prognosis is good. If infection occurs, the prognosis depends upon the promptitude with which this condition is recognised and radically treated, although in very acute infections death may occur in 2 or 3 days despite immediate operation.

Treatment.—A small sterile hæmothorax may be left untouched. In medium and large-sized effusions, recovery is accelerated by aspiration. The possible danger of renewal of the hæmorrhage, as the result of lowering the pleural tension by this operation, is very slight, and negligible if it is delayed until 2 or 3 days after the wound. If the temperature in a case of hæmothorax rises suddenly to 102° or 103° F. in the evening, it is criminal to wait until the next morning to see what will happen. A specimen of fluid should be withdrawn and examined microscopically. Direct films may occasionally reveal the presence of organisms, but the important point to determine is the number of polymorphonuclear leucocytes present. When these are numerous, operation should be performed without awaiting the findings of aerobic and anaerobic cultures. A rib should be resected as in empyema, and the blood and clots should be removed from the pleural cavity and drainage established.

CHYLOUS AND OTHER MILKY EFFUSIONS

A milky fluid is occasionally obtained on exploratory puncture or aspiration of a pleural effusion. It is usual to classify such fluids into three groups—(1) Chylothorax; (2) chyloform fluid; (3) pseudo-chyloous fluid.

1. CHYLOTHORAX.

There is an effusion of pure chyle or of serous fluid mixed with chyle.

Ætiology.—Chylothorax is usually the result of injury to, or disease of, the thoracic duct. The traumatic form is, as a rule, secondary to crushing of the chest-wall with fracture of the ribs. In disease, the thoracic duct may be pressed on by a malignant growth or enlarged mediastinal glands,

or the flow may be obstructed by thrombosis of the left subclavian vein. Invasion of the thoracic duct by the *Filaria sanguinis hominis* may also be a cause.

Pathology.—The fluid in true chylothorax is a milky emulsion which remains so on standing, although a cream-like layer may form at the top. With the microscope fat globules can be seen, which stain with the usual fat stains and can be dissolved by ether.

2. CHYLIFORM EFFUSION.

In this condition fat is present, but it is not derived from the thoracic duct.

Ætiology.—Chyliform effusions occur in association with tuberculosis and carcinoma of the pleura or lung.

Pathology.—The fluid is milky and contains fat in emulsion, although in smaller quantities than in true chylothorax. On microscopical examination large fat droplets are seen, and numbers of cells, chiefly leucocytes undergoing fatty degeneration. It is, no doubt, from this process that the fat is derived.

3. PSEUDO-CHYLOUS EFFUSION.

In this condition the milky appearance is not due to fat, but to other particles causing opalescence.

Ætiology.—Pseudo-chylous fluid has been observed in chronic effusions due to heart disease, nephritis, tuberculosis and malignant disease.

Pathology.—The milky appearance is due in some cases to a lecithin globulin complex (Wallis and Schölberg). Other rare causes of milky, opalescent or turbid effusions are the presence of particles of calcium phosphate, cholesterol or filarial embryos. These fluids are distinguished from the above by showing a deposit on standing.

Diagnosis.—This can only be established by microscopical and chemical investigation of the fluid withdrawn.

Prognosis.—The prognosis in most cases of milky effusions is serious, owing to the gravity of the primary condition. Some traumatic cases of true chylothorax recover.

Treatment.—The treatment is for the most part symptomatic and dependent upon the primary condition. In true chylothorax, removal of the fluid is inadvisable, unless it is causing dyspnoea or other symptoms of pressure. The drain of fat caused by it is a serious loss, especially if the tapping has to be repeated frequently. In chyliform effusions there is a marked tendency to recur after removal of the fluid.

PNEUMOTHORAX

In pneumothorax, gas, usually air, collects between the layers of the pleura, which now becomes a real instead of a potential space. When serous fluid is present as well as the gas it is called a hydro-pneumothorax, when pus forms the condition is described as pyo-pneumothorax, and when blood and gas collect the term hæmo-pneumothorax is applied.

Ætiology.—Pneumothorax is more common in men, and the maximum incidence is between the ages of 20 and 40 years, but it may occur at any age. The air may gain access to the pleural cavity in the following ways:

(1) Through the visceral pleura from the air in the lungs and bronchi. This

accounts for 95 per cent. or more of the cases. The commonest cause is rupture of a subpleural tuberculous focus. Rupture of an empyema into the lung is the next most frequent antecedent condition. Other pulmonary causes are gangrene, abscess, septic infarct, bronchiectasis, new growths of lung and pleura, and rupture of an emphysematous bulla or vesicle. Puncture of the lung during paracentesis, or rupture of the pleura over a diseased focus, owing to rapid expansion of the lung during the same operation, may lead to pneumothorax. A broken rib perforating the lung can also induce it. It may occur as a complication of artificial pneumothorax treatment, especially when this is bilateral. (2) Through the chest-wall, as a result of penetrating wounds, although pneumothorax is not a common result. An abscess in the chest-wall opening externally and through the pleura, or a discharging *empyema necessitatis*, may be a cause. (3) Through the mediastinum, by ulceration of an œsophageal growth, or of a diseased bronchial gland, into the pleura, or from accidental perforation of the œsophagus during the passage of an œsophageal bougie or œsophagoscope. (4) Through the diaphragm, from some hollow abdominal viscus, *e.g.* an ulcer of the stomach or duodenum may perforate, leading to the formation of a subphrenic abscess, which in turn may break through the diaphragm into the pleura. (5) Gas may accumulate in the pleura owing to infection of a pleural effusion by gas-producing organisms. This is generally the result of wounds.

Sudden spontaneous pneumothorax in apparently healthy persons occurs more commonly than is generally recognised, and is described as simple or benign pneumothorax. The causation is obscure. Rupture of an emphysematous vesicle, or of a latent or healed tuberculous focus, have both been suggested, though the latter is improbable, since there is usually no pleural reaction and the lung rapidly re-expands. In rare cases, however, the collapse of the lung is long-continued and may even be permanent. The condition is often recurrent, and is exceptionally bilateral. Complete recovery is the rule. Very occasionally spontaneous hæmopneumothorax occurs. The symptoms are usually more severe and a fatal result is not uncommon.

The exciting cause of pneumothorax may be physical strain or violent cough, but many cases occur while the patient is at rest or even during sleep.

Pathology.—The entrance of air between the layers of the pleura disturbs the pressure relations in the thorax in a similar way to the effusion of fluid; but whereas with the latter the process is gradual, in pneumothorax it is rapid, and the pressure within the pleura changes from the normal negative figure to that of the atmosphere, often in a few minutes or less. Mediastinal and cardiac displacements like those in pleural effusion, and due to the unopposed traction of the sound side, are also rapidly produced. The subsequent pressure relations depend upon the source of the air. If the opening is in the chest wall, the intrapleural pressure will remain equal to the atmospheric, until the opening becomes closed. If the opening is in the lung, three varieties occur: (1) the opening may remain patent, when the pressure keeps at atmospheric level; (2) the opening may be valvular, permitting the entry of air into the pleura during inspiration, but preventing its escape during expiration. In this case the pressure in the pleura rises above that of the atmosphere, and

the air within it is at a positive pressure, causing further cardiac and mediastinal dislocation with downward displacement of the diaphragm; (3) the opening becomes sealed, and there is a condition of closed pneumothorax in which the pressure may be equal to, greater or less than, that of the atmosphere.

To demonstrate pneumothorax post mortem, the autopsy may be performed under water, or a flap being made of the skin and muscles at the side of the thorax, this may be filled with water before puncturing the intercostal spaces. A third method is to dissect carefully through an intercostal space down to the pleura, when the lung will be found to be retracted. On opening the thorax the appearances vary. If the air entering the pleura is sterile, no inflammatory reaction occurs, the pleura remains shiny and no fluid is formed, the condition being one of simple pneumothorax. More commonly, bacteria gain access to the pleura with the ingoing air, or subsequently through the opening when this remains patent, with the result that either serous fluid or pus collects. In the former case the condition is described as hydro-pneumothorax, in the latter as pyo-pneumothorax. The appearances of the pleural membrane are similar to those found in sero-fibrinous pleurisy and empyema respectively. The lung is collapsed in every case of pneumothorax, and lies retracted towards the hilum and the spine. In tuberculous disease, a caseous focus or small cavity just under the pleura is the most frequent cause. The perforation may be a large circular rent or a small pin-hole, but multiple apertures may occasionally be present. The opening can usually be found, even if small, by submerging the lung under water while pumping air down the trachea. When extensive adhesions are present, the collapse of the lung is largely prevented and the pneumothorax is only partial. In such cases the perforation is frequently near the adhesions. In cases where fluid is present the diaphragm may be seen to be depressed on the affected side and its curvature lessened or reversed.

Symptoms.—In a considerable proportion of cases the onset is sudden and the condition of the patient becomes alarming at once. On the other hand, pneumothorax may develop insidiously, with surprisingly little pain and dyspnoea, so that its occurrence may be overlooked or only discovered on routine physical and radiographic examination, including a lateral film. This is more likely to be the case when perforation occurs in a lung extensively diseased or when the aperture is small, and the leak of air is slow. In the acute form of onset the patient is seized with severe pain while coughing or engaged in some extra exertion. There is often a feeling of "something having given way," and at once great dyspnoea develops with signs of collapse and severe mental anguish. The patient may appear blue, cold and clammy, breathing is rapid and shallow, the temperature falls to subnormal, the heart beats quickly and the pulse becomes small and weak. The patient is often restless, very alarmed and unable or afraid to speak. Occasionally death occurs in a few minutes. As a rule, the more acute symptoms subside in a few hours, but the temperature rises and the rapid breathing usually persists for some time. On examination the patient will usually be found sitting up, with *alae nasi* working and with rapid shallow breathing. The affected side is almost or entirely immobile and is usually bulged. The displacement of the cardiac impulse towards the unaffected side is generally

obvious, and is almost immediate. Palpation confirms the absence of movement, and vocal fremitus is found to be absent, except where the collapsed lung remains in contact with the chest-wall, over which area it may be increased. The exact position of the cardiac impulse should also be determined: in right-sided cases it will be found in the left axillary region; in left-sided cases it may be under or beyond the right nipple. The liver may be felt much depressed in right-sided cases. The note over a pneumothorax is characteristically tympanitic or drum-like, as a rule, but in cases with positive pressure the tympany may be flat and muffled. The tympanitic area should be carefully mapped out; it may be found to extend across the middle line, or to encroach on or obliterate the liver dullness in right-sided cases. On the other hand, in partial pneumothorax, the area may be small and easily escape recognition. In left-sided cases, the cardiac dullness may be completely wanting on that side, and a dull area found to the right of the sternum. This may give a useful hint as to the diagnosis. On auscultation, the breath-sounds are often absent, but they may be present at the apex, although weak. In other instances distant tubular breathing may be audible from the collapsed lung; in cases with a large patent opening hollow cavernous breathing may be heard. The voice sounds have an amphoric or metallic quality, and an amphoric echo may occur with any sound produced near the pneumothorax. Metallic tinkling is an example of this, being the quality conveyed to râles or other adventitious sounds produced in breathing. The bell sound or *bruit d'airain* is a valuable sign, but is not invariably present. It is elicited by listening to the chest, near where a coin is placed flat on it and tapped with another. A similar sound may be heard with the stethoscope on flicking over a pneumothorax with the thumb and finger. The displacement of the heart can be confirmed by auscultation, and the heart sounds may be found to have a metallic character. When air and fluid are present in the pleura the signs are somewhat modified. There is dullness at the base, which shifts its level with the patient's movements, the upper limit being straight, in contrast with the curved line of ordinary effusions. A marked succussion splash may be heard and felt on shaking the patient, or the patient may demonstrate the sign by a sudden shake or jerk.

Complications and Sequelæ.—Cardiac failure and rapid death occur occasionally. The chief complications are due to the entry of infective organisms into the pleura, leading to pleurisy and the effusion of sero-fibrinous fluid or pus. The sequelæ may be pleural adhesions in cases that recover, especially if effusion occurs. There may be also permanent collapse of the lung in long-standing cases, and in pyo-pneumothorax a fistula, either pleuropulmonary or external, may remain in spite of treatment.

Diagnosis.—The recognition of a large or of a complete pneumothorax is easy as a rule, the signs being characteristic. When a large quantity of fluid is present in an open pneumothorax, the presence of air may not be recognised until after paracentesis or X-ray examination. The latter gives information of the greatest value and sometimes demonstrates the presence of local pneumothorax where it has not been suspected. The air space between the lung and pleura shows most clearly in radiograms, and if fluid is present as well, the dead level of the upper border of the shadow, varying with position, is most characteristic. Diagnosis is more difficult in cases where

pleural adhesions exist, or where the pneumothorax is small and localised, especially if X-ray examination is not available. The following conditions may give rise to difficulty and should be considered in doubtful cases. (1) Total excavation of a lung, or a large pulmonary cavity, in either of which the note may be boxy or even tympanitic, the breath sounds amphoric and the râles metallic or tinkling, while the coin sound may be obtained. These conditions can usually be distinguished by the flattening and retraction of the chest-wall over them, and the absence of cardiac displacement, or if it exists, the traction of the heart towards the affected side by fibrosis. (2) Advanced emphysema, with complete obliteration of the cardiac dullness, may be confused with pneumothorax. Large bilateral bullæ may be mistaken for bilateral pneumothorax. (3) Massive collapse of one lung, with compensatory emphysema of the opposite side, may also be mistaken for it. In both these conditions careful examination will establish the real nature. (4) A subphrenic abscess containing gas (subphrenic pyo-pneumothorax); in this condition the diaphragm may be displaced upwards, and the note over the lower ribs may be markedly tympanitic. These signs are more suggestive when right-sided. Succussion splash and bell sound may be elicited. The heart, if displaced, is pushed upwards. The history of previous abdominal disease may be helpful, and a radiogram may give conclusive evidence of the subphrenic origin of the condition. (5) A hernia of the stomach or bowel through the diaphragm, or eventration of the diaphragm, all rare conditions, may simulate pneumothorax, but in all there is generally abdominal flattening and little if any cardiac displacement. Examination by X-rays after an opaque meal will, as a rule, establish the nature of the condition.

Course and Prognosis.—The course and prognosis of pneumothorax are profoundly influenced by the cause. In cases due to rupture of an emphysematous vesicle, or of a small localised healed tuberculous focus, where the pleura remains sterile and the aperture of entry closes, the air is usually completely absorbed in a few weeks and recovery is often complete. In tuberculous cases with moderate disease, in which the pleura remains sterile, pneumothorax may exert a favourable influence. In tuberculous cases with extensive disease, the pleura becomes infected and death usually results in a few weeks or months, although with judicious treatment life may be prolonged for years in some cases especially where surgical treatment such as thoracoplasty becomes practicable. In pneumothorax secondary to some grave disease, such as carcinoma or gangrene, the course is brief and the prognosis is grave in the extreme. In cases secondary to empyema, surgical treatment may be followed by complete recovery.

Treatment.—The indications in cases of acute onset are to relieve the patient's pain, distress and anxiety, and to lessen the intrapleural pressure, if this is positive. A hypodermic injection of morphine, gr. $\frac{1}{4}$ for an adult, with oxygen inhalations if necessary, may achieve the first of these. If dyspnoea is extreme and the cardiac displacement marked, a trocar or large hypodermic needle should be inserted through an intercostal space to allow air to escape. An initial pneumothorax needle, with a long rubber tube, the end of which is placed under water, is the safest. This simple manoeuvre may be the means of saving the patient's life in valvular pneumothorax, as well as of relieving distress. In less urgent cases, the pressure may be taken with

an artificial pneumothorax apparatus, and if the pressure be positive, as much air as is necessary may be removed by its means. In simple cases no other treatment may be required, although the puncture may need to be repeated. In cases of recurrent spontaneous pneumothorax or of persistent valvular pneumothorax, it is sometimes helpful to induce an aseptic pleurisy by means of various injections into the pleural space. Chandler recommends for this purpose a solution of gomenol in olive oil. At first 2 c.c. of a 2 per cent. solution are used, and this is gradually increased if necessary up to 20 c.c. of a 20 per cent. solution. A strong solution of dextrose is sometimes employed for the same purpose. In valvular pneumothorax Chandler has used a self-retaining catheter with a valvular attachment allowing the escape of air. If serous fluid or pus collect in the pleura, they may be withdrawn, preferably by siphonage, and in this case, as also with removal of air, too much should not be withdrawn in the early stages, as a slight positive pressure may assist in closure of the aperture in the lung, whereas a negative pressure may open it, after it has begun to close.

The question of operation in pneumothorax may be difficult to decide. In cases secondary to empyema, resection of a part of a rib and drainage often lead to satisfactory results. In cases of moderately severe or advanced tuberculosis with pyo-pneumothorax, open operation is generally contra-indicated, and if performed is liable to result in a permanently open pneumothorax. It is preferable to remove fluid from time to time by aspiration, with or without air replacement until thoracoplasty can be considered. Pleural wash-outs with mild antiseptics, such as weak methylene blue, flavine, or eusol, are often useful. This method of aspiration sometimes seems to assist the lung to re-expand. Surgical methods sometimes employed are intercostal tube drainage with slight suction, water-sealed drainage, or thoracoplasty in several stages.

HYDATID DISEASE OF THE PLEURA

Hydatid cysts may be primary in the pleura, or may encroach on the pleura, although originating in adjacent structures such as the lung, liver, spleen or mediastinum (parapleural hydatid).

Ætiology and Pathology.—Primary pleural hydatid is rare, but secondary invasion of the pleura is more common. In this situation the cyst may reach a large size, even 5 or 6 inches in diameter, before rupture occurs. As in other situations, a fibrous capsule is developed around the cyst from the irritative changes set up in the adjacent tissue. Contrary to what might be expected, extensive pleurisy is uncommon until rupture or suppuration of the cyst occurs. The pressure of the cyst may lead to collapse of the contiguous areas of lung and to displacement of the heart and mediastinum.

Symptoms.—These may be absent until the cyst is large enough to produce pressure symptoms, such as dyspnoea, pain and cough. There is little or no expectoration, unless rupture into a bronchus occurs, when cyst wall, daughter cysts and hooklets may be found in it. There is no fever until suppuration occurs. The signs are practically identical with those of encysted pleural effusion.

Complications and Sequelæ.—Rupture and suppuration are the two most important complications. Rupture may take place into the lung, into the pleural cavity, rarely through the chest-wall or through the diaphragm. At the time of rupture an urticarial rash may develop. This is probably an anaphylactic phenomenon associated with the liberation of toxin present in the fluid of the cyst.

Course.—The cyst may be latent for some time, but it usually enlarges and produces increasing symptoms, culminating in rupture or suppuration. Very rarely death of the cyst occurs and its contents become inspissated.

Diagnosis.—The symptoms and signs generally suggest either pleural effusion or new growth, and hydatid disease may not be suspected. Obscure basic signs, in patients coming from countries where hydatid disease is common, should suggest special methods of investigation as to the possibility of its presence. Should it be suspected, aspiration is to be deprecated, unless all preparations for immediate operation are complete, if the diagnosis is confirmed. These methods comprise X-ray examination, an eosinophil blood count, the complement-fixation test, the Casoni intra-dermic test and the precipitin reaction (see page 315).

Prognosis.—If untreated until rupture occurs, a fatal result is most probable. If diagnosed and treated before rupture, the prognosis is not unfavourable.

Treatment.—The former practice of aspiration and injection with formalin or iodine, although sometimes successful, is dangerous and should be discarded. Exposure of the cyst by thoracotomy, and its removal entire, should be the treatment if practicable, or if too large, it may be aspirated and then dissected out.

STREPTOTRICHOSIS (ACTINOMYCOSIS) OF THE PLEURA

The general characters of infection by the streptothrix group of organisms, and the special features of the pulmonary localisations have been described. It is possible, although improbable, that the infection may be primarily pleural, more commonly clinical manifestations may point to a predominating involvement of the pleura, although the primary lesions may be in adjacent structures, such as the lungs, mediastinum or liver.

Symptoms.—The symptoms and signs in such cases are those of empyema, but the following points are noteworthy. The empyema is rarely large, and it commonly extends through the chest-wall, producing a local swelling which soon discharges through the skin if untreated, causing a suggestive infiltration and puckering around. Exploratory puncture of a streptothric empyema often fails, since the grumous caseous material it contains may be too thick to pass through the needle.

Diagnosis.—The characteristic "sulphur granules" in the pus may draw attention to the real nature of the condition, but they are not always present. Direct films should always be made from the pus obtained from empyemata. The streptothrix may be found in this way, when culture fails. If the lung is involved as well as the pleura, the organism may be found in the expectora-

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tion, and the nature of the pleural condition may thus be established before operation.

Prognosis.—Some cases respond to treatment, but prognosis is in general unfavourable, death resulting from exhaustion or toxæmia due to dissemination of the disease.

Treatment.—The pultaceous pleural contents should be removed as far as possible by operation, and large doses of potassium iodide given by the mouth, increasing the quantity until 60 to 90 grains, three times a day, are given. An autogenous vaccine may be tried if the organism can be grown. A radium pack has sometimes given successful results.

SIMPLE TUMOURS OF THE PLEURA

These are very rare and are, as a rule, only discovered after death. They are almost invariably of extrapleural origin and their presence in the pleura is due to the direction taken by the growth. Lipoma of the subpleural or of the mediastinal fat may occur as small pedunculated tumours or very rarely as a large mass. They can be differentiated from tumours of the lung by X-ray examination after a diagnostic pneumothorax.

MALIGNANT TUMOURS OF THE PLEURA

Primary malignant disease of the pleura is rare, and may take the form of endothelioma, carcinoma or sarcoma. Secondary carcinoma and sarcoma are more common.

Ætiology.—Primary endothelioma of the pleura is more common in late adult life and in the male sex. Sarcoma is more likely to occur in children and in young adults. Secondary growths may occur at any age, but more commonly in later life.

Pathology.—Endothelioma of the pleura is a growth of obscure origin. It has not been conclusively established that it is derived from the pleural endothelial cells, and by some writers it is classed as a carcinoma. It is at first unilateral, but it involves the affected pleura over a wide area, sometimes universally. The membrane appears to be overlaid with an irregular, rough hard covering, sometimes nodular. In other cases there is more thickening and the condition may be localised. There is nearly always a large amount of blood-stained serous effusion. The condition may spread to the bronchial or supraclavicular glands, the lung, the spine, the diaphragm and the peritoneum.

Primary carcinoma of the pleura has also been described, but is very rare. Primary sarcoma is also extremely uncommon, but the round-celled and spindle-celled varieties may occur, and angio-sarcoma, fibro-sarcoma, myxo-sarcoma and chondro-sarcoma have all been recorded.

Secondary carcinoma and sarcoma of the pleura are relatively common, and may occur from direct *extension* in growths of the lung, bronchi and mediastinum, by *metastases* of growths in almost any distant part, or by *lymphatic permeation* in mammary carcinoma. In the last-named condition

pleural and pulmonary growths are a not infrequent form of recurrence, sometimes occurring months or years after removal of the primary growth.

Symptoms.—These are not characteristic, and increasing dyspnoea due to an accumulation of fluid may be the first indication. More commonly pain and cough, similar to those of pleurisy, may occur acutely or develop more gradually. Although afebrile as a rule, the occurrence of fever does not exclude malignant disease. Cachexia and wasting are often not marked until the condition is advanced. The signs are generally indistinguishable from those of ordinary pleural effusion, unless secondary growths become manifest in the cervical or axillary glands. Sometimes coarse dry friction may be heard, or there may be signs of pleural thickening without fluid. There is often local pain and tenderness over the chest. Exploratory puncture may demonstrate the hæmorrhagic character of the effusion. The specific gravity is generally 1018 or over, and the cytology of the fluid may be suggestive, especially if excess of endothelial cells, often aggregated into plaques, is found.

Complications.—The growth may spread to the lung and cause cough and expectoration, often blood-stained, or it may involve the chest-wall. Metastases sometimes develop along the course of the needle track after aspiration of the fluid. The secondary growths, especially those in the glands, may exert pressure, *e.g.* the axillary glands may cause œdema and swelling of the arm.

Course.—This is almost invariably progressive, the duration being rarely more than 2 years, and occasionally much less.

Diagnosis.—A chronic pleural effusion in a middle-aged man, not associated with fever, and not due to tuberculosis, should arouse suspicion of malignant disease of the lung and pleura. Evidence of fluid in one pleura, at an interval after excision of the breast for malignant disease, is very suggestive of secondary pleural growth. A hæmorrhagic effusion, not due to tuberculosis or renal disease, should also arouse suspicion of malignancy, especially if reaccumulation after tapping is rapid, and if the subsequent tapplings show increasingly hæmorrhagic characters. When aspiration of a considerable quantity of fluid gives little relief to symptoms, or when irregular dull areas remain where resonance might be expected, the probability of growth must be borne in mind. Growth involving the chest-wall, or the presence of cervical or axillary glandular metastases render it certain. Radiological examination after removal of some of the fluid may show characteristic plaques on the pleura.

Prognosis.—Malignant growth of the pleura is invariably fatal unless removal is possible.

Treatment.—From the nature of the condition this can only be palliative. Analgesic drugs may be given freely for the relief of pain, morphine being reserved for the severe forms and later stages, as far as possible. Repeated tapplings may be almost compulsory, if there is much distress from the reaccumulation of the fluid, but it must be remembered that in hæmorrhagic effusions the loss of blood by this means is considerable. Air replacement may sometimes give relief for a longer period than simple aspiration. In rare cases, removal by operation may be practicable if the diagnosis is made early and the growth is localised in an accessible position.

INJURY

Injury to the pleura may occur in fracture of the ribs, the fragments piercing or tearing it. Similarly in penetrating wounds of the chest, the pleura may be extensively lacerated. It may also be torn by direct violence without breaking of the ribs, and in rare cases a hernial protrusion of lung may occur, forming a small swelling in an intercostal space, protruding with inspiration and emptying with expiration.

R. A. YOUNG.

G. E. BEAUMONT.

DISEASES OF THE DIAPHRAGM

SPASM OF THE DIAPHRAGM

Diaphragmatic spasm may be either clonic or tonic, the former being termed *hiccough*.

Clonic spasm.—This may be due to a variety of causes, namely: (a) *Alimentary*: From irritation of the œsophagus or stomach by pungent or irritant substances such as pepper, pickles, or tobacco. It occurs also as a symptom in gastritis, dilatation of the stomach, enteritis, intestinal obstruction, tympanites and peritonitis, and in the late stages of debilitating disease. (b) *Nervous*: as in hysteria, cerebral tumour, meningitis, hydrocephalus, epilepsy and alcoholism. It may also result from peripheral nerve irritation, in such conditions as mediastinal tumour, mediastinitis, enlarged thoracic glands, diaphragmatic pleurisy, or pericardial effusion. *Epidemic hiccough* has been regarded as a form of encephalitis lethargica. There is usually some slight pyrexia, and the condition may persist without intermission for several days. (c) *Renal*: As in chronic nephritis and uræmia.

Tonic spasm.—This may be met with in tetanus, strychnine poisoning, laryngismus stridulus, eclampsia, epilepsy and hydrophobia. If there is associated intercostal or laryngeal spasm, there is grave risk of death from asphyxia.

Treatment.—Simple hiccough may often be relieved by holding the breath, pressure on the chest, or by simple inhalations, such as of ammonia, ether, or spirits of chloroform. Hiccough due to organic disease or to peripheral irritation may only be relieved by removal of the cause. In epidemic hiccough, in obstinate cases of hiccough due to other causes and in the tonic form of spasm, various antispasmodic measures may be tried, such as trinitrin, bromides, or phenobarbitone, by the mouth; adrenaline, or adrenaline and pituitary (posterior lobe) extract, hypodermically; or the inhalation of chloroform.

DIAPHRAGMATIC PLEURISY

This condition is described on page 1247 under the heading of Pleurisy.

PARALYSIS OF THE DIAPHRAGM

Definition.—Paralysis and inactivity of either leaf of the diaphragm, or of both.

Ætiology.—Paralysis of the diaphragm may be caused by disease damaging the centre in the spinal cord, by conditions affecting the phrenic nerve in its course, or by reflex inhibition of the centre. Causes involving the centre include poliomyelitis, hæmorrhage into the spinal cord, and tumours of the spinal cord or its membranes, or of the spine itself. The phrenic nerves may be affected by diphtheritic neuritis. Either or both of the nerves may be compressed by mediastinal tumours, or by inflammatory exudates. They may be severed or injured by wounds in the neck. Evulsion or crushing of the phrenic nerve is now frequently employed therapeutically, in order to promote collapse of the base of one lung and closure of cavities in cases of tuberculosis and in bronchiectasis.

Symptoms.—Diaphragmatic paralysis results in the affected leaf of the diaphragm becoming immobile and remaining at a higher level in the thorax than normal, or showing paradoxical movement, *i.e.* ascending with inspiration. This can easily be seen on X-ray examination. Sometimes this is noted as a reversal of the ordinary abdominal movements during respiration, with the result that there is epigastric recession during inspiration.

Treatment.—This is, in general, that of the condition causing the paralysis.

HERNIA OF THE DIAPHRAGM (see p. 553, 554)

EVENTRATION OF THE DIAPHRAGM (see p. 666)

DISEASES OF THE MEDIASTINUM

The mediastinum is the interpleural space, and occupies the median part of the thorax, from the superior aperture above to the diaphragm below. Strictly speaking, any affection of any of the important structures occupying this space, such as the pericardium, heart, great vessels, air passages and the thymus, might be included under this heading. They are, however, more conveniently grouped under the various systems to which they belong, and diseases of the mediastinum are commonly restricted to conditions arising in, or affecting the connective tissue and glands found in this space.

MEDIASTINITIS

Mediastinitis, or inflammation in the mediastinal connective tissue, may be acute or chronic. In the acute forms there may be an inflammatory serous exudate causing œdema, or the inflammation may progress to abscess formation. The chronic forms are indurative or fibroid in character, although chronic abscess may occur.

ACUTE SIMPLE MEDIASTITIS

Ætiology.—Acute mediastinitis without suppuration may result from injuries to the chest-wall or sternum, and from lacerating wounds of the œsophagus or trachea. It is sometimes secondary to inflammatory processes in the lungs, pleuræ, pericardium or peritoneum, and to periostitis of the sternum or vertebræ. Pneumonia is a not uncommon cause.

Pathology.—There is hyperæmia of the mediastinal connective tissue with inflammatory œdema. Mediastinal serous effusions have been described, but these are, without doubt, encysted pleural effusions encroaching on the mediastinum.

Symptoms.—The clinical manifestations of acute mediastinitis are vague and not characteristic. There is a mild pyrexia, the temperature reaching 99° or 100° F. Pain under the sternum may be complained of, and on auscultation over it a few fine crepitations may be heard on deep breathing, or they may occur synchronously with the heart beats.

Course.—The affection may subside or proceed to abscess formation. It may result in fibroid thickening or adhesions.

Diagnosis.—Mediastinitis is often not recognised or suspected, since it is masked or overshadowed by the clinical manifestations of the primary condition.

Treatment.—No special treatment is required, apart from that appropriate to the condition inducing it.

ACUTE SUPPURATIVE MEDIASTITIS

Ætiology.—Acute suppurative mediastinitis or mediastinal abscess is more common in males, and may occur at any age, although it is more frequently seen in early adult life than at other periods. Some cases are of traumatic origin, and follow perforating wounds or blows on the sternum, not necessarily causing fracture. Perforation or injury of the œsophagus is a comparatively frequent mode of access of pyogenic organisms to the mediastinum. This may occur from ulceration of an œsophageal new-growth, from injury due to a swallowed body such as a tooth-plate, or from the passage of an œsophagoscope or bougie. Perforation of the trachea or main bronchi by an inhaled foreign body is sometimes the cause of mediastinal suppuration. Various pulmonary conditions may lead to pyogenic infection of the mediastinum, such as pulmonary abscess or gangrene, pneumonia and bronchiectasis. Periostitis or osteomyelitis of the sternum, vertebræ or ribs, suppuration in the mediastinal glands, or tracking down of deep cervical abscesses may all lead to mediastinal abscess. Extensions of pyogenic processes from the pericardium, pleura or peritoneum may also be causes. A suppurating hydatid or dermoid cyst may rupture into the mediastinum, and, lastly, the infection is blood-borne in some cases from infective endocarditis, pyæmia, erysipelas or enteric fever. Dieulafoy pointed out that certain cases of empyema, originating near the mediastinum, may, by encroaching on this region, induce predominating mediastinal symptoms, which he described as the "mediastinal syndrome." Such cases, although abscesses in the mediastinum, are not mediastinal abscesses, but are in reality special instances of encysted empyema.

Pathology.—The suppuration may be limited to any part of the anatomical subdivisions of the mediastinum, or may spread from one compartment to another. The pus sometimes tracks in various directions, *e.g.* upwards to the neck, downwards to the abdomen, or it may point in the chest-wall. The abscess may rupture into the œsophagus, trachea, aorta, pleura or pericardium.

Symptoms.—The onset may be insidious or acute. In the latter case it may be ushered in by severe pain under the sternum, radiating to the back and shoulders. The symptoms may be divided into those due to the inflammatory process, and those resulting from the pressure exerted by the collection of pus. The former comprise malaise, fever and sometimes rigors, while blood examination may demonstrate a leucocytosis of 10,000 per c.mm. or over. The pressure symptoms vary according to the amount of pus produced and its situation. They include dyspnœa and paroxysmal or brassy cough, from compression of the vagus nerve or direct pressure on the trachea. There may also be dysphagia from obstruction of the œsophagus, and hoarseness from pressure on the left recurrent laryngeal nerve. Pressure on the spinal nerve roots, intercostal nerves, or brachial plexus may lead to severe neuralgic pains. Partial or complete obstruction of the great veins may be apparent from distension of the superficial thoracic veins or of those in the neck. Œdema of the chest-wall is sometimes seen from this cause, or it may result from the inflammatory process extending to the chest-wall. The signs in severe cases will be those caused by the pressure effects just described. The patient looks ill, distressed, dyspnœic and more or less cyanosed. The respirations may be noisy, as there is sometimes inspiratory dyspnœa with stridor, this being known as the *bruit de cornage*. The dilated veins may be apparent and the direction of the current may help to localise the seat of the obstruction. There is sometimes local redness and œdema from pointing of the abscess, near the sternum, in the neck, or in the interscapular region on either side. Palpation may reveal local tenderness and even fluctuation in any of these areas. There is often dullness over the sternum, sometimes extending to one or other side, or the dullness may be found in the interscapular region. It is said that the dullness may shift with the position of the patient in some cases. Breath sounds are distant, and weak or bronchial over the dull area, except when it is behind the sternum, when they are harsh.

Complications and Sequelæ.—The important complications are those due to rupture of the abscess. If this occurs into the lung or the œsophagus, pus is expectorated, or passes into the stomach. Gangrene of the mediastinum may follow, or death may occur from suffocation or hæmorrhage. Extension of the abscess may lead to purulent pleurisy, pericarditis or peritonitis, or to suppuration in the neck. In cases that recover, chronic mediastinitis with matting together of the mediastinal contents may be a sequel.

Course.—The disease is acute and rapidly progressive, unless relieved by operation or by spontaneous external drainage in a few fortunate cases.

Diagnosis.—The “mediastinal syndrome” of dyspnœa, stridor, paroxysmal cough, hoarseness and dysphagia with signs of pressure on arteries, veins and nerves is common to many conditions causing mediastinal pressure, notably mediastinal new-growth, enlarged mediastinal glands, aneurysm and

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pericardial effusion. The differential diagnosis of these is more fully considered under mediastinal new-growth. The occurrence of fevers and rigors, the presence of a pointing swelling, and the demonstration of a leucocytosis may give strong suggestion as to the inflammatory origin of these symptoms and signs. The X-rays may reveal a localised mediastinal shadow, often non-pulsating, although it must be remembered that in rare cases a mediastinal abscess may pulsate.

Prognosis.—This is very grave, and the majority of cases die unless recognised and treated early. If gangrene develops, a fatal result is inevitable. The outlook is more hopeful when the anterior mediastinum alone is involved.

Treatment.—**PROPHYLACTIC.**—Foreign bodies in the œsophagus and trachea should be removed as soon and as gently as possible. The utmost care should be exercised in the passage of a bougie or the œsophagoscope in cases of œsophageal stricture.

CURATIVE.—As soon as mediastinal suppuration has been diagnosed and localised, surgical measures should be adopted. The mediastinum can be reached by resection of pieces of costal cartilage or by trephining the sternum.

CHRONIC MEDIASTITIS

This also occurs in two forms, chronic indurative mediastinitis and chronic abscess.

CHRONIC INDURATIVE MEDIASTITIS.—This may occur as a sequel of any form of acute mediastinitis. The best known is that associated with chronic adhesive pericarditis, and usually known as chronic indurative mediastino-pericarditis (pp. 961-963). Other forms include the chronic inflammation and thickening which occur around enlarged, sclerotic and pigmented mediastinal glands, and around the same glands when affected by caseous or calcareous tuberculous lesions.

CHRONIC MEDIASTINAL ABSCESS is generally of tuberculous origin, arising from breaking down caseous bronchial or mediastinal glands, or from tuberculous disease of the spine or ribs. A chronic abscess may, however, be caused by a foreign body, such as a bullet.

Symptoms.—Simple indurative mediastinitis may give rise to practically no symptoms or signs. Chronic abscess may cause symptoms of ill-health and of mediastinal pressure, or may only become apparent when it points superficially.

Treatment.—The treatment of chronic mediastinal abscess is practically the same as that for other "cold" abscesses due to tuberculosis, incision and drainage being avoided if possible in favour of aspiration and injection of anti-tuberculous substances. Other cases may require operation.

EMPHYSEMA OF THE MEDIASTINUM

In performing tracheotomy, the pretracheal layer of deep cervical fascia is of necessity incised. If difficulty arises in inserting the tube into the tracheal incision, air may be drawn deep to this fascia by the vigorous attempts at respiration and thus pass into the superior mediastinum, or superficial to

it into the anterior mediastinum. Rupture of the trachea, bronchi or œsophagus, or rupture of air vesicles or pulmonary lesions where the pleura is adherent, may also cause it. In acute interstitial emphysema of the lungs, the escaped air may track along to the root and reach the mediastinum.

Symptoms.—Emphysema of the mediastinum may give rise to very indefinite indications. A few fine crackling sounds may be heard on listening over the sternum, sometimes varying with respiration or with the heart movements. The percussion note over the præcordium may be hyper-resonant, and the heart sounds may be distant and muffled. Small quantities of air escaped into the mediastinum can be rapidly absorbed and may not be of serious import.

Diagnosis.—This is often a matter of speculation, unless the air spreads upwards to the neck and causes superficial surgical emphysema.

Prognosis.—This depends entirely on that of the underlying cause, which is often of serious nature.

Treatment.—No special treatment is required, as a rule, apart from that of the primary condition, except that pain may necessitate the use of analgesic drugs at the onset.

ENLARGED MEDIASTINAL GLANDS

The mediastinal lymphatic glands are arranged in groups. A few small ones are found in the anterior compartment, another group is situated in the posterior mediastinum. The most important of these is the tracheo-bronchial group, situated around the bifurcation of the trachea and extending along the bronchi. It is enlargement of this group that most often gives clinical manifestations.

Ætiology and Pathology.—A simple inflammatory enlargement of these glands may occur in many acute affections of the bronchi and lungs, and in certain acute specific fevers, notably influenza, pertussis and measles. A more chronic enlargement, associated with indurative changes, results from chronic respiratory diseases, such as chronic bronchitis and the pneumoconioses. In the latter case, considerable pigmentary changes may be found, from deposition of the particles derived from the dusty inspired air. In town-dwellers, these glands are often grey or black in colour from deposited carbon. Tuberculosis is the commonest cause of enlargement of the mediastinal glands, particularly of the tracheo-bronchial group, those about the right bronchus being most affected as a rule. This is a frequent early localisation of tuberculous disease in children. The infection spreads from the lungs in the majority of cases (Ghon), but in some instances the path of infection is from the tonsils through the cervical lymphatics and glands, while in others the mode of entrance is from the intestines through the mesenteric glands. The lesions may be miliary tubercles, or small caseous nodules which calcify subsequently, or which may soften and lead to local spread or generalisation. In other cases a fibroid hyperplasia of the glands results.

In syphilis, mediastinal adenitis may occur in the secondary or tertiary stages. In Hodgkin's disease and in lymphatic leukæmia, the mediastinal glands may share in the general adenopathy, and in the former the condition

may be primary in these glands. Enlargement due to malignant disease is of great importance and receives separate consideration.

Symptoms.—These may be slight and escape notice, unless the enlargement is sufficient to produce pressure or irritation. Cough is the commonest symptom; it is usually dry, irritative, noisy and ineffective. It may occur in paroxysms, somewhat suggestive of those of whooping-cough. Dyspnoea and dysphagia occur only when the enlargement is considerable. Vomiting sometimes develops, probably reflexly from vagal stimulation. Pain behind the sternum or in the upper thoracic region posteriorly may be complained of. In children with tuberculous disease in these glands, there is often languor, anorexia, anæmia and wasting, sometimes with slight irregular fever and night sweats. Such symptoms in a child of 5 to 12 years of age are very suggestive. The signs are also variable and frequently inconclusive. In tuberculous cases, the appearance of the child, pale, delicate looking or sallow, with long eyelashes and fine hairy growth over the back, may also be suggestive. In glandular enlargement from any cause, there may be dilated veins over the front or back of the chest, especially in the upper part, and a "hilum dimple" has been described as appearing in the second intercostal space beside the sternum, on holding the breath at the end of inspiration. One pupil may be larger than the other, owing to sympathetic stimulation. Small areas of dullness may be found at the back, near the upper thoracic spines, or in front close to the manubrium. Breath sounds over these areas may be bronchial or harsh. Occasionally the enlarged glands impede the air entry to a lower lobe, generally the right, in which case breath sounds are notably weakened over this area, while the percussion note may be impaired. Normally, whispering pectoriloquy ceases at the seventh cervical spine; with enlarged mediastinal glands it may be heard along the middle line or close beside it, in the upper thoracic region from the first to the fifth thoracic spines. This is known as d'Espine's sign or tracheophony. It is a confirmatory sign, when other indications are present. Eustace Smith's sign is of little value. It consists in a venous hum, audible over the manubrium sterni, when the child's head is thrown back as far as possible. Occasionally pressure on the recurrent laryngeal nerve may lead to an abductor paralysis of one vocal cord. In cases of tuberculosis, syphilis, Hodgkin's disease or leukæmia, enlarged glands may be present in other parts of the body, and may thus assist in diagnosis.

Complications.—A caseous gland may ulcerate into a bronchus or into the trachea, and death has resulted from glottic impaction of a portion of the gland. Ulceration into the œsophagus has been described. Rupture into the mediastinum may lead to mediastinal abscess. Invasion of the pleura, lung or pericardium may occur, or generalisation causing widespread miliary tuberculosis.

Diagnosis.—Whenever the condition of mediastinal glandular enlargement is suspected, an X-ray examination should be made if possible. It may help to distinguish between other conditions causing mediastinal pressure, such as aneurysm, abscess and malignant growth. Unfortunately in regard to tuberculous disease, it shows best the condition of least importance, namely, the old healed calcified glands. "Soft" or "woolly" shadows are regarded as indicative of active disease, but in doubtful cases it is wise to act upon the clinical indications.

Prognosis.—This varies with the cause, being serious in Hodgkin's disease and leukaemia. In tuberculous cases, the prognosis is as a rule good, apart from complications, provided treatment is prompt and adequate.

Treatment.—In tuberculous adenitis, the general condition should be improved by every possible means. The child should be taken from school, rest and exercise are to be carefully graduated, and a liberal diet supplied, with extra milk, cream and butter. In England, the Isle of Thanet seems especially valuable in the climatic treatment of glandular tuberculosis. Cod-liver oil, malt extracts and the syrup of the iodide or phosphate of iron are useful. In afebrile cases, tuberculin cautiously given may be of value in children of 8 years or over, but it is not necessary, as a rule. If given, the initial dose should be small, $\frac{1}{1000}$ mgrm. B.E., and the dosage gradually increased. In glandular enlargements due to syphilis, Hodgkin's disease and leukaemia, the treatment appropriate to these diseases should be employed, and symptoms due to pressure relieved as far as possible.

MEDIASTINAL TUMOURS OR NEW-GROWTHS

The mediastinum may be the seat of either simple or malignant new growths, the latter being much more common.

SIMPLE TUMOURS OF THE MEDIASTINUM.—These, except retrosternal goitre, rarely give rise to symptoms, and the recorded cases have, as a rule, only been discovered in the course of a routine X-ray or post-mortem examination. The chief varieties found are retrosternal goitre and persistent thymus, lipoma, fibroma, chondroma, osteo-chondroma and myoma.

MALIGNANT TUMOURS OF THE MEDIASTINUM.—Although it is certain that some malignant growths arise primarily in the mediastinal tissues, while others invade the mediastinum secondarily by extension or metastasis, it is often impossible, even at autopsy, to determine whether a mediastinal growth originated in the mediastinal tissues or in one of the adjacent organs, particularly the lungs and bronchi. The differentiation between primary and secondary growths is therefore less sharp than in other situations.

SARCOMA OF THE MEDIASTINUM.—Recent research has proved that the majority of primary mediastinal growths are sarcomatous, but these are less common than was formerly supposed. A primary sarcoma may arise in the lymphatic glands, connective tissue, periosteum of the sternum or vertebræ, or in the remains of the thymus gland. The commonest variety is the lympho-sarcoma, but spindle-celled and chondro-sarcomata may occur. Mediastinal sarcoma is commoner in males than females; it may occur in early life, and the majority of cases occur before the age of 40 years. Oat-celled tumours invading the mediastinum and formerly regarded as lympho-sarcomata are now believed to be of bronchial origin and carcinomatous nature.

CARCINOMA OF THE MEDIASTINUM.—This is rare as a primary tumour. It occurs in older people. It may originate from the trachea, bronchi or œsophagus, in the remains of the thymus or in a retrosternal goitre.

SECONDARY MALIGNANT GROWTHS OF THE MEDIASTINUM.—These usually result from direct extension of primary growths of the lung, bronchi, trachea, œsophagus, chest-wall or breast, but true metastases may occur from

mammary growths or from more distant primary tumours. Endothelioma has been described in the mediastinum, but is probably generally secondary to endothelioma of the pleura.

Pathology.—The morbid appearance depends upon the situation of origin, the directions of growth, and the nature of the tumour. Sarcomata are generally soft, pinkish in colour and vascular, while carcinomata are paler and firmer. There may be one large mass weighing several pounds, or there may be multiple growths. When the tumour reaches a considerable size it may infiltrate, surround, compress or displace contiguous structures. This is particularly the case in the lympho-sarcomata. The trachea, œsophagus, and large vessels may be surrounded, the pericardium and heart may be extensively infiltrated, and the nerve trunks may be enclosed and compressed. Secondary deposits are common in other glands, but not infrequently the pigmented bronchial glands may be seen entirely enclosed in growth without being infiltrated.

Symptoms.—The onset is often insidious, and the condition may not be suspected until cachexia and pressure signs develop. Malaise, weakness, shortness of breath, cough and pain are often early symptoms, which become more pronounced as the case progresses. The pressure symptoms and signs constituting the “mediastinal syndrome” comprise—

1. *Pressure on the air passages*, giving rise to dyspnoea, cough and expectoration. The dyspnoea may be inspiratory and associated with stridor, or expiratory and paroxysmal. The cough is harsh and may be “brassy”; it is often associated with mucoid, blood-stained, or even “prune juice” sputum. Bronchiectasis may result in some cases.

2. *Pressure on or infiltration of the lung*, leading to collapse and sometimes breaking down of lung tissue. If the pleura is reached or invaded, pleural effusion, often blood-stained, may result.

3. *Pressure on arteries*.—Compression of branches of the pulmonary artery may lead to local gangrene, or in other cases the growth may ulcerate into a larger vessel and cause fatal hæmorrhage. Pressure on the subclavian artery may cause inequality of the radial pulses, and, according to Ekgren, this may only be present when the patient is lying and not when he is standing.

4. *Pressure on veins*.—Dilated tortuous veins may be seen over the front of the chest and abdomen, or in the neck. The flow of blood in these superficial veins may be reversed in direction, owing to the obstruction of the superior vena cava or its main radicles. The current then runs from above downwards, instead of from below upwards, as normally. There may be œdema of the chest-wall or of the face and neck from the same cause.

5. *Pressure on nerves*.—The vagus may be compressed, causing paroxysmal dyspnoea and cough. Laryngeal paralysis or spasm may result from involvement of the recurrent laryngeal nerve. Dilatation of the pupil, followed later by constriction, drooping of the upper lid and enophthalmos, occurs when the sympathetic is involved. Paralysis of the diaphragm on one side from compression of the phrenic nerve, and pain from involvement of the intercostal nerves, may be present.

6. *Pressure on the œsophagus* may lead to dysphagia of increasing degree.

In addition to the signs afforded by these various conditions, there may be glandular enlargements in the neck, the suprasternal notch, or in the axillæ.

The growth may invade the chest-wall at any spot, and in rare cases it may cause visible or palpable pulsation. The pulmonary physical signs are dyspnoea, sometimes orthopnoea and cyanosis. In some instances the patient prefers to lean forward; this is said to be due to the fact that in this position the antero-posterior diameter of the mediastinum is increased, and the tension caused by the growth is thereby lessened. There may be dullness over the sternum or over the upper thoracic spines, and over any part of the lung invaded or compressed by the growth. The breath sounds heard over the dull area may be harsh, bronchial, tubular, weak or absent. The signs due to any secondary condition, such as bronchitis, bronchiectasis or a pleural effusion may be found in addition.

Complications.—These include the secondary conditions just mentioned. Others are due to ulceration of the growth through the chest-wall, or into the trachea, bronchi, œsophagus or aorta. Pericarditis may occur if the growth invades the pericardium, and hæmopericardium may result from ulceration of a vessel.

Course.—The growth enlarges progressively and the course is often rapid, particularly in lympho-sarcoma. Fulminating cases lasting only a few weeks occur; more commonly the patients live from 6 months to 2 years from the onset, rarely more.

Diagnosis.—When signs of mediastinal pressure become apparent, new-growth should be suspected, in common with aneurysm, mediastinal abscess or cyst, enlarged mediastinal glands and pericardial effusion. The history, the general condition of the patient, the physical signs, blood examination, and the X-rays may all help in distinguishing between these conditions. The evidence afforded by the X-ray may be of the utmost value. The pulsating shadow of an aneurysm, the large area of a pericardial effusion, the indefinite edge of an infiltrating growth extending into the lung, may be shown clearly, but the appearance should always be interpreted in the light of the other clinical features, and a diagnosis should not be made on X-ray findings alone, since a growth may pulsate, or may give rise to an effusion, while a mediastinal abscess or a cyst may give a sharp shadow. An œsophageal new-growth can sometimes be differentiated by the œsophagoscope, but this should only be employed when aneurysm can be excluded. Diagnosis from pulmonary or bronchial new-growths may be almost impossible. Before the onset of pressure symptoms, growth may be suspected from the cough and emaciation, and here again the X-rays may give valuable indications. Chronic tuberculous disease should always be excluded by repeated sputum examinations. The diagnosis of mediastinal growth may sometimes be obscured by some of the complications it induces, notably pleural effusion and bronchiectasis. The rapid onset and progress of these conditions and the bloodstained character of an effusion may all suggest the possibility of a malignant cause. The presence of enlarged glands in the neck or axillæ, or of nodular growth in the chest-wall or episternal notch, may afford almost conclusive evidence of malignancy.

Prognosis.—This is practically hopeless and death occurs from exhaustion, starvation, toxæmia, asphyxia or hæmorrhage.

Treatment.—The treatment of simple tumours is surgical if they are capable of removal. The treatment of malignant tumours is that of inoperable malignant disease elsewhere. X-ray applications, or radium treatment in some

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form may be tried. Otherwise treatment is symptomatic and palliative. Pain may be relieved by aspirin, codeine or morphine. Sleep may be induced, if there is insomnia, by chloral hydrate, papaveretum (alupon, opoidine, omnopon) or other hypnotics. If effusion is causing dyspnoea it may be tapped, but the fluid usually collects again rapidly. Air replacement is sometimes useful.

CYSTS OF THE MEDIASTINUM

SIMPLE CYSTS.—These are usually small and of no clinical importance.

DERMOID CYSTS AND TERATOMATA.—These are rare and become apparent generally in young adult life. They may enlarge, giving rise to symptoms and signs similar to those of a mediastinal tumour, or they may lead to empyema. They usually contain pultaceous material, and sometimes hairs, muscle, cartilage, bone and teeth. Such cases are almost certainly teratomatous in nature and derived from included embryos. This condition may sometimes be diagnosed during life by the expectoration of hair, teeth, bone or cartilage. The prognosis is, as a rule, serious, but some cases recover under appropriate surgical treatment.

HYDATID CYSTS.—A hydatid cyst may be primary in the mediastinum and may give rise to signs of mediastinal pressure, but the condition is extremely rare. Its presence may be shown by X-rays and its nature demonstrated by the blood and skin reactions. Such cysts have been successfully treated surgically.

Other rare mediastinal conditions are hernia of the stomach or colon through the diaphragm into the mediastinum. A retrosternal goitre may also form a mediastinal swelling.

R. A. YOUNG.

G. E. BEAUMONT.

SECTION XVI

DISEASES OF THE KIDNEYS

THE CHARACTERS OF NORMAL URINE

THE amount of urine normally secreted in 24 hours is 50 ounces or 1500 c.c. The specific gravity of the total should lie between 1015 and 1025, though individual specimens will vary considerably more, according to the amount of fluid imbibed or the quantity excreted by the skin and bowels. The reaction is generally acid, due to the presence of acid sodium phosphate, NaH_2PO_4 . The total acidity is such that about 650 c.c. of decinormal caustic soda will neutralise the daily output. This is equivalent to 82 grains of NaHCO_3 , but it requires a rather larger quantity (120 grains according to Spriggs) of bicarbonate of soda by the mouth to effect this neutralisation. Expressed in terms of H-ion concentration, the pH varies between 4.7 and 10. The total acidity of the urine rises very considerably in acidæmia, and may be more than doubled despite large doses of alkalis. Normally, urine is more acid during fasting than during absorption of food, the acid and alkaline tides being thus produced. The alkaline tide may be partly due to the absorption of organic vegetable salts, as it is more marked in herbivora, but the increased activity of the respiratory centre after getting up in the morning, is chiefly responsible for an increase in the pH of the urine (diminished H-ion concentration), by removal of excess of CO_2 from the body; in fact, it alone may be responsible for a morning alkaline tide. On decomposition, either in the bladder or after excretion, the urine becomes alkaline, from the conversion of urea into ammonium carbonate.

The constituents of urine are partly derived from the food (exogenous) and partly from the katabolism of the tissues (endogenous). We may briefly consider the source and significance of the principal constituents.

Nitrogenous constituents.—The total nitrogen excreted each day on an ordinary mixed diet is about 18 grammes or 270 grains. Of the various nitrogenous constituents urea is by far the most abundant, its output being 33 g., which contains 15.4 g. of nitrogen, or 85 per cent. of the urea total. As so much of the urea comes directly from the food, the amount of urea falls both absolutely and relatively in starvation; the total nitrogen drops to 5 g. or even less, of which urea nitrogen forms about 60 per cent. On a diet rich in carbohydrates and fat, but containing hardly any nitrogen, these figures may fall still lower, as the assimilation of other food-stuffs reduces the waste of tissue nitrogen to a minimum. This is often forgotten, and in nephritis undue importance is attached to a drop in the output of urea, which is simply due to the diet prescribed being poor in nitrogen, whereas the urea excreted depends mainly on the quantity of protein ingested.

Next in importance come the purin bodies. Purins contain the group C_5N_4 , and the best known is tri-oxy-purin or uric acid $C_5N_4H_4O_6$. A small quantity of the less oxidised purins, xanthin and hypoxanthin is also excreted. Exogenous purins come mainly from meat juices, from the nuclei of cellular organs (such as liver and sweetbread), and from tea, coffee or cocoa. Only from one-tenth to one-half of the ingested purins are excreted as such, the remainder being destroyed by the liver, ultimately appearing as urea. The alkaloids of tea, coffee and cocoa give rise chiefly to xanthin and hypoxanthin, the rest to uric acid. On a diet rich in meat the daily output of purins amounts to 0.34 g. of nitrogen, and on a purin-free diet to 0.202 g. This endogenous purin, which forms the larger part, comes mainly from the disintegration of the nuclei of maturing red blood corpuscles, and also the leucocytes and muscles. Anything increasing the leucocytes in the circulation increases the output of endogenous purins, and in leukæmia the excretion of uric acid may rise to 5 g. a day. Unaccustomed exercise diminishes the output of uric acid, while increasing that of the less oxidised xanthin and hypoxanthin, the total purin excretion remaining the same. Uric acid is only excreted as such when the urine is highly acid; normally it appears as acid sodium urate. (See also Urinary Deposits.)

Creatinin is, according to Folin, the most constant of the nitrogenous constituents on a meat-free diet, and serves as a measure of endogenous nitrogenous metabolism. In a healthy young man on a diet consisting entirely of bread, about 0.9 g. is excreted daily, while on a diet containing meat extracts more than 2 g. may be passed. During muscular wasting its output is increased, while in a subject already wasted it is diminished. All this suggests that it is derived from the creatin of muscle—both of the body and of the food. It may be recognised in urine by Weyl's test; with sodium nitroprusside and caustic soda it gives a ruby-red colour, which, unlike that given by acetone, is at once destroyed by glacial acetic acid. Jaffe's test depends upon the deep orange colour, given by even dilute solutions of creatinin on the addition of a saturated solution of picric acid and some 10 per cent. solution of caustic soda; this has been utilised for the colorimetric estimation of creatinin by Folin. Creatinin can be obtained from creatin by dilute boiling mineral acids. Creatin, which is abundantly present in muscles, is not normally present in the urine.

Ammonia is normally excreted to the extent of about 1 g. a day. An increase in this amount is not, as was formerly thought, a sign of incapacity on the part of the liver to form urea, but a sign of acidæmia. The body protects itself against acids in the circulation by forming ammonia from the proteins of the tissues. The increased excretion of ammonia is, therefore, a measure of the degree of acidæmia, and it is estimated by the amount of acid set free from the urine on the addition of formalin, which combines with the ammonia to form hexamine. In the acidæmia of diabetes, the output of ammonia may rise to 4 g. a day, or even more.

Hippuric acid is not an important nitrogenous constituent of urine, but it is of interest as being made by the kidney itself by the combination of benzoic acid with glycoooll. It is, therefore, increased by a diet of green vegetables, and is diminished when there is conspicuous degeneration of the renal tubules.

The pigments of urine are nitrogenous. The principal one, urochrome, to which urine normally owes its colour, though closely related to urobilin, has an independent origin from hæmoglobin. Even when all the bile escapes from the body through a biliary fistula the excretion of urochrome is unaltered. Urobilin, on the other hand, is a reduction product of bile pigment. The reduction is effected by bacterial action in the bowel, whence it is reabsorbed by the blood and excreted by the kidney. Normally it is not excreted as such, but as a colourless chromogen. The appearance of pre-formed urobilin is evidence either of increased hæmolysis or of septic infections of the gall-bladder or bile-ducts, or of increased intestinal putrefaction, or of increased time for reabsorption, as in intestinal obstruction. It can be recognised with the spectroscope by the absorption band it gives in the blue, or by the green fluorescence it shows on the addition of zinc chloride and ammonia. Very little is known of uroerythrin; it is an unstable body and is readily carried down by urates, to which it imparts the characteristic pink colour. A trace of hæmatoporphyrin is also normally present in the urine; but an obvious amount is an abnormality, which will be considered later.

Non-nitrogenous constituents.—These are principally salts. Chlorides are the most abundant, averaging about 10 to 13 g. of sodium chloride a day. Chlorides are retained whenever the body retains excess of fluid. This explains the reduced output of chlorides in such diverse conditions as cedema, serous exudates, pneumonia and acute dilatation of the stomach. Reduced chloride intake or loss through excessive vomiting are other causes. On the other hand the output is much increased in Addison's disease. The phosphates are partly excreted as acid phosphates of sodium and potassium, partly as earthy phosphates of calcium and magnesium. The former are not precipitated on neutralisation, while the latter are. A phosphatic deposit, as stellar crystals of calcium phosphate or tables of magnesium phosphate, is no proof of a real increase in the output of phosphates, but is usually merely an indication of diminished acidity. Ammonio-magnesium phosphate, on the other hand, is evidence of ammoniacal decomposition. It forms a deposit of "coffin-lid" or "knife-rest" crystals. The amount of phosphoric acid excreted daily amounts to about 2.5 to 3.5 g., of which the earthy phosphates form half. Sulphates are present in the urine to the extent of 1.5 to 3 g. of SO_3 a day. Very little sulphate is taken in the food, and most of that which is taken either as food or medicine is excreted by the bowel, so that the urinary sulphates come almost entirely from the oxidation of the sulphur in the protein molecule. About nine-tenths are excreted as sulphates of the alkalis, and the remaining one-tenth as ethereal sulphates, formed by conjugation with putrefactive products from the tyrosin and tryptophan of the protein molecule. Of these, the most striking is indican, or indoxyl-sulphate of potash. It is best detected by adding an equal quantity of strong hydrochloric acid to some urine, then a few drops of hydrogen peroxide, and shaking up the mixture with some chloroform to which it imparts a blue colour. Its presence in excessive amount is some evidence of excessive intestinal putrefaction, especially when due to obstruction of the small intestine. Not so much importance, however, is attached to indicanuria as formerly. All the sulphur in the urine is not excreted as sulphates; some 6 per cent. appears as neutral sulphur, derived from the

sulphocyanide of the saliva, the taurine of the bile salts and substances allied to cystin. The neutral sulphur is diminished in insanity.

Many other substances are normally present in traces in the urine, but except diastase, they are of little clinical importance. Ten to 30 units of diastase are normally present, but less will be found in some forms of impaired renal capacity and a great deal more in most pancreatic diseases. The presence of 50 units suggests a pancreatic lesion, while 100 or more make this certain. In severe pancreatitis 300 to 500 may be found.

THE ESTIMATION OF RENAL FUNCTION

It may be necessary to determine (1) the total renal capacity, or (2) the adequacy of either kidney separately. Generally speaking, the first is more the concern of the physician, and the second that of the surgeon. Estimation of the latter is of vital importance before nephrectomy is considered, lest the remaining kidney should prove inadequate to maintain life. Estimation of the former is an assistance both to diagnosis and prognosis. Some of the tests under the first heading have for their object the determination of the part of the kidney involved. These will be considered first.

A.—ESTIMATION OF CAPACITY OF BOTH KIDNEYS

1. **EXAMINATION OF THE BLOOD.**—The damaged kidney will fail to excrete substances which it should, and examination of the blood may reveal their presence in undue amount. The quantity of urea in the blood throws important light on renal capacity; normally this ranges from 15 to 40 mgm. per cent. in health, but after middle age figures up to 50 mgm. per cent. (urease method) may be within normal limits. The urea content of the blood, as well as that of the cerebro-spinal fluid, is raised in various kidney diseases, and also in alkalosis. A blood urea figure of 200 mgm. per cent. and over is of serious clinical significance. It may rise higher than this, even to 280 mgm. per cent., in acute nephritis, and gradually fall to normal with complete recovery. In chronic nephritis such figures generally indicate a terminal phase of few months' duration, but a patient may live for a year or more with a blood urea of 190 mgm. per cent. The amount of sodium chloride in the blood may be raised from the normal 0.45 to 0.5 g. per cent. to 0.6 or higher. When there is extreme renal failure there may be an increase in the H-ion concentration, the uric acid and the indican of the blood, while the calcium content may fall from the normal 10 mgm. per cent. to 6.

2. **THE UREA CONCENTRATION TEST.**—Although ordinary estimation of the percentage of urea in urine gives no information of value, the response of the kidney to a given dose of urea does. On this MacLean and de Wesselow based their useful urea concentration test. Fifteen grammes of urea dissolved in 100 c.c. of water, and flavoured with a little tincture of orange, are given to a patient just after he has emptied his bladder. The urea in the urine passed one, two and three hours afterwards is estimated by the hypobromite method. If this amounts to 2 per cent. or over in one or more of the three specimens the kidney is efficient according to the test. A concentration of 2.5 per cent. or over is more satisfactory. The volume of urine should not exceed 120 c.c. in the first hour, or 100 c.c. in each of the second and third hours. Excessive

diuresis may be due to release of water previously retained in the tissues, and the test should be repeated. This test is of less value if the patient is taking a low nitrogen diet.

3. **THE BLOOD UREA CLEARANCE TEST.**—This test was introduced by Möller, McIntosh and Van Slyke as a simple and reliable method of estimating the urea-excreting function of the kidneys. In principle it is based on the relation of the blood-urea concentration to the urea excretion in the urine. The result is expressed as cubic centimetres of blood cleared of urea per minute. For details a textbook of clinical pathology should be consulted. It is claimed that this test is more sensitive and will reveal minor defects not revealed by other tests.

4. **VOLHARD'S TESTS AS MODIFIED BY ROSENBERG.**—On the first day the patient, after passing urine, drinks 1500 c.c. of water within half an hour. Urine is passed at half-hourly intervals for the next 4 hours, each specimen being saved separately and tested for volume and specific gravity. Normally the whole 1500 c.c., often more, is excreted within the 4 hours, and the specific gravity falls to 1002, or less. The second day, ordinary meals are given, but the amount of fluid is limited to 500 c.c. for the whole 24 hours, taken in four roughly equal portions. Fruit should not be given, or should be reckoned as fluid. Urine is passed as and when the patient wishes, and each specimen is again collected separately and tested for volume and specific gravity. The total urine for the day should not exceed 750 c.c., and the specific gravity should rise to at least 1027. These are known as the dilution and concentration tests respectively.

In cases of renal insufficiency the volume of urine on the first day is too little, while that on the second day is too much, as excretion tends to continue at the same rate irrespective of variations in the requirements of the body. The limits of variation in specific gravity become more and more narrowed as the disease progresses, until at length a fixed point of about 1009 is reached. Often the dilution test will show a minimum of about 1005, and the concentration test a maximum of about 1015, long before the patient complains of any symptoms or the blood shows any evidence of uræmia.

In our opinion the concentration test is more reliable than the dilution test, but both of them set up too rigid a standard of what constitutes normal function.

5. **FIXATION OF SPECIFIC GRAVITY.**—This is a simpler method. The patient takes no fluid drinks or liquid foods or fruit from after breakfast one day until breakfast-time the next day. The urine passed in the first 12 hours need not be kept, but that secreted in the second 12 hours is collected and pooled. If the renal function is satisfactory the specific gravity of this urine should be at least 1024, and the concentration of urea should be more than 2 per cent.

B.—ESTIMATION OF CAPACITY OF EACH KIDNEY

Catheterisation of each ureter under the direct view of the cystoscope is the only reliable method of obtaining the required information. It is usual to encourage secretion during examination by giving some tea or simple diuretic. Additional information is gained by the intramuscular injection of 15 minims of a 5 per cent. aqueous sterilised solution of methylene-blue. It is first excreted as a colourless chromogen and, later, as methylene-blue itself.

The chromogen turns blue when boiled with acetic acid, and should appear in the urine in from 15 to 20 minutes, after which the excretion of unaltered methylene-blue should begin. It should reach its maximum in from 4 to 5 hours, and should have disappeared in from 40 to 50 hours. Obviously, catheterisation of the ureters cannot be continued all this time, so that observation is directed towards a marked delay in the appearance of blue on one side as compared with the other. Afterwards, hexamine should be given as a precautionary measure, and the patient kept in bed for 36 hours. As in acute or subacute nephritis the rate of methylene-blue excretion is entirely unaffected, the utility of this test is confined to unilateral chronic disease.

Indigo-carmin may be used for a similar purpose; 10 c.c. of a 0.4 per cent. solution is injected intravenously or intramuscularly. The urine should be coloured in about 10 minutes, first appearing green and then blue. Excretion reaches its maximum in about an hour, so that this test has advantages over the methylene-blue method. Delay in the appearance of the dye and a feeble staining of the urine may be taken as evidence of disease.

Pyelography is a valuable means of determining the position of the kidneys and their relation to shadows in or in the neighbourhood of the urinary tract. By this means the position of renal or ureteric calculi may be defined, and such shadows as those caused by calcareous tuberculous glands, gall-stones, and faecal calculi may be recognised as outside the urinary tract. It is an invaluable means of demonstrating the presence of a hydro-nephrosis, especially when small. It will show dilatation or irregularities in the course of the ureter. By the absence of the shadow caused by the dye, a failure in function of one kidney or its absence may be indicated. Renal growths and tumours of the renal pelvis may be diagnosed by abnormalities in the pyelogram, and calculi not evident in a plain radiogram may be shown by this means. The intravenous method is now commonly adopted in the first approach to a urinary case, but instrumental pyelography is often required to confirm the findings obtained.

A drug, opaque to X-rays, which is eliminated by the kidneys, is introduced intravenously, and radiograms are taken at short intervals after its injection. Uroselectan B (a non-toxic iodine-containing substance) is the best preparation for this purpose. For instrumental pyelography a 12 to 20 per cent. solution of sodium iodide or sodium bromide is used. Before iodine is given for the purpose of pyelography, the patient's tolerance of the drug should be tested by giving five or ten grains by mouth, in order to exclude an idiosyncrasy.

ABNORMALITIES OF THE URINARY SECRETION

1.—POLYURIA

Polyuria may be due to—

1. Increase in the quantity of fluid imbibed.
2. Increase in the molecular concentration of the urine as in diabetes mellitus, or after saline diuretics. More water is thereby attracted into the blood stream by osmotic pressure.

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3. Incapacity of the kidney to excrete a concentrated urine, as in chronic interstitial nephritis.

4. Dilatation of the kidney vessels, as produced by stimulating diuretics of the caffeine group. These diuretics have been shown by Curtis, using experimental methods, to act directly on tissue cells, causing the cells to part with their water; hydraemia results, and the excess of water in the blood is immediately excreted by the kidneys.

"Diabetes insipidus" is frequently due to disease of the pituitary gland or of the overlying hypothalamus, or to damage in this neighbourhood by syphilitic meningitis of the base of the brain. It is also probable that hysterical polyuria is due to a temporary inhibition of pituitary secretion through the sympathetic. This appears to affect the renal vessels directly, since pituitrin will check diuresis, even in the denervated kidney.

2.—ANURIA

Suppression, as opposed to retention of urine, may be due to—

1. Acute nephritis with intense congestion and nephrosis, whether the result of an infection or of drugs, such as turpentine, cantharides or carbolic acid.

2. Bilateral obstruction to the ureters.

3. Reflex causes, such as operations on the kidney or trigone of the bladder.

4. Vasomotor conditions, as collapse, shock or irritation of the vasomotor centre. Probably the anuria in diphtheria is due to the last of these (Garratt). In cholera there is not only collapse, but depletion of water by other channels.

5. Hysterical. This condition has been described by Charcot. It is, however, rare and the element of fraud must be eliminated. Thus, in one case, urea was found abundantly present in the contents of the washing-bowl, and this explained how the urine was disposed of.

3.—ALBUMINURIA

Albuminuria should be more correctly termed proteinuria—since blood serum contains two proteins—albumin and euglobulin—and either may appear in the urine, though search is seldom made for the latter. The ordinary tests of heat coagulation, nitric acid or salicyl-sulphonic acid give positive results with either. The presence of euglobulin may be shown by the addition of dilute acetic acid (33 per cent.) to urine in the cold. The acid is added drop by drop, and the precipitation of globulin is shown by an opalescence in the urine to which the acetic acid is added. Mucin is also deposited by the addition of acetic acid, but it is not redissolved by an excess of acid. A more distinctive test is the precipitation of globulin in distilled water. Single drops of urine are dropped into a glass vessel containing distilled water. As the drop of urine falls through the water it assumes a ring form, and the ring has a milky appearance due to precipitated globulin when the latter is present. The globulin can be precipitated for quantitative examination by making the urine alkaline with ammonia, and then half saturating it with ammonium sulphate.

Proteinuria may be classified thus—

I. WITHOUT ORGANIC DISEASES OF THE KIDNEYS, as in—

1. *Functional or orthostatic proteinuria*.—This is common in males between puberty and adolescence; it is much less common in females of the same age. Dukes found it in 16 per cent. of all boys entering Rugby School. Protein appears only in the urine secreted in the upright posture, and is absent from the urine passed on first rising. There is no evidence that the amount of protein in the food influences it, though some constituent of raw eggs may excite a transient albuminuria by a toxic action on the kidneys. Severe physical exercise will excite proteinuria in most healthy young adults. Collier found it present in every one of the Oxford crew of 1906 after rowing a course; to such a condition the term “physiological” proteinuria may fairly be applied. When the protein appears apart from exertion, the subject is often an anæmic weedy youth with a dull heavy aspect and a tendency to fainting. The heart is irritable, and the blood pressure unstable, and fluctuates with change of posture. There may also be a few hyaline casts, and frequently calcium oxalate crystals. In any case of proteinuria in a boy or young man the diagnosis of a kidney lesion should not be made unless casts other than hyaline are discovered, unless the tension of the pulse is definitely and permanently raised, and unless there are signs of cardiac hypertrophy. In the absence of such evidence, the urine passed on first rising should be examined. If this is free from protein, the condition is almost certainly functional. Then 15 grains of calcium lactate should be given three times a day for 3 days, after which the urine should be examined again. If this checks the proteinuria no further anxiety need be felt. Some milder forms of toxæmic kidney may simulate functional proteinuria, so that a search should be made for toxic foci, such as septic tonsils, tuberculous glands, or chronic appendix trouble, in all cases. A holiday is advisable if the patient has been doing hard mental work, as the condition is apt to appear under the strain of competitive examinations. A tepid bath, with cold sponging down the spine, and followed by vigorous towelling, is advantageous, and a general tonic such as strychnine, with iron if there is anæmia, should be prescribed. The condition soon rectifies itself when adolescence is past, and any case of proteinuria in a patient approaching thirty probably does not fall into this category.

2. *Febrile*.—Any acute specific fever may be accompanied by proteinuria due to cloudy swelling of the kidney. It should subside soon after the temperature falls to normal. This type of albuminuria is referred to again under the heading of Toxæmic Kidney, to which it more properly belongs.

3. *Congestive*.—In failing heart there is usually proteinuria from venous congestion of the kidneys. Hyaline casts may also be found. Unlike the urine of nephritis the urine is loaded with urates. After an epileptic fit there is often a transitory proteinuria, probably due to the congested condition of the veins during the fit. For a similar reason protein is apt to be present in the urine of any unconscious person.

4. *Toxic*.—This forms an intermediate group between those with and those without organic disease of the kidney, for if the action of the toxin be prolonged a definite nephritis may be established. Thus the proteinuria of pregnancy is generally regarded as toxic in origin, and may clear up

completely. The proteinuria sometimes seen in jaundice is also toxic in character.

II. WITH ORGANIC DISEASES OF THE KIDNEYS.—

1. *Nephritis, acute and chronic.*

2. *Residual albuminuria.*—This term is applied to cases in which albuminuria persists after complete recovery from an attack of nephritis. Observation of the case over a period of years may be necessary to exclude a low-grade progressive chronic nephritis. If and when residual albuminuria occurs it has the same significance as the scar of a perfectly healed wound in the skin. It would seem that residual albuminuria may persist throughout life unchanged, and there is no reason to think that the persistent passage of albumin of itself damages the kidney.

3. *Amyloid disease of the kidneys.*

4. *Tumours and infarcts in the kidney* may cause proteinuria, but more usually simple hæmaturia.

4.—ALBUMOSURIA

Albumose, or more correctly proteose, may be found in urine during autolysis of the tissues. It is not of great clinical importance except to distinguish it from Bence-Jones proteinuria. Proteose can be recognised by the fact that although it is precipitated by saturation with ammonium sulphate it is not coagulated by heat. Proteose precipitates disappear on heating and reappear on cooling. It can be separated from albumin by saturating the urine with crystals of ammonium sulphate, boiling and filtering. The precipitate on the filter paper is washed with water, when any proteose will be redissolved and carried through the filter paper. It can then be detected by the pink colour it gives on the addition of strong caustic soda and a drop of dilute solution of copper sulphate. With these reagents native proteins give a violet colour. The Bence-Jones protein, which is found in considerable amounts in the urine of sufferers from multiple myelomata, is not a true proteose though possessing similar solubilities. On treatment as above it yields a violet colour, showing that it has affinities with native proteins. It begins to be precipitated at 40°–55° C., but on approaching boiling-point most of the precipitate is redissolved. This is probably due to the influence of certain salts in the urine, and is not a property of the isolated protein. As Bradshaw showed, it also gives a ring of coagulum on contact with strong hydrochloric acid. Its recognition is of great diagnostic value, as it is pathognomonic of multiple tumours of the bone marrow, and enables them to be detected before there is any external sign, but only pain and tenderness in the bones. At a later stage the tumours may break through the investing bone and give rise to palpable swellings. Sometimes the Bence-Jones protein is spontaneously precipitated, causing the urine to appear milky. Considerable excess of phosphates may be found in this milky precipitate, probably derived from the autolysis of the surrounding bone.

True peptone is exceptionally found in the urine in pneumonia and phthisis, but is of no clinical importance.

5.—HÆMATURIA

When blood is intimately mixed with the urine it is held to be in favour of its renal origin. Bleeding from the bladder is more apt to occur into the last part of the urine voided, while urethral bleeding is said to occur chiefly into the first part. When the quantity of renal bleeding is not great, it imparts a smoky appearance to the urine, owing to the conversion of some of the hæmoglobin into methæmoglobin, which on spectroscopic examination gives an absorption band in the red in addition to the two bands in the green characteristic of oxyhæmoglobin. The chief causes of hæmaturia are best classified as follows :

1. *Prerenal*.—The altered condition of the blood which occurs, for instance, in scurvy, purpura hæmorrhagica and certain hæmorrhagic fevers, leads to the escape of some of the blood through the kidney without any evidence of a definite kidney lesion.

2. *Inflammations of the kidney*, due to (a) Bright's disease, both acute and chronic. Hæmaturia is a constant feature of acute nephritis and of exacerbations of chronic nephritis. It may also occur in the course of chronic interstitial nephritis and arterio-sclerotic kidney without any acute symptoms. "Renal epistaxis" is usually an early sign of an interstitial change, which is sometimes, as shown by Hurry Fenwick, confined to a single papilla where the vessels are dilated. There are a few cases in which no cause for the bleeding, either in the condition of the blood or the urinary tract, can be discovered in spite of the most careful examination of the kidney, the removal of which has been necessitated by the severity of the hæmorrhage. These are true cases of renal epistaxis or essential hæmaturia.

(b) Tuberculosis or a *Bacillus coli* infection. The latter more usually affects only the pelvis of the kidney.

(c) Certain drugs, such as turpentine, cantharides and carbolic acid, or occasionally hexamine.

3. *Vascular causes*.—Congestion due to heart failure, thrombosis and embolism (e.g. septic endocarditis) are common causes of hæmaturia.

4. *Irritation of the kidney by foreign bodies*, such as

(a) New-growth.

(b) Crystals, such as oxalates or uric acid, and calculi.

(c) Parasites, such as *Bilharzia*.

Traumatic, vesical and prostatic causes are not considered here.

6.—HÆMOGLOBINURIA

This is due to some hæmolytic agent. It may be—

1. *Paroxysmal*, as in Raynaud's disease and in syphilis. Most cases are syphilitic. The corpuscles are broken down by a hæmolysin which is present in the blood of 5 to 10 per cent. of cases of tertiary syphilis. Those who suffer from paroxysmal hæmoglobinuria are presumed to have some constitutional peculiarity which renders them susceptible to this hæmolysin. The hæmolysin acts as an amboceptor, unites with the red corpuscle in the cold and on return to warmth the normal complement in the plasma causes hæmolysis. In addition to this there are some rare forms of non-syphilitic paroxysmal hæmoglobinuria.

2. *Toxic.* In this group the toxic agent produces the hæmoglobinuria without an additional factor. Striking examples of this are blackwater fever (*q.v.*), poisoning by arseniuretted hydrogen, and transfusion of incompatible blood. Hæmoglobinuria may also occur in Lederer's anæmia. The chemical tests for hæmoglobinuria are the same as for hæmaturia, but the microscope will fail to reveal red corpuscles. Some of the pigment is excreted as methæmoglobin, especially after drugs of the aniline group, nitrites, or potassium chlorate.

7.—PORPHYRINURIA

Sometimes the hæmoglobin molecule is broken down in the blood stream and the pigmentary portion is excreted apart from the protein and iron. This is usually due to poisoning by sulphonal, trional or sulphanilamide, particularly when the drug has been taken regularly for a long time. It is then of grave prognosis; large doses of alkalis should be given. It is commoner in females than in males. Occasionally porphyrinuria occurs apart from these drugs, when it is not of grave import. It has been met with in cirrhosis of the liver, gastric ulcer and as a congenital abnormality of metabolism, when it may be associated with sensitivity to light and with hydroa vacciniforme. Exceptionally toxic symptoms occur even when it is not associated with drugs, as in two cases recorded by Ranking and Pardington, and by one of us. In these, some intestinal toxin with a reducing action appeared to be at work. The intestinal flora has been found rich in yeasts in such cases. Hæmatoporphyrin sometimes imparts a port-wine colour to the urine, but sometimes it is excreted as a porphyrinate. In the latter case the urine is brown, from the admixture of some unknown pigment, and the spectroscope shows two bands closely resembling those of oxyhæmoglobin. On the addition of an acid, however, the characteristic bands of acid hæmatoporphyrin appear.

8.—CHOLURIA

Another derivative of hæmoglobin, bile pigment, appears in all forms of jaundice due to obstruction of the main or intrahepatic ducts. In a true hæmolytic jaundice, such as acholuria family jaundice, as the name implies, bile does not appear in the urine. Bile pigment can often be recognised by noting the tinging of the froth caused by shaking the urine, but is best detected by the addition of a drop of fuming nitric acid to filter paper dipped in the urine, when rings of colour appear, green being the essential one. The green colour given on addition of a solution of iodine to the urine is a less delicate test. Bile-salts are often absent from the urine when bile pigment is present. Matthew Hay's test is the only reliable one for their presence there. On putting flowers of sulphur on the surface of the urine, they sink to the bottom, owing to the lowering of surface tension by the bile-salts.

9.—MELANURIA

Melanin only appears in the urine in melanotic sarcoma. Garrod has shown that in all other diseases in which melanuria has been recorded the test employed has been unsatisfactory. The melanin is excreted as melano-

gen which darkens on standing, and gives a black precipitate on addition of ferric chloride, which is soluble in excess of the reagent, yielding a black solution. A more delicate test is made by the addition of sodium nitroprusside and sufficient caustic soda to render the urine alkaline. The ordinary ruby-red colour, due to creatinin, is developed. The urine is now made acid with acetic acid, and if melanogen is present a prussian-blue colour appears.

10.—ALKAPTONURIA

This is not the manifestation of a disease, but is rather of the nature of an alternative course of metabolism, harmless and usually congenital and lifelong (Garrod). The individual is incapable of completely breaking down the tyrosin in the protein molecule, so that the intermediate product, homogentisic acid, appears in the urine. The urine reduces Fehling solution on boiling, but it does not ferment, and it darkens on standing, or at once on the addition of alkalis. It may stain the linen brown. When a dilute solution of ferric chloride is allowed to fall drop by drop into the urine, each drop produces a transitory deep blue colour. The urine reduces ammoniacal silver nitrate in the cold, giving a silver mirror on the sides of the test tube. Ochronosis—a blackening of the cartilages and ligaments, and sometimes of the conjunctivæ—may occur, and usually there is also a chronic arthritis, which may lead to a curious “goose-gait.”

[For other reducing substances in the urine, including sugar, see article on Diabetes.]

11.—KETONURIA

Ketonuria is a term used loosely to include the appearance in the urine of diacetic acid and its derivatives, acetone and β -oxybutyric acid. Acetone, however, being merely a decomposition product of diacetic acid, is relatively unimportant; β -oxybutyric acid, formerly regarded as the source of diacetic acid, is more saturated and less toxic and has been shown by Hurtley to be formed out of diacetic acid by the liver, as an attempt at detoxication. Diacetic acid is derived from the incomplete oxidation of fats or of the fatty acid groups in protein. It is probably always made in small quantities, but when there is an abundant consumption of carbohydrate, it is completely oxidised. In starvation the store of glycogen is quickly exhausted and the body chiefly lives on its fats; hence ketonuria. Persistent vomiting, advanced carcinoma of the digestive tract and rectal “feeding” also are equivalent to starvation, and will excite ketonuria, though without such a degree of acidæmia as to cause toxic symptoms. In conditions where the liver is thrown out of gear, such as post-anæsthetic poisoning, ketonuria may occur with toxic symptoms, because of the severe disturbance of all metabolic processes. But there are other agents at work besides diacetic acid which may be responsible for those symptoms. Only in advanced diabetes do we find toxic symptoms directly due to diacetic acid. Here there may be complete inability to utilise carbohydrates, so that the body perforce lives on protein and fats. If these are freely given in the food the amount of diacetic acid produced may be very large. But if a diabetic be fasted there is a great drop in ketonuria, showing that most of this is

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exogenous in origin (see Diabetes). The test formerly used for diacetic acid was the mahogany red colour given on the addition of ferric chloride. This has the disadvantage of being masked if the patient is taking any salicyl body. The nitro-prusside test was formerly regarded as showing the presence of acetone, but Piper demonstrated that it is really a much more sensitive test than ferric chloride for diacetic acid. A crystal of nitro-prusside of soda is dissolved in the urine, and then a strong solution of ammonia is poured on the top. A ring, the colour of Condyl's fluid, speedily develops at the junction of the liquids and spreads upwards. The intensity of colour is a rough measure of the degree of ketonuria. The reaction is made still more sensitive by previous addition of crystals of ammonium sulphate to saturation (Rothera).

12.—DRUGS WHICH ALTER THE COLOUR OF URINE

Methylene-blue is used as a colouring matter of sweets and also as an ingredient of certain proprietary pills. It is also given for coli infections of the urinary tract, gonorrhoea and bilharzia, or less commonly as an analgesic in rheumatism, sciatica and migraine. In small quantities it imparts a green colour to the urine, when it may be precipitated with the mucin. In larger doses it turns the urine blue. It can be recognised by its presence in suspension, so that it can be removed by simple filtration. It can be dissolved from the filter paper by chloroform, and is turned pink by the addition of alkalis. Eosin may be used in sweets and turns the urine a fluorescent pink. Prontosil rubrum and pyridium turn the urine a reddish-orange colour, though, if the urine is alkaline, this may not appear until it is acidified. Amidopyrine (pyramidon) may have a like effect. Rhubarb and senna may turn the urine reddish-brown from the chrysophanic acid they contain. The urine turns pink on the addition of an alkali. Santonin turns the urine a vivid yellow, which becomes rose-pink with alkalis. Carbolic acid may turn the urine greenish-black on standing, from the formation of hydroquinone. In carbolic acid poisoning the urine withdrawn by a catheter may even be found olive-green without exposure to the air. Other drugs, which may have this effect are salol, creosote, naphthalene and uva ursi. In chronic carboloria, ochronosis may occur as in alkaptonuria.

Certain drugs can readily be recognised in the urine by some colour reaction. Thus, salicylates are excreted as salicyluric acid, which gives a violet colour on the addition of ferric chloride. Copaiba, which is precipitated by nitric acid, can be distinguished from albumin by the solubility of the precipitate in alcohol. On the addition of hydrochloric acid a urine containing copaiba turns cloudy, the cloud soon becoming rose pink. Iodides in urine give a blue colour with guaiacum, and on the addition of hydrochloric acid impart a violet colour to chloroform shaken up with the urine.

13.—PYURIA

Pus may come from the urethra, prostate, bladder or kidney. The diagnosis of the source is discussed under septic diseases of the kidney. The best test for pus in the urine is the microscope. If the amount of pus be considerable it will yield a ropy mass on the addition of liquor potassæ.

If ozonic ether be shaken with the urine, bubbles of oxygen are evolved. With tincture of gualacum a blue colour may be given even without the addition of ozonic ether.

14.—CHYLURIA

True chyluria is due to blocking of the thoracic duct, most commonly by the *Filaria sanguinis hominis*, but sometimes the result of inflammatory or neoplastic conditions, with consequent rupture of lymphatics of the bladder through back pressure. Fat may be found in the urine in the lipæmia of diabetes, in growths of the kidney, and after fracture of long bones, when fat may be liberated into the circulation. Accidental contamination by an oily lubricant for a catheter and fraudulent addition of milk to the urine must be excluded. Pseudo-chyluria is due to a lecithin compound of globulin, and is sometimes found when there is a great excess of globulin in the urine. Unlike true fat, this substance is not extracted by shaking up with ether.

15.—PNEUMATURIA

Osler gives the following causes for gas in the urine: (1) Mechanical introduction of air in vesical irrigation or cystoscopic examination in the knee-elbow position. (2) Infection of the urine as by the *Bacillus aerogenes capsulatus*. (3) Vesico-enteric fistula.

16.—CRYSTALLINE DEPOSITS IN URINE

These may be:

1. *Uric acid*, which is characterised by multiplicity of forms and the yellow colour due to the urinary pigment they absorb. The chief varieties found are derived from the barrel and the whetstone types. Thus with a small whetstone stuck at either end of a barrel we get the lemon-shaped crystal. If the whetstones at the end of the barrel are larger, we obtain the "bicycle-handle" crystal. A very characteristic form is that derived from two whetstones with their broader ends apposed. The rosette crystal is a group of whetstones joined by their bases. The factors in the excretion of uric acid are considered under renal calculi; the chief factors in the deposit of uric acid crystals as such are high acidity, high percentage of uric acid, and poverty in mineral salts. The first two are the most important, especially the first. Deposits of urates are usually amorphous, but ammonium biurate may crystallise out as spheres with projecting spines.

2. *Oxalate of lime* is found in the urine, usually as small regular tetrahedra, which under the microscope appear as "envelope" crystals. They may arise (a) from ingested oxalates. Rhubarb, spinach, asparagus and sarrisel are the foods most likely to produce oxaluria sufficient to excite symptoms, for each contain more than 2 g. of oxalic acid per kilogram, though many other articles of diet contain some oxalates. Some individuals seem sensitive to strawberries which, however, only contain 0.06 g. per kilogram. (b) In either achlorhydria or hyperchlorhydria; the former permitting fermentation of carbohydrates, the latter promoting absorption of oxalic acid. (c) In crises in neurasthenics, with irritability, lassitude and neuralgic pains, without discoverable cause.

Oxaluria may cause smarting on micturition and may excite both albuminuria and hæmaturia. Its importance as a starting-point for renal calculi is considered later. Paralytic distension of the bowel has also been described in oxaluric crises.

3. *Phosphates*.—See Characters of Normal Urine (p. 1283).

4. *Cystin* is an amino-acid containing sulphur, and is contained in many proteins, being especially abundant in hair. Its presence in more than minute traces in the urine appears to be due to an inborn error of metabolism, affecting only the endogenous protein, since it is not increased by the administration of cystin by the mouth (Garrod). It is deposited as hexagonal plates, and is often accompanied by a variable amount of diamines, such as putrescin and cadaverin, pointing again to an incomplete breakdown of the tissue proteins. If the urine becomes infected, these cystin crystals may aggregate to form a calculus.

5. *Tyrosin* rarely appears in the urine as sheaves of fine glistening crystals. It is then generally accompanied by *Leucin*, which does not appear until the urine is concentrated by evaporation, when it forms spheres with concentric rings. The presence of these substances is sometimes regarded as pathognomonic of acute yellow atrophy of the liver, but they are occasionally seen in other severe disintegrations of the liver, such as cirrhosis.

17.—ORGANISED DEPOSITS

of red blood corpuscles, pus, epithelium, casts and spermatozoa do not call for detailed description here. The first two have already been referred to. For Epithelium and Casts see sections on Inflammatory Diseases of the Kidney.

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CIRCULATORY DISTURBANCES

1. *Active congestion*.—There is no distinction to be drawn between active congestion of the kidney and the early stage of acute nephritis.

2. *Passive congestion*.—Anything which raises the pressure in the renal vein must produce a passive congestion of the kidney. Failing compensation in valvular disease of the heart is the commonest cause of this; but it may also be brought about by respiratory diseases or by pressure on the renal vein by abdominal tumours or ascites. A transient congestion may result from an epileptic fit.

The cardiac kidney, as it is called, is the most typical example of passive congestion. The organ is firm and dark in colour, especially the pyramids. The capsule strips normally. The stellate veins are engorged. The kidney may drip with blood on section, and if placed in a dish after section soon exudes œdematous fluid.

The urine is scanty, high-coloured and of high specific gravity. Unlike the urine of chronic nephritis it is loaded with urates. It contains a variable amount of albumin and hyaline casts, with a few red blood corpuscles, if the congestion is at all considerable. Renal inadequacy does not reach

the high grade seen in true nephritis, nor is death from uræmia likely. The prognosis and treatment are those of the cardiac condition causing it. Stimulating diuretics are of much more service than in nephritis, since there is no primary disease of the secreting structures.

3. *Infarction*.—This, which is a common complication of infective endocarditis, may take two forms—(a) Multiple minute hæmorrhagic infarcts, producing the “flea-bitten” kidney, which may lead to foci of embolic nephritis with fibrinous exudate and leucocytic infiltration. (b) Larger anæmic infarcts, “map-like” areas of coagulation necrosis, roughly wedge-shaped, but with irregular edges and with the base reaching the surface of the organ. Their formation may cause a sudden pain in the loins, if they are large. Either of these conditions will cause both albuminuria and hæmaturia.

4. *Thrombosis of the renal vein*.—This is rare, and is usually significant of a terminal infection, as in a marasmic infant. In thrombosis of the inferior vena cava the process may reach as high as and spread into the renal vein. This would produce the same effects as the cardiac kidney, but in a much more intense form.

BRIGHT'S DISEASE

Bright described an acute inflammation of the kidney accompanied by dropsy and albuminuria, and a chronic form in which dropsy is absent. There has been much controversy as to what should be included in the category of “Bright’s disease,” but there is no doubt as to its essential features. It is a bilateral, non-suppurative affection of the kidneys, accompanied by albuminuria and cylindruria. Except in nephrosis, there is generally hæmaturia in the acute or active stages; œdema and effusion in the serous sacs are commonly present. The renal lesion is diffuse in acute and chronic nephritis, but in “chronic interstitial nephritis” it is chiefly localised in wedge-shaped areas, separated by renal tissue which remains relatively normal. The actual lesion in all forms of nephritis is obviously inflammatory, as shown by proliferation of cells, particularly the cells of Bowman’s capsule, the layers of which become adherent while the multiplication of their cells leads to crescent formation. There is also small-cell infiltration and œdema of the interstitial tissue of varying degree. Accompanying these inflammatory changes are degenerative changes, chiefly evident in the renal tubules, namely, cloudy swelling, fatty, hyaline and other forms of cellular degeneration, and necrosis. In some forms of Bright’s disease the inflammatory changes predominate; in others the degenerative. In one uncommon form the degenerative changes are so marked a feature of the histological picture, while the changes which are without doubt inflammatory are so slight or even absent, that this form is called Nephrosis in contradistinction to Nephritis.

There are other affections of the kidney, such as toxæmic kidney, hyperpietic kidney (benign nephrosclerosis) and senile or atheromatous kidney, which would be better separated from the category of Bright’s disease, because in them the disease of the renal parenchyma is neither the first established nor the primary condition of disease. It is, indeed, but part of a widely distributed pathological change in other organs of the body.

They are, however, included in the present classification and description of Bright's disease, because there are intermediate forms which link them to Bright's disease, and because in some cases they develop into Bright's disease.

There is so much overlapping of the various types of Bright's disease that more is lost than gained by pressing distinctions in detail. Volhard, Van Slyke and others recognise three types of Bright's disease which are essentially different in their genesis and pathological nature, namely (1) the hæmorrhagic or glomerular, marked primarily by glomerular inflammation, with hæmaturia and usually diminished renal function (even in the acute stage); (2) the sclerotic disease, marked primarily by pathological changes in the small arteries of the kidneys (and usually other organs), with hypertension as the first sign, and diminished renal function only as a terminal phenomenon; and (3) the degenerative disease or diseases, called nephrosis, marked primarily by degenerative changes in the kidneys, without hypertension or hæmaturia. This classification has long been the accepted basis for the description of Bright's disease, and in our opinion it best harmonises the anatomical, pathological and clinical phenomena.

I. DEGENERATIVE FORMS:

(A) *Toxæmic kidney.*

(B) *Nephrosis.*

II. INFLAMMATORY FORMS.

(A) *Glomerulo-tubular nephritis. (Diffuse nephritis).* 1. Acute nephritis. 2. Chronic nephritis: (a) secondary type; (b) primary type.

(B) *Embolic focal nephritis* set up in infective endocarditis.

III. VASCULAR GROUP.

(A) *Chronic interstitial nephritis, including malignant nephrosclerosis.*

(B) *Hyperpietic kidney* (benign nephrosclerosis).

(C) *Senile or atheromatous kidney.*

TOXÆMIC KIDNEY

Definition.—Certain toxic substances may excite degenerative rather than inflammatory lesions in the kidneys, which are nevertheless capable of complete recovery. Characteristically, as in febrile albuminuria, the affection of the kidneys is dependent, both for its inception and persistence, on some other disease, and, generally speaking, its intensity varies with the severity of the primary disease. In its onset, intensity, course and termination, it simply reflects the toxæmia which causes it.

Ætiology.—The commonest cause is bacterial toxæmia. As fever in itself does not necessarily cause albuminuria, all the so-called "febrile albuminurias" should be referred to this group. Thus the acute specific fevers—pneumonia, typhoid fever, diphtheria, small-pox, tonsillitis and scarlet fever (notwithstanding the fact that the two last often cause a true nephritis)—are common causes of the slighter degrees of toxæmic kidney. More potent are exogenous and endogenous poisons. Mercurial salts, arsenic,

phosphorus and cantharides are important causes clinically, while uranium and bichromate salts are frequently used in the experimental production of the condition. Jaundice and the ketosis of diabetes mellitus are not uncommon causes. The toxæmias of pregnancy belong to this group, but in their tendency in some cases to develop into chronic nephritis and their frequent association with a raised blood pressure and visual disturbance, these cases differ from other members of the group.

Pathology.—The post-mortem appearances are not distinctive. The cardio-vascular system is normal. The kidneys are pale and likely to be increased in size and weight. On section the cut surface of the cortex is pale in contrast to the congested pyramids: it is increased in thickness and its structure is blurred. On microscopical examination the parenchyma shows degenerative changes, particularly affecting the convoluted tubules. Apart from the presence of some swelling of the glomerular tufts, and the presence of an albuminous exudate in the intercapsular space, the glomeruli show little damage. The absence of tissue reaction that is undoubtedly inflammatory and the presence of tissue changes that are certainly degenerative are the distinctive features of the histological picture. Similar changes are to be found in other organs of the body, and especially in the liver, which may show various degrees of damage, namely, cloudy swelling, fatty degeneration and focal or diffuse necrosis.

Symptoms.—When due to bacterial toxæmia the condition does not give rise to symptoms. It is recognised by the presence of a trace or cloud of albumin in the urine on boiling, and by the presence, in the centrifuged deposit, of granular, hyaline and epithelial casts. In addition, there may be a few white blood corpuscles; when red blood corpuscles are present, or when there is frank hæmaturia, the differential diagnosis from an acute nephritis cannot be made with certainty. In the severer types with insidious onset, as in mercurial poisoning or in the toxæmia of pregnancy, the first symptom is often malaise, disturbance of digestion and constipation, accompanied by albuminuria and oliguria. Headache is a prominent symptom, and is often persistent. In the toxæmia of pregnancy a rising blood pressure or œdema may be the initial sign; the œdema is either general or it appears first in the lower extremities, as in cardiac œdema. Eye symptoms are important; there may be dimness of vision and flashes of light before the eyes, or rarely sudden blindness. On examination of the fundus oculi the disc may appear normal, or there may be œdema of the disc or partial detachment of the retina. The vessels are normal and hæmorrhages are rare. These symptoms may be followed by fits (hypertensive crises), but sometimes the fits occur without previous warning. In general, the symptoms of a fully developed case are clinically indistinguishable from those occurring in the uræmia of true nephritis. The urine contains up to 3 or even 4 per cent. protein. Hyaline, granular and epithelial casts may be present, and may be very numerous; white blood corpuscles may be present, though few in number. In severe cases, the urine contains blood-cell casts, the result of capillary thrombosis and extravasation of blood.

Diagnosis.—The diagnosis depends on the recognition of the signs and symptoms of kidney disease in a patient affected by one of the known causes of toxæmic kidney, and on certain biochemical tests by which a true nephritis can be reasonably excluded. In milder cases, the possibility of the symptoms

being due to heart failure must be excluded. Complete recovery is in favour of the diagnosis of toxæmic kidney. Exacerbation of a chronic nephritis can be recognised by a history of previous nephritis and the presence of definite cardiac hypertrophy and arterial changes. The difficulties are sometimes considerable, however, since a marked rise of blood pressure may occur in a toxæmic kidney, while advanced degrees of chronic nephritis may occur without cardiac hypertrophy, increased blood pressure or clinical evidence of arterial disease. The blood urea is normal in the toxæmic kidney; whereas in chronic nephritis the blood urea tends to rise, and may reach 300 mgm. or even more. In the toxæmia of pregnancy, the appearance of albuminuria in the early months of pregnancy is in favour of the condition being one of chronic nephritis, whereas the albuminuria due to toxæmia generally makes its first appearance in the later months.

Prognosis.—The importance of recognising the toxæmic kidney is that both the immediate and ultimate outlook are better than in nephritis of apparently equal severity. The prognosis in the single case depends on the nature of the cause, the degree of its severity, and the possibility of its early and complete removal. Recovery, when it occurs, is complete, but in the severe cases chronic nephritis may supervene.

Treatment.—If not already in bed on account of the condition responsible for the toxæmic kidney, the patient should be immediately confined to bed. Treatment is directed towards eliminating the toxins and resting the kidneys. Barley water and milk and soda should be given. An easy but not loose evacuation of the bowels must be secured daily by the use of magnesium sulphate, jalap, senna, or compound liquorice powder. A simple diuretic and diaphoretic mixture, such as potassium citrate grs. 15, liq. ammon. acetatis min. 60, sp. ætheris nitrosi min. 15, aq. chloroformi ad $\frac{1}{2}$ oz., is given in water every four or six hours. The intake of solids is limited. At the same time, since there is no retention of urea in the blood, it is unnecessary strictly to limit the intake of protein. Soups, meat extractives and condiments are to be withheld. The action of the skin may be stimulated with hot packs or hot baths. The fits are best treated by venesection. The above outline of treatment is for the severer cases; for the febrile albuminurias, special treatment for the renal condition is not required.

NEPHROSIS

This form of Bright's disease is characterised by cedema, marked albuminuria, and two characteristic changes in the blood, namely, a fall in the plasma albumin and increase of cholesterol. It is distinguished from the glomerulotubular type of acute nephritis by the absence of hæmaturia, cylindruria, hypertension and urea retention, and also by the fact that anæmia is less often present in nephrosis or, if present, tends to develop only at a late stage.

Ætiology.—In most cases no ætiological factor is established, and the first evidence is the onset of cedema without previous illness. In the remainder the best established cause is syphilis. Tuberculosis and osteo-myelitis may be ætiological factors. There may be a recent history of chill, of upper respiratory tract catarrh, or of gastro-enteritis. It may be that streptococcal

infection, more especially of the upper respiratory tract, is a cause of the complaint.

Pathology.—The condition of the kidney is that of toxæmic kidney in a more severe form, the fatty changes in the tubules being very marked.

Symptoms.—The disease is generally first recognised by the gradual or rapid onset of œdema, which gradually increases and tends to become massive. The œdema may be generalised, affecting the scalp, hands, trunk and legs. It is often first noticed as puffiness of the eyelids, or it may first appear as a swelling of the feet and ankles extending up the legs. The patient may feel quite well apart from the disability caused by œdema. On the other hand there is more usually complaint of malaise and fatigue, loss of appetite and nausea, and sometimes of epigastric pain. There may be cough and slight shortness of breath due to slight bronchial catarrh, œdema of the lungs or hydrothorax. Swelling of the abdomen may be the result of œdema of the abdominal wall or ascites. The face is pale and the eyelids and cheeks are puffy, but the mucous membranes are of a good colour, and the blood count is normal. (The urine is reduced in quantity, its specific gravity is normal or raised, it contains a large amount of albumin, often amounting to 0.5 or even 1 per cent., and readings of 4 per cent. or even more have been recorded. The urinary deposit contains but a slight excess of cells and few or no casts. Red blood corpuscles are generally absent. The heart and blood pressure are normal, but a pericardial effusion may develop. There is no retinitis and no urea retention. Characteristic changes are found in the blood. The plasma albumin falls more considerably in nephrosis than in other forms of Bright's disease. The normal figures are plasma albumin 4.1 g. per cent., globulin 2.6 g. per cent., total protein 6.7 g. per cent., which gives an albumin-globulin ratio of 1.6 to 1. In nephrosis, plasma albumin may be 2 to 1 g. per cent., globulin 2.6 to 3 g. per cent., so that albumin-globulin ratio varies between 1:1 and 1:3. The blood cholesterol is raised to 300–800 mgm. per cent. (normal 130–200 mgm. per cent.).

Course.—The disease pursues a chronic course. In the first stage there is a gradual increase of symptoms. When the disease is fully developed it may remain more or less stationary for a number of months, at any time during which there may be some exacerbation or remission of symptoms. During an exacerbation the symptoms increase and convulsions may occur. After remaining stationary for some time, even up to 6 or 12 months, there may be a gradual remission of symptoms, and then complete recovery. In other cases an intercurrent infection, such as pneumococcal peritonitis, broncho-pneumonia, or erysipelas, is responsible for a fatal termination. Or the clinical picture of the disease may gradually take the form of chronic nephritis, in which case the œdema tends to become less, lethargy and fatigue increase, anæmia develops, the blood pressure gradually rises, cardiac hypertrophy follows, and death results from uræmia.

Diagnosis.—The differential diagnosis from nephritis is made on the absence of hypertension and hæmaturia, and the presence of a normal or low blood urea. The fall in plasma albumin and the rise in blood cholesterol are both greater in nephrosis than in nephritis. In nephrosis, cylindruria is relatively slight or absent, and anæmia is uncommon. The differential diagnosis from amyloid kidney may not be possible during life. The presence of splenomegaly, anæmia, and especially a chronic infection, such as chronic

osteo-myelitis (a potent cause of amyloid disease), would be in favour of the diagnosis of amyloid change.

Prognosis.—The importance of the recognition of nephrosis depends largely on the fact that complete recovery may occur even after the disease has been present for many months.

Treatment.—The possibility of a toxic cause, particularly syphilis, and to a slight extent bacterial or other toxæmia, should be borne in mind. Syphilitic cases may respond to specific treatment, which, however, must be prescribed with caution. Obvious sources of sepsis should be removed wherever possible. During the first stage of the disease, and indeed as long as there is good hope of recovery, the patient should be kept in bed and nursed between blankets. Fluid intake is limited to that short of causing thirst. During the early stage, when there may be some doubt as to the differential diagnosis from subacute nephritis, the patient should be put on a diet of low protein and salt content. When the diagnosis is established, and the low plasma protein confirmed, adequate protein is given to maintain nitrogen equilibrium. This means something more than 1 gr. per kilo of body weight per diem: in some cases a high protein intake seems to be beneficial, though this treatment has not fulfilled the expectations originally formed. It is not usual now to order a completely salt-free diet, though it is advisable not to permit the addition of salt at the table. If the blood cholesterol is high, the intake of fat should be restricted. The bowels are regulated with magnesium sulphate or some other bland laxative, such as senna pods or compound liquorice powder. Constipation and loose stools are both to be avoided. In some cases the administration of potassium salts is effective in producing diuresis and reducing the œdema, but must be given very cautiously to avoid depressing the heart. The maximum dose given is 120 grs. daily of a mixture containing equal parts of potassium bicarbonate and potassium citrate. Perhaps the best diuretic in such cases is urea by the mouth in doses of 30 to 60 grs. three times a day; considerably larger doses are sometimes given, but in that case the blood urea should be watched. Thyroid has been advocated by some observers. It acts presumably by raising the basal metabolic rate, which is often low in nephrosis. When other means have failed and considerable œdema persists, decapsulation should be seriously considered. It is quite often temporarily or even permanently effective in curing, or at least greatly reducing, the œdema. It has not, in our experience, affected the albuminuria so dramatically as it may the œdema, but on occasion it has seemed to determine a favourable turn in the course of the disease. Opportunities for symptomatic treatment should be looked for, such as the treatment of anæmia with iron, or some slight degree of heart failure with digitalis, or the control of sleeplessness, loss of appetite and dyspepsia, depression and nervous agitation.

ACUTE NEPHRITIS

The classical form of acute nephritis is hæmatogenous in origin and essentially glomerulo-tubular in distribution. Such a definition would exclude an ascending infection of the tubules from the pelvis of the kidney, such as occurs in pyelonephritis. It would also exclude the embolic nephritis of

infective endocarditis, where inflammatory foci are set up in the kidney as the result of septic emboli reaching it from the heart. These produce marked fibrinous exudation and infiltration with leucocytes; but only some capillaries in some of the glomeruli are affected.

Ætiology.—Acute nephritis was formerly not a common disease. Herringham found, at St. Bartholomew's Hospital, where the average number of medical cases is 7000 a year, that there were, in a period of 9 years, only 166 cases, 120 being in males. On the other hand, a large number of cases occurred in the epidemic of acute nephritis in the War of 1914–1918, 1500 being recorded in Flanders alone during 1915. Since the War of 1914–1918 it would appear to have become more frequent.

The causes usually given for acute nephritis are as follows:

1. **ACUTE SPECIFIC FEVERS.**—Scarlatina is undoubtedly the commonest specific fever to produce it. Goodall found nephritis in 8·4 per cent. of all cases of scarlatina. Nephritis is an occasional complication of typhus, small-pox, chicken-pox and mumps. Syphilis, malaria and yellow fever may also cause it. Many cases of nephritis are preceded by tonsillitis, or otitis media, and it is probable that the throat is often the door of entry for the infection.

2. **DISEASES OF THE RESPIRATORY TRACT.**—It may also occur as a complication of other acute infections of the respiratory tract. The commonest bacterial agent is the streptococcus.

3. **DISEASES OF THE SKIN.**—The frequency with which acute nephritis may follow burns or extensive skin diseases is interesting, in view of the physiological connection between the kidney and the skin. It is a not infrequent complication of erysipelas, impetigo, boils, pemphigus and dermatitis. It must be remembered, also, that children who have been burnt are very liable to develop true scarlatina as well as a mere septic rash, and that streptococcal infection may be the responsible agent.

4. **DISEASES OF OTHER SYSTEMS.**—Acute nephritis may also be a complication of acute infections of other systems. Purpura, which is probably toxic in origin, may be accompanied by a true nephritis.

5. **EPIDEMIC TYPE.**—In the American Civil War and in the War of 1914–1918 acute nephritis occurred as a primary disease in an epidemic form, characterised by dyspnoea at the onset, and in general by a benign course. In the fatal cases, inflammatory and thrombotic lesions were found in the lungs and spleen.

It is a very common idea that cold or chill is a cause of acute nephritis. The statistics of the army epidemic go far to disprove this. For, during the first winter, when there was much wet weather, and the men were much exposed, cases were few and far between, and not until the weather was better did the disease assume epidemic proportions. On the other hand, a patient who has nephritis is more susceptible to cold, which may provoke an exacerbation. Where exposure seems to be responsible for acute nephritis, examination will generally reveal some definite evidence of an old-standing lesion of the kidneys. Conformably with that, after the first winter of the war, there was an agreement between the incidence of nephritis in the army and low temperature.

Pathology.—The kidney is swollen, with occasional punctiform hæmorrhages over a pale, greyish surface. The cortex is increased and, on section, its pallor contrasts with the deep red medullary cones. Microscopically, the

glomeruli are swollen, becoming pear-shaped and protruding into the first part of the convoluted tubules, with Bowman's capsule tightly stretched over them. In these glomeruli the nuclei are less distinct, and the capillaries show fatty changes in their walls. The capillary loops become filled with exudate and empty of red blood corpuscles; their lumina contain a fine network of coagulated substance and leucocytes. There is proliferation of the endothelial cells, and mitotic figures are not infrequent. A serous exudate and a varying number of red and white blood corpuscles may be extravasated between the layers of Bowman's capsule. The convoluted tubules have their lumen blocked either by the swelling of their epithelium or by debris, casts and blood. The interstitial tissue is swollen and œdematous, with hæmorrhages here and there, and sometimes lymphocytic infiltration. The arteries of the kidney show little alteration except that some of the afferent arterioles share in the glomerular changes.

Symptoms.—The onset is usually acute, though occasionally it may be rather insidious. In the latter instance the patient may complain of biliousness, nausea, vomiting and abdominal pain, with headache and sometimes diarrhœa before the onset of renal symptoms. In the cases with acute onset, he may have more or less severe pain in the back, and œdema soon develops. It usually starts in the face; the legs and scrotum are generally involved next, and the swelling soon spreads all over the body. Occasionally the dropsy is curiously localised and fugitive. Though dyspnœa is not regarded as a common feature of acute nephritis apart from uræmia or cardiac failure, in the army epidemic it was almost invariable at the onset. As a rule, shortness of breath started at the same time as the dropsy, but did not last so long, having ceased at the end of 2 or 3 days. There is usually only slight fever, though occasionally a temperature of 102° or 103° may be reached. Some irregularity of temperature, however, is common in the first week or 10 days. The pulse may be raised in tension and the blood pressure is generally raised. Occasionally the serum is milky, as was pointed out by Bright. The skin may be dry and itching, with occasionally a papular or erythematous eruption. Retinal hæmorrhages rarely occur.

The urine is greatly reduced in volume, and may be entirely suppressed. Eight to 12 ounces would be an ordinary figure. It is dark in colour and usually contains obvious blood. This may render the urine as dark as porter, but it may be bright red or merely smoky. Sometimes the blood forms a flocculent, reddish-brown precipitate. The urine is usually loaded with albumin, and casts will be found on microscopical examination. At first blood casts and epithelial casts will alone be found; but, at a later stage, granular and hyaline casts will appear. Fatty casts are not found in the first attack of acute nephritis. Their presence suggests a recrudescence of a chronic disease. Isolated renal cells, transitional epithelium and squamous cells from the lower urinary tract are also commonly found. Micro-organisms are not usually observed, and their presence in any number would suggest that the case is more probably one of pyelonephritis. A sudden rise in the secretion of water after a few days is usually a sign of definite improvement.

The sedimentation rate is raised in acute nephritis, and, in favourable cases, its fall is closely related to the reduction in hæmaturia, but when there is continued activity of the disease-process a raised sedimentation rate is the

rule (Oakley). In the presence of nephritic œdema the sedimentation rate is very high in contrast to the low rate of cardiac œdema. This is due to the alteration in plasma proteins in the former condition, and especially to the increase in plasma fibrinogen.

Complications may be due to three main causes.

1. *Renal failure, i.e. uræmia* may develop. Some slight uræmic symptoms are common in acute nephritis, such as headache, dizziness, nausea and vomiting. But any of the forms of uræmia described later may assert themselves. Convulsions are the most common of the severe symptoms, but are not as grave in significance as in chronic nephritis. If treated promptly, recovery may follow.

2. *Extension of the œdema*.—Water-logging of the lungs may occur, producing serious dyspnoea; but this is sometimes chiefly due to cardiac failure. In any case it is serious. A milder degree of bronchial catarrh is quite common. A rare but very dangerous complication is œdema of the glottis, which calls for prompt treatment.

3. *Secondary infections*.—The subjects of nephritis are always liable to secondary infection, and these are particularly apt to affect the serous membranes; therefore pleurisy, pericarditis, and peritonitis are not uncommon complications. The last two are very dangerous.

Sequelæ: If complete resolution does not occur, the patient will develop chronic nephritis of the secondary type (see p. 1309).

Diagnosis.—The combination of dropsy, albuminuria, hæmaturia, casts and scanty urine usually makes the diagnosis quite easy. The differential diagnosis of acute nephritis from an exacerbation of chronic nephritis may be difficult. Definite evidence of cardiac hypertrophy and arterial changes would be in favour of the latter. The presence of granular casts at the outset, or of fatty casts at any time, is suggestive of chronic disease. An infarct in the kidney which causes a pain in the back and hæmaturia may simulate nephritis, but general dropsy is not likely to occur, nor are casts present in the early stages. Great reduction in the volume of urine is not usual. It must be remembered, however, that infarcts may start foci of nephritis.

Signs of septic endocarditis would suggest infarction. In chronic interstitial characteritis there may be a smart hæmorrhage, but the abundant urine of low gravity and the cardio-vascular signs would lead to a correct diagnosis. In the hæmaturia and the cardio-vascular signs would lead to a correct diagnosis. In the hæmaturia and the cardio-vascular signs would lead to a correct diagnosis.

It is a very confusion with acute nephritis is not likely to occur. Moreover, The statistics of the could not be found, though a large blood cast from the first winter, when this is a very characteristic feature. Pyelitis may give rise to exposed, cases were severe may be small hæmorrhages, especially at the beginning better did the disease of micro-organisms in a catheter specimen and abundant a patient who has nephritis only a haze of albumin, in the absence of casts, will an exacerbation. When diagnosis clear. Moreover, general dropsy does not occur examination will generalise up severe nephritis as a sequel. In any case of hæmorrhage of the kidney when it is associated with profound constitutional disturbance, there was an eight, tachycardia, continued fever and peripheral neuritis, and low temperature, peri-arteritis nodosa must be considered (q.v.). Lastly, in

Pathology.—Especially streptococcal and complicating an ascending infection rages over a pale, glist, the possibility of acute interstitial nephritis should be its pallor contrasts with the diagnosis will be suggested by the presence of albumin-

uria and oliguria, or by symptoms of uræmia complicating septicæmia. The diagnosis is usually made post mortem.

Prognosis.—The prognosis naturally depends on the severity of the disease. It is better in those cases where there is a discoverable cause, an acute onset, and where the patient comes under treatment promptly. Recovery is usually slow, and the criterion of the cessation of the acute stage is the disappearance of red blood corpuscles from the urine.

Volhard distinguished a separate form of acute nephritis under the name of acute focal nephritis. Its onset is sudden, and recovery is the rule. It is recognised clinically by the presence of hæmaturia and albuminuria without œdema, hypertension or urea retention. There is little constitutional disturbance. It is more frequent in children than in adults, and it is said to occur in epidemics. Its ætiology is the same as that of acute diffuse nephritis. It is doubtful if it can be regarded as a distinct clinical entity, but we recognise it to this extent that acute nephritis having these clinical features carries with it a good prognosis, and recovery is to be anticipated in 2 to 4 months. Mild cases recover sooner. When in addition to the above clinical picture there is added hypertension, urea retention, moderate albuminuria and some degree of anæmia, the course of the disease is likely to be longer, and 12 months' duration with complete recovery is not uncommon. We have experience, too, of complete recovery after an illness of 2 years' duration in severe cases of the above type. When in addition to the above there is considerable œdema and massive albuminuria with hæmaturia, and with or without hypertension, the ultimate outcome of the disease cannot be foretold, but in general terms a somewhat better prognosis should be given in acute and subacute nephritis than the present condition of the patient seems to justify, especially for the reason that it encourages persistence with treatment. The disease is rarely fatal in the acute stage. In the subacute stage, namely, after the first 3 weeks of illness, it may enter a stationary phase, or become slightly progressive. The patient may die from uræmia, secondary infections or extension of the œdema to vital structures. The longer the duration of the hæmaturia, even if it be a microscopic hæmaturia, the more likely is there to be some permanent damage to the kidney, and the development of chronic nephritis.

Treatment.—(a) **PROPHYLACTIC.**—The best prophylactic measure is prompt and efficient treatment of any infective process liable to set up nephritis. There is evidence to show that the routine administration of alkalis in scarlet fever diminishes the incidence of acute nephritis. The enucleation of obviously infected tonsils, especially when an attack of tonsillitis has been accompanied by albuminuria and cylindruria, is advisable. The early administration of scarlatina antitoxin serum in a severe case of scarlet fever is prophylactic treatment of nephritis complicating this disease.

(b) **CURATIVE.**—The indications are to remove, if possible, the microbic or toxic cause at work and to ensure such physiological rest for the kidney as is practicable; to promote elimination of nitrogenous and saline waste by other channels; to treat complications as they may arise and to correct the resulting anæmia. In this way much may be done to steer the patient towards recovery, although we can do little to control the course of the inflammatory process. The patient is naturally kept recumbent in bed. To counteract the congestive effects of gravity, it is well to move him from

side to side, and occasionally to put him on to his chest. He should be clad in a flannel nightgown, and be placed between blankets to guard against chills and to encourage free action of the skin. The room should be warm and well ventilated. If suppression of urine threatens, dry cups or poultices should be applied over the loins. This measure is sometimes successfully adopted to diminish hæmaturia.

Diet.—In acute nephritis, the danger of overloading the inflamed kidney with nitrogenous substances is hardly sufficiently recognised; whilst in chronic nephritis the dietetic restrictions are apt to be too severe. The dictum that “in acute affections we concentrate our attention on the diseased organ, whilst in chronic cases we keep the general condition of the patient more in view,” applies particularly to the treatment of nephritis. Nitrogen retention is common and a source of danger, so that the free administration of milk usually recommended is open to objection, since cow’s milk contains 4 per cent. of protein, which equals 0.56 per cent. of nitrogen. It will do little harm to deprive the patient of nitrogen for a time, and von Noorden advises restriction of the diet at the outset to fruit juice, water and sugar. Where there is no nausea, toffee is allowed, which, being composed of butter and sugar, throws no work upon the kidney. It is generally appreciated by children and allays hunger. Barley water, with a little milk added, may also be given, and as the patient improves the proportion of milk may be increased. It is quite unnecessary to give anything else for a few days, and the relatives’ fear of starvation may be allayed by explaining the rationale of the treatment. The excretion of nitrogen is reduced to its lowest level by giving a diet of fats and carbohydrates, when it may fall below that of a fasting person, as was shown by Folin; but excess of fat is inadvisable for reasons given under chronic nephritis. It is well to restrict or withhold table salt and substitute a mixture of formates, citrates and phosphates, such as ruthmol. The fluid intake and urine output should be measured, and a written record kept of total quantity of fluid taken in and excreted every 24 hours. The patient should not be allowed to be thirsty, but generally speaking the amount of fluid allowed in the day should not exceed 3 pints for an adult or 30 oz. for a child of 12. There may be a sudden diuresis after some days, and it is a sign of recovery. It is sometimes termed a “critical diuresis,” and after its occurrence the quantity of water and milk taken may be increased. A drink prepared by adding 1 pint of boiling water to 60 grains of potassium acid tartrate, half a lemon, and some sugar, stirred occasionally until cold and then strained, may be allowed throughout in moderate quantities. The citric acid and the tartrate become bicarbonates in the blood and may render the urine less irritating by making it less acid; apparently it is not as easy to render urine alkaline in a severe case of acute nephritis as it is in the normal individual. Beef-tea, broth and meat juices are all to be condemned as imposing work on the kidney with very little corresponding nutritive advantage.

GENERAL TREATMENT.—The bowels are kept open by a daily laxative such as pulv. jalapæ co., magnesium sulphate or sodium sulphate, or a preparation of cascara. It is important to secure an adequate and easy evacuation of the bowels, but loose stools should be avoided. Occasional constipation is better treated with an enema than by a large dose of laxative. The function of the skin is promoted, and protection from chill is secured by

keeping the room warm and well aired. The patient is nursed between blankets, and wears a flannel nightgown or a vest, preferably with long sleeves, under the night attire to which he is accustomed. By this means the skin is kept warm and at an even temperature. In addition, the patient should be sponged with hot water followed by friction with warm dry towels. More drastic measures are seldom called for in acute nephritis unless uræmia is impending, when the hot-air bath may be of service.

In the acute stage stimulating diuretics are contra-indicated. Saline diuretics, such as potassium citrate, may be given. In so far as saline diuretics, such as potassium citrate, produces diuresis, they do it by raising the osmotic pressure of the blood, and thus drawing water from the œdematous tissues. The following prescription is mildly diaphoretic and diuretic :

Pot. cit., grs. 15.

Liq. ammon. acetatis, min. 60.

Sp. æth. nitr., min. 15.

Aq. camph. ad fl. oz. 1. To be taken every 6 hours.

The addition of 5 minims of tincture of digitalis is advisable if the heart's action becomes weak.

TREATMENT OF COMPLICATIONS.—For the treatment of renal failure, see Uræmia. Pleurisy, pericarditis, or peritonitis should be treated on ordinary lines. Œdema of the glottis may call for scarification of the larynx or even tracheotomy.

AFTER-TREATMENT.—Bed is imperative until red blood corpuscles have disappeared from the urine and is advisable until albuminuria has ceased altogether. This may be impossible, since acute nephritis may go on to chronic nephritis, but there is a considerable advantage in prolonging the rest as much as possible. Bread, butter, vegetables, puddings, eggs and then fish may be gradually added to the diet, according to the scale given under chronic nephritis, as the hæmaturia and albuminuria diminish, but abstention from meat is advisable for some time, and meat extracts had better be altogether avoided. If anæmia develops, iron is given in the form of ferrous carbonate 45 grains, or ferrous sulphate 9 grains, daily in divided doses after food. Chills should be guarded against in every possible way, and the loins may be protected by wearing a well-fitting cholera belt.

CHRONIC NEPHRITIS

(a) SECONDARY TYPE.

It is generally agreed that chronic nephritis involving the parenchyma of the kidney is diffuse from the first, though naturally the interstitial changes take longer to manifest themselves. It is certain that when parenchymatous nephritis has existed for any length of time, there will be interstitial change as well. On the view here adopted, "chronic parenchymatous nephritis" or large white kidney is the subacute stage of a glomerulo-tubular inflammation. If the patient lives long enough, the kidney will pass into the contracted stage, formerly known as small white kidney.

Ætiology.—It is most frequently the sequel of acute nephritis, though

the initial attack may have been so mild as to have escaped notice. Severe forms of toxæmic kidney, such as mercury poisoning, and the kidney of pregnancy, as also nephrosis, develop into chronic nephritis if they fail to clear up.

Pathology.—The kidneys are swollen. The capsule strips easily, leaving a smooth whitish-grey or mottled surface on which the engorged stellate veins are very obvious. On section the cortex is increased in thickness and pale in colour; the normally distinct fine radial markings are blurred; the pyramids are relatively engorged and contrast with the pale cortex. Microscopically, the glomeruli are large and irregular, with an increase in the number of nuclei, and individual endothelial cells have undergone hyaline or fatty degeneration. Proliferation of the cells of Bowman's capsule is found with crescent formation, and adhesion of the visceral to the parietal layer of Bowman's capsule. There is peri-glomerular infiltration of leucocytes, and the capillaries outside the glomeruli are engorged with blood, the glomeruli themselves being relatively bloodless. The cells of the convoluted tubules undergo cloudy swelling and fatty degeneration to a greater or less extent. Desquamation of the cells occurs, and the tubules contain hyaline and epithelial casts, or red and white blood corpuscles. In some cases of chronic nephritis the tubule changes are most marked, and the glomeruli are relatively little affected in the earlier stages of the disease. As time goes on fibrosis increases, the kidneys shrink in size, their surface becomes granular, and the capsule thickened and adherent. The kidneys are tough on section; the whole surface is a more uniform brownish colour, or in extreme cases of fibrosis, whitish-grey. The cortex is narrowed; the cut vessels may be a little prominent. Microscopically, many of the glomeruli may have undergone hyaline degeneration and fibrosis. In others, crescent formation is marked; there is increase of fibrous connective tissue around the capsules, and small cell infiltration. The tubules tend to dilate and become tortuous, and their lining cells flattened. In others there is hypertrophy of the tubules to compensate for units which atrophy and disappear completely. In some cases the vasa afferentia undergo intimal hyperplasia and fatty degeneration, and there may be hypertrophy of the media. When the blood pressure was persistently raised during life, the heart, especially the left ventricle, will be found hypertrophied. The aorta is thickened, and ordinary atheromatous changes may occur at an unusually early age.

Pathology of œdema in chronic parenchymatous nephritis.—Dropsy is one of the most characteristic features of the disease. Various explanations have been given of its causation. One of the earliest was that it was due to hydræmia from retention of water which the kidney could not excrete. But even total anuria need not cause dropsy, and Rowntree has shown that in glomerulo-nephritis the blood volume may be within normal limits. The next hypothesis was that the capillary endothelium was damaged by toxins, and therefore became unduly permeable (Cohnheim). It has been shown experimentally, however, that such damage may actually hinder the passage of fluid from the blood to the tissues. Widal attributed œdema to the defective elimination of salt by the kidney, which led to accumulation of water by raising the osmotic pressure of the tissues. But if water is retained, salt must also be retained, and when diuresis is produced, salt is excreted also. An important observation was made when Epstein showed that a feature peculiar to nephrosis and to chronic nephritis with marked

cedema was a great reduction in the protein content of the blood and exudates, almost entirely affecting the albumin, so that the amount of globulin is always increased relatively and sometimes absolutely. The daily drain on the protein may even amount to 10 per cent. of the total protein in the blood. This causes a fall in the osmotic pressure of the blood, giving the tissues the controlling power to absorb and retain fluid. In support of this view it may be mentioned that the cedema produced in perfusion experiments with normal saline or Ringer's solution is prevented by the addition to the perfusing fluid of colloids which are in osmotic equilibrium with the colloids of the lymph and tissues. A weak point in this hypothesis is its failure to explain the dropsy of acute nephritis, which comes on long before any depletion of the proteins of the blood can occur.

But the most usually accepted explanation of renal dropsy to-day is an alteration of the affinity of the tissue cells for water as the result of an altered salt metabolism, especially in respect of the sodium ions. In other words, the cedema is regarded mainly as a result of damage to the extra-renal tissues by the same agent that damaged the kidneys, rather than as a consequence of the failure of renal function.

That the blood serum in nephritis may be milky was noted by Bright, and subsequent observers have called attention to pseudochylous ascites in this disease. This is due to the increased cholesterol content, which is more marked in this type of Bright's disease than in any other, except nephrosis.

Symptoms.—These may be continued from those of acute nephritis. More usually there is an interval of apparently normal health. Then the patient begins to suffer from languor and digestive disturbances, followed by the combination of anaemia and dropsy, which gives rise to a very characteristic aspect. Hence the saying "large white kidney, large white man." The dropsy may extend to the serous sacs. The urine is scanty, probably 20 ounces or less in the day; its specific gravity is high, but urates are not so abundant as in the urine of the cardiac kidney. It contains a large amount of protein, usually about 0.5 per cent., as measured by Esbach's method. Numerous tube casts will be found on sedimenting the urine, epithelial, fatty, granular and hyaline forms all being present. Red blood corpuscles may be found from time to time. Examination of the blood may show no increase in the blood urea.

Vomiting and diarrhoea are common and troublesome. Ulceration of the colon, probably due to the vicarious elimination of toxins by the bowel, is an occasional and dangerous complication. Areas of exudate, known as "cotton-wool" patches, and cedema of the optic disks—constituting albuminuric retinitis—may be found in severe cases. There may be dyspnoea, due to acidæmia, the result of diminished excretion of acid sodium phosphate. Secondary infections of the lung, pleura, pericardium or peritoneum may occur.

If hypertrophy of the heart and a rise of blood pressure fail to take place, the outlook is very grave, and death from uræmia or secondary infection is likely to close the scene. If, on the other hand, the blood pressure rises, the heart hypertrophies, the dropsy subsides, and a more chronic stage supervenes. The output of the urine then increases, probably up to 80 oz. or more; the specific gravity being persistently low. This is due to failure of the kidney's

capacity to concentrate the urine. The quantity of albumin is very variable, but is always more than that of "chronic interstitial nephritis." Epithelial, fatty, granular and hyaline casts continue to appear unless they are disintegrated by the extreme dilution of the urine. Towards the end the secretion is sure to fail, and uræmia is likely to follow. Signs of cardiac hypertrophy can be detected, and the blood pressure is generally raised to something between 160 and 220. Albuminuric retinitis is more likely to occur now. Later, silver wire arteries, retinal hæmorrhages, which are often flame-shaped, and even glistening white patches are sometimes to be noted. Infarction of the lung may occur, causing pain, dyspnœa, hæmoptysis with signs of consolidation, and perhaps pleural friction. It results from detachment of a clot in the right auricular appendix, and, being generally due to a secondary infection, marks a definite step downwards. There may be other signs of infection, such as pericarditis or peritonitis. But in the absence of complications life may be prolonged for several years.

Diagnosis.—The combination of dropsy, anæmia, albuminuria and cylindruria generally makes the diagnosis of chronic nephritis easy. In the dropsy with albuminuria of failing heart the œdema first occurs in the most dependent parts, while in nephritis the eyelids are first affected. In cardiac dropsy the liver will probably be enlarged and tender, and the urine will be high in colour and loaded with urates; the only casts it will contain are hyaline; renal function is not seriously impaired. Amyloid kidney may be accompanied by cachectic dropsy; but the heart will not be hypertrophied and the blood pressure is not raised. Moreover, a cause for amyloid disease, and the presence of amyloid disease elsewhere, are usually obvious.

If there is no œdema the diagnosis has to be made from functional albuminuria, residual albuminuria and chronic interstitial nephritis. Functional albuminuria only occurs before thirty and generally about puberty, albumin is absent from the urine secreted in the recumbent posture, casts are absent, with the possible exception of the hyaline variety, and calcium lactate may clear up the albuminuria for a time. Residual albuminuria, an uncommon condition, is not an indication of a progressive disease. The albuminuria is detected accidentally, there being no symptoms. The blood pressure may, however, be slightly raised. There are no cells in the centrifuged deposit of urine. In "chronic interstitial nephritis" the specific gravity of the urine is very low, and there is little albumin. Unless the heart is failing there will be no œdema. The estimation of blood urica should be carried out whenever there is a question as to renal efficiency. As in acute nephritis the erythrocyte sedimentation rate is always raised.

Prognosis.—The outlook in chronic nephritis is always serious. It is essentially a progressive disease, but with care life may be prolonged for several years. Death may occur from uræmia, heart failure or secondary infections. Retinal changes make the prognosis more serious, and "woolly" exudate with swelling of the optic disc generally foretells death within two years. Retinal hæmorrhages and discrete white patches of degeneration in the retina are, however, of less serious significance.

Treatment.—It is essential, as a prophylactic measure, that the treatment of all cases of acute nephritis should be thorough and prolonged. Septic foci, especially in the tonsils, should be looked for and thoroughly treated, as also should any syphilitic or malarial infection. Confinement to

bed is only advisable during exacerbations, when dropsy is extreme, or when uræmia is threatening. The skin should always receive attention, and patients should sleep between blankets and be careful to avoid exposure to cold and wind.

Diet.—There has been a tendency to restrict the protein intake too much, since there is no evidence that the albuminuria is influenced by the amount of protein in the food. Epstein has urged, indeed, that a high protein diet is indicated in order to raise the low protein content of the blood, while fats should be avoided to diminish lipæmia. That such a diet may markedly reduce œdema is true, but not necessarily by raising the protein content of the blood. Probably the diuretic action of the urea formed from the high protein diet is partly responsible. It is, therefore, wise to estimate the blood urea and, if it is not raised, to carry out the urea concentration test (p. 1286), and only to make use of the high protein diet if this test shows at least 2 per cent. of urea. If it is below that figure the protein intake may be calculated on the basis of 1 G. of protein a day for every kilo of body-weight. Naturally, meat extracts and cellular organs, such as liver, kidney and sweetbread, should be avoided, because they contain a large amount of purin; that has to be excreted by the damaged kidney, which eliminates uric acid with difficulty. This is contrary to the principles of physiological rest but, equally, such restrictions of diet must be avoided as would lead to failure of appetite and consequent wasting, while incapable of diminishing the albuminuria. A much greater variety of diet than is usually allowed might be permitted; cooked eggs and dishes made from eggs may certainly be taken. Raw eggs, however, contain certain indeterminate substances which may irritate the kidney. The distinction drawn between red and white meat is fallacious. Red meat is assumed to be more injurious, presumably because it is supposed to contain more purin, whereas the reddest meat contains far less than sweetbread. Chronic nephritis should not be restricted to milk, which is too dilute a form of food for them, and may increase the dropsy. An entirely salt-free diet is not to be recommended, though moderate restriction in this respect is probably wise. Salt can be replaced by lemon juice or ruthmol. In this way we can avoid increasing the miseries of an incurable disease by unnecessary restrictions. If nitrogen retention exists as shown by estimations of blood urea, a diet poor in protein should be taken on one day in each week. Indeed, a day when the diet is restricted to fruit and sugar is often as useful in chronic nephritis as is the day of vegetable and egg diet in diabetes. But prolonged nitrogen starvation is as bad for the nephritic as for any one else, and in some cases increases the water-logging of the tissues.

Generally speaking, alcohol is inadvisable in any form, and should never be ordered to those unaccustomed to it. In those who have been taking it regularly, deprivation may interfere with appetite, in which case a little well-diluted whisky is probably as innocuous as any form of alcohol can be. But the strictest moderation must be enjoined. Tea and coffee used to be forbidden, because of the methyl-purins they contain, but in our opinion this restriction is unnecessary.

Diuretics.—Diuretics should be used with caution in chronic nephritis. When there is marked œdema the fluid intake should be limited to a litre or perhaps 2 pints in the 24 hours, but the patient should not be allowed to be thirsty. In some cases the administration of potassium salts by mouth acts

as an efficient diuretic (*vide* treatment of nephrosis). Saline diuretics in the form of citrates and acetates may be safely given, provided that the dose is moderate, and that the possibility of alkalosis developing in severely damaged kidneys is remembered. Urea may be given in those cases in which the blood urea is low, and in which the danger of a rising blood urea is not anticipated. The caffeine group of drugs should be employed with caution, and when used they are best given in small doses, and then withheld if diuresis does not result. Theophylline and sodium acetate grs. 4, or theobromine and sodium salicylate (diuretin) grs. 10, administered twice or three times daily may be prescribed if there is no hæmaturia. In general terms organic mercury preparations are contra-indicated in chronic nephritis on account of the danger of mercury-poisoning. Nevertheless, in cases in which œdema is persistent, renal function is adequate and there is an absence of anæmia, injection of mersalyl (salyrgan) may sometimes be used with advantage. Sixty or 120 grains of ammonium chloride are given on each of 2 days before $\frac{1}{2}$ to 1 c.c. of the drug, and, if tolerated, this treatment is repeated at intervals of 5 to 9 days. Œdema in chronic nephritis may be in part due to heart failure. In this case digitalis may prove a valuable and efficient diuretic. A raised blood pressure is not a contra-indication to its use.

A marked feature of chronic nephritis is the defective adjustment of the kidneys to varying water supply. As in acute nephritis, the drinking of large amounts of fluid may, therefore, merely increase the œdema.

As explained under Acute Nephritis, the saline diuretics are the least open to objection; since they draw the extra water from the tissues they cannot increase and may diminish the œdema. Urea as the natural diuretic of the body is often given in doses of 45–60 grains three times a day, when there is no nitrogen retention.

Diaphoretics.—The arguments for and against diaphoretic measures will be found under uræmia. Diaphoretic drugs are not suitable for the routine treatment of chronic nephritis, as a moist perspiring skin renders the patient more liable to chills—always a danger in this disease. A course of hot-air baths may sometimes be helpful when there is evidence of salt retention. If they are followed by diuresis they are doing good.

Purgation.—Although efficient action of the bowels must be maintained, habitual loose stools are to be avoided, because they weaken the patient and promote the absorption of intestinal toxins. The special liability to mercurialism renders calomel unsuitable for routine treatment.

Acupuncture.—The patient is placed in a cardiac bed with the head raised and feet lowered for a day or two before acupuncture is performed so that the fluid gravitates into the lower limbs. The preparation for acupuncture consists of a mackintosh sheet placed under the lower limbs, and arranged to form a chute leading into a pail on the floor: the lower limbs from the knees to and including the toes are cleaned with ether soap, and then with spirit, and placed on sterile towels: the skin is covered with a coating of Lassar's paste. Acupuncture is performed with a medium-sized trocar from a Southey's tube set, the trocar being stabbed through the paste and skin to the subcutaneous tissues. Beginning at the side of the tendo Achillis, 15 punctures are made in line posteriorly in the lower two-thirds of the leg. Two lines of punctures about one inch apart are made in each

leg. Each leg is then wrapped in a piece of sterile jaconet, wrapped firmly round the thigh immediately above the knee, and secured with adhesive tape. Below the jaconet is wrapped loosely round the limb forming a tube which conducts the fluid into the mackintosh gutter at the foot of the bed. Drainage may continue for a week. By this method fluid may even be drained from serous cavities when there is marked dropsy.

Decapsulation.—This procedure is now reserved for the treatment of œdema in the nephrotic type of Bright's disease if adequate treatment on conservative lines has failed to relieve it.

Climate is a valuable help. In this country, Ventnor or anywhere on the south coast from Bournemouth westward is the most suitable climate that can be obtained. Egypt generally suits such patients particularly well. Madeira or California are also quite suitable. The wind and the more violent fluctuations of temperature on the Riviera render it much less advisable.

Treatment of complications.—These are uræmia, heart failure and secondary infections, such as pericarditis, pleurisy, colitis and peritonitis. Their treatment is discussed under those headings.

In conclusion, it must be recognised that the kidney, once chronic nephritis is established, cannot completely recover, and the main thing is to attune the mode of life to a low key, subjecting the patient to as little strain as possible. He may have a considerable variety of food, provided that the intake of protein is regulated in the way described above, and that he takes very little purin and salt. He can be helped by saline diuretics and un-irritating preparations of iron, such as liquor ferri acetatis. He will do all the better if his medical man realises that many of the methods recommended in the treatment of this disease are impotent, where not actually harmful.

(b) PRIMARY TYPE—RENAL DYSBIOTROPHY

Chronic nephritis may develop without any known cause. In such a case there is no past history of acute nephritis; of symptoms of Bright's disease, such as œdema, hæmaturia or pain in the back; nor indeed any history of infection, such as scarlet fever or tonsillitis to which the onset of the disease can be attributed. The complaint may be found accidentally by the discovery of albuminuria in the course of routine examination. More often the diagnosis is first made at a later stage of the disease when there is complaint of asthenia, anæmia, or liability to fatigue. Sometimes, indeed, there are no symptoms of the disease until it has reached the terminal stage of uræmia.

Ætiology.—This form of chronic nephritis probably belongs to the group of congenital-developmental diseases of inborn and familial type, as defined by Parkes Weber. According to this view certain forms of chronic nephritis are inborn constitutional diseases which, though they may manifest themselves soon after birth, may sometimes be delayed in their appearance until years later. When the disease runs its course and ends fatally within a few weeks or months of birth, there can be no doubt of its having been present in utero. When it does not manifest itself until after many years of antecedent good health, we may assume that the disease was only potentially present at birth, the inborn tendency to its development being due to a congenital tissue inferiority or dysbiotrophy. The reasons for this view are : (1) the absence of a discoverable ætiology already referred to ; (2) the not

uncommon familial occurrence of the complaint; (3) the finding post mortem of congenital abnormalities in the kidneys or urinary tract; and (4) that in its course, which is latent, progressive and invariably fatal, the malady resembles many other diseases which belong to this congenital-developmental class.

Familial hæmorrhagic nephritis, which is also hereditary and apparently congenital, is a rare condition. In one family 16 cases occurred among 28 individuals in 3 generations, 8 being in males, 7 of which ended fatally in early life, and 8 in females with only 1 death. The disease evidently runs a more benign course in the female. In one patient the first symptom occurred when only 3 weeks old, in another when 2 years old, while in the remaining cases the onset was quite early in life. Most of the cases have been characterised by recurrent attacks of hæmaturia, sometimes considerable in amount. As such attacks may be preceded by an increased nitrogen output, some inherited form of protein sensitivity seems a probable factor. As deafness may also be familial, it is interesting to note that it was marked in 5 of the 16 patients, and one otherwise healthy member of the family was also deaf. The condition usually terminates in uræmia.

Pathology.—The kidneys are small, pale and fibrosed. One kidney may be smaller than the other. Congenital deformity is not uncommon: occasionally one kidney is absent or represented by only a nodule of fibrosed tissue, from which an atrophied ureter has its origin. On microscopic examination there is diffuse fibrosis of the kidneys; many glomeruli are atrophied, others show varying degrees of inflammatory reaction.

Symptoms.—In the rare cases that appear in infants of a few weeks or a few months old, the symptoms are those of uræmia, namely, dyspepsia and loss of weight leading to diarrhoea, vomiting and cachexia. In those that develop about the age of puberty the disease may cause infantilism (renal dwarfism); and bone deformities resembling rickets often develop, and may be associated with a low calcium content of the blood, leading in some instances to a compensatory enlargement of the parathyroids. In one group of cases there is cardiac hypertrophy, a considerably raised blood pressure, and retinitis. In these arteriolar changes (diffuse hyperplastic sclerosis) are found post mortem. In others the blood pressure is normal or subnormal. In another group (first described by Rose Bradford in 1904) the disease makes its first appearance between the ages of 20 and 30 years. It may appear suddenly in the form of uræmia without warning, and become rapidly fatal. Even in those cases in which the kidneys are found post mortem to be white, fibrotic and greatly reduced in size, with diffuse inflammatory changes of long standing, early adult life may have been apparently perfectly healthy, and symptoms of the disease may have been present for only a few months.

Differential Diagnosis.—Loss of weight, a sense of fatigue, anæmia and dyspepsia, may have been so marked as to raise the question of tuberculous disease or neoplasm. When there is marked asthenia and pigmentation, Addison's disease may be suggested, or the differential diagnosis may lead to the suspicion of pernicious anæmia. In cases with high blood pressure a juvenile form of hyperpiesia must be excluded. When there is marked polyuria with urine of low specific gravity, the possibility of diabetes insipidus arises.

Treatment.—The disease is essentially progressive, and treatment can only be symptomatic.

CHRONIC INTERSTITIAL NEPHRITIS, INCLUDING
MALIGNANT NEPHROSCLEROSIS**Synonym.**—Granular Kidney.

When this term was first used it included a number of clinical entities, which together formed a somewhat motley group. Among these certain forms of chronic nephritis have now been identified as such, particularly the type recognised by Rose Bradford as the small white kidney, and classified by us under chronic nephritis, primary form. Clifford Allbutt identified hyperpiesia, Samuel West and others recognised the senile kidney. Some authors are inclined to think that after excluding these clinical forms only a single entity, malignant nephrosclerosis, remains. "Chronic interstitial nephritis," however, probably still includes more than one clinical entity, and it is for this reason that we have retained the term.

Ætiology.—The causation of malignant nephrosclerosis is unknown, but probably those factors which apply to hyperpiesia already described are concerned. Malignant hypertension, however, tends to make its appearance at an earlier age, and is not uncommon at any age after 35 years, and is frequent between the ages of 40 and 55. According to Ellis it is more common in men than in women. As to the other cases included under the term "Chronic Interstitial Nephritis," the ætiology is unknown, but gout, lead poisoning and alcohol are thought by some to be factors. Syphilis is not a cause.

Pathology.—The kidney tends to be reduced in size, is tough, and red in colour. The capsule is adherent, leaving a finely granular surface on stripping. Sometimes the capsule is thickened and splits on attempting to strip it, thus giving an erroneous impression of a smooth surface. Retention cysts may be seen, both on the outside and the inside of the organ. On section the cortex is reduced; not only is it shrunk from without inwards, making the organ smaller, but the increase of intrapelvic fat shows that it has also shrunk from within outwards. The vessels are unduly prominent. The glomeruli show signs of inflammatory reaction, and the interstitial tissue in their neighbourhood is increased and infiltrated with small cells, generally of the mononuclear type. These areas of disease are patchy, and form wedge-shaped areas, with their apex towards the cortico-medullary zone. The intervening areas of renal tissue show little or no change. The histological lesions in the arteriæ interlobulares and vasa afferentia, as also the patchy small-cell infiltration in the kidneys and evidence in some glomeruli of proliferation of the cells of Bowman's capsules, provide evidence of inflammatory reaction. The cardiovascular changes are those described under hyperpiesic kidney (benign nephrosclerosis). In addition a characteristic lesion is acute arteriolar necrosis, while focal necrosis of the capillary tuft is common. Hæmorrhagic infarction of some glomerular tufts may be found. The distribution of the renal lesion in chronic interstitial nephritis is distinct from that in chronic nephritis, for in the latter the lesion is diffuse and every glomerulus is more or less altered in structure. In advanced cases of chronic interstitial nephritis, however, the extent of structural alteration extends and becomes more diffuse.

Symptoms.—The subject of chronic interstitial nephritis often fails to seek medical advice until the condition has become well advanced, and this

is partly because the body adjusts itself to the changes which are taking place in it as a result of the disease, and partly because the disease may develop very rapidly in its early stages. Common early symptoms are loss of energy, proneness to fatigue, lack of power of concentration, headache and dyspepsia. In other cases the patient feels well, and consults his doctor on account of hæmorrhage. The source of the bleeding is most commonly from the nose (epistaxis), or frank hæmaturia. In other cases it may be a cerebral vascular accident, thrombotic or hæmorrhagic: less often it takes the form of hæmatemesis or melæna. Disturbance of vision due to retinal hæmorrhages, or sudden blindness due to hæmorrhage into the vitreous are not uncommon early symptoms. A woman may complain of bleeding from the uterus, and rarely a blood-stained seminal discharge is the initial symptom in a man. In other cases the initial symptoms are cardiac, and the patient complains of cardiac pain, shortness of breath, or consciousness of the heart's action. Nocturnal frequency of micturition may be an early symptom. On examination the patient will be found to have an increased blood pressure, the systolic ranging between, say 200-240 mm. or even higher, and the diastolic over 120 mm. There is also cardiac hypertrophy, but the case differs from a hyperpietic kidney (benign nephrosclerosis) in one or more of the following ways. Thus, the complexion tends to be sallow, and often there is loss of weight and some degree of secondary anæmia. The urine may contain only a trace of albumin, but more frequently it contains an appreciable quantity, namely 0·1 per cent. or more. The urinary deposit characteristically contains red blood corpuscles and granular and hyaline casts. When the disease is fully established, the urine has a low fixed specific gravity, varying between 1008 and 1012. Further examination may show impairment of renal function, and the blood urea may be raised to 70 mgm. per cent. at least. On examination of the eyes, in addition to arterio-sclerosis of the retinal vessels, retinal hæmorrhages and exudates, cedema of the disc and retina, and some detachment of the retina may be found. Ellis has emphasised the diagnostic importance of papilledema in malignant nephrosclerosis.

There are cases of chronic interstitial nephritis which run a most rapid course in the initial stage. The clinical features are hypertension, cardiac hypertrophy and hæmorrhage. Toxic symptoms make their appearance within 3 to 6 months of the onset, and by this time some degree of failure of renal function may be demonstrated by clinical tests, or the patient's symptoms may already be those of uræmia. The whole evolution of the disease and its fatal termination in uræmia, cerebral hæmorrhage or cardiac failure, may take place within a period of 12 to 18 months.

There are other cases in which there may be a preceding period of five or more years during which time the patient is known to have hyperpiesia, and complains only of cardiac symptoms which are readily explained in terms of it. Meanwhile the disease-process seems to be more or less stationary, and then for no obvious reason the disease takes a progressive form, and a terminal acute phase lasting only 3 to 6 months is ushered in by an attack of hæmaturia or epistaxis, by retinitis, or by symptoms of renal failure, and the toxic picture of chronic interstitial nephritis rapidly develops.

On the other hand, there are chronic forms of the complaint which pursue a varied course different from the grave progressive course of malignant hypertension. The variety of the clinical picture which these cases present

makes their description difficult, and it is to allow of their recognition, and in order to leave the subject open for further clinical and histological observation, that the term "chronic interstitial nephritis" has been retained. Thus, a man complains of malaise, loss of health and strength, undue fatigue or loss of weight. On examination, the chief finding is an appreciable albuminuria. At this stage there may be no abnormal cellular deposit, or there may be a few red blood corpuscles and hyaline casts in the urinary deposit. Renal function may be normal. With limitation of his activities and general measures of medical treatment, the patient may recover his health to a considerable extent, though the albuminuria persists. From now on, over a period of 2 to 5 years, or perhaps longer, reasonably good health is maintained. Sometimes the blood pressure is but little raised. There may be an attack of hæmaturia or epistaxis from time to time. The blood urea may be a little raised, and may even remain more or less stationary at a level of 60 to 80 mgm. urea per cent. for several years, until finally physiological adjustments fail, and uræmia, a vascular accident, cardiac defeat or an intercurrent infection determines the fatal termination.

Complications.—The principal complications are due to failure of the pump, the tubing, or the filter. In other words, the heart may fail, causing venous congestion; the artery may give way, as in cerebral hæmorrhage; or the renal excretion become so inadequate as to lead to uræmia. Glycosuria is sometimes found. This may be due to alcoholic excess, a factor in the causation of the interstitial nephritis.

Diagnosis.—This rests on the combination of urinary and cardiovascular signs. The differential diagnosis from hyperpiesia is discussed on p. 1068, and that from secondary contracted kidney on p. 1312.

Prognosis.—The disease is usually progressive. Its course may be very slow, but after the development of retinitis a fatal ending occurs in the great majority of cases within 2 years. In some cases, however, even including those in which all the characteristic signs of the disease are present, including microscopic hæmaturia, persistent hypertension and retinitis, the disease may reach a stationary phase, and considerable recovery may occur, the fatal termination being delayed on occasion for as long as 6 years. For this reason a definite prognosis should not be given until a late stage in the disease. Tests of renal function afford useful prognostic evidence. Any evidence of cardiac dilatation, or of uræmia, even of the chronic variety, makes the outlook much less satisfactory. Retinal changes are of ill-omen.

Treatment.—In general terms, this should be carried out on similar lines to that for hyperpietic kidney (*q.v.*), though the response is not likely to be satisfactory. In addition, the anæmia often calls for treatment.

HYPERPIETIC KIDNEY

Synonym.—Benign Nephrosclerosis.

In this form of renal disease the vascular changes are of greater importance than the renal, and it will only be necessary to make a brief reference to it here.

Ætiology.—This is the same as that described under Hyperpiesia (see page 1065).

Pathology.—The chief kidney changes are in the small vessels. They consist of thickening of the intima and medial hypertrophy. The former may go on to fatty degeneration and obliteration of the lumen of the vasa afferentia, and so cause ischæmic fibrosis of the glomeruli with atrophy of the associated tubules. Thus, the changes in the renal parenchyma are largely degenerative in character, rather than the inflammatory changes seen in chronic interstitial nephritis. The kidney is slightly reduced in size and is somewhat firmer than normal. On section, the fine radial striation in the cortex is preserved, and throughout the organ the small arteries are prominent. Histologically the essential lesion is a thickening of the intima of the vasa afferentia and the interlobular arteries, with hypertrophy of the media. In the early stages there is cellular proliferation in the intima and increase of hyaline material. At a later stage there is fatty degeneration in the terminal arterioles in contrast to their parent vessels, in which little or no fatty degeneration is found. The thickening of the intima may lead to obliteration of the lumen, with fibrosis and atrophy of the glomerulus and its tubules. At a later stage, too, owing to fatty degeneration and atrophy of the muscle fibres of the media, the media may be actually thinner than normal. These changes, like those described in chronic interstitial nephritis, have a patchy distribution in the organ. The fibrous connective tissue in the immediate neighbourhood is thickened, but there is no glomerulitis and little or no small-celled infiltration, in contrast to the inflammatory reaction found in chronic interstitial nephritis. The vascular changes described above were originally termed arterio-capillary fibrosis by Gull and Sutton. They were first accurately described by Jores under the term diffuse hyperplastic sclerosis.

Symptoms.—These are those of hyperpiesia (see pp. 1066, 1067).

Treatment.—See that of hyperpiesia (pp. 1068–1070), the heart in hypertension (p. 996), and renal uræmia (pp. 1327, 1328).

SENILE OR ATHEROMATOUS KIDNEY

In this form of kidney disease also the vascular changes are of greater importance than the renal, and it is only necessary to deal briefly with the affection.

Pathology.—The kidneys show depressed red areas, which are due to contraction of fibrous tissue along the distribution of particular interlobular arteries, and, therefore, tend to be conical in form, with their base to the surface of the organ. There is an absence of cardiac hypertrophy; the pressure in the diseased arteries falls below that necessary for glomerular excretion. The affected glomeruli accordingly shrink, and the connective tissue around them becomes condensed and thickened. The degenerate glomerulus and its capsule fuse together, and undergo fatty and fibrotic changes. The atheromatous kidney is, therefore, generally due to atrophy following insufficient circulation, with consequent fibrosis.

Symptoms and Diagnosis.—There may be gradual failure of the physical and mental powers—described by Allbutt as “contraction of the spheres of bodily and mental activity”—rather than the more dramatic events of chronic interstitial nephritis. There is a trace of albumin in the urine. The radial artery is thickened and tortuous. The blood pressure is not high, and

there is an absence of cardiac hypertrophy. Death by cardiac failure or intercurrent affections is the commonest ending, while cerebral hæmorrhage and uræmia are unlikely.

URÆMIA

Uræmia is the name which has been given in the past to the toxic state which complicates or terminates severe kidney disease, and in which urea retention occurs. More recently it has been recognised that a high grade of urea retention may develop as a result of extra-renal factors. It is proposed to group these conditions under the name of Extra-renal Uræmia in order to emphasise the fact that although the kidney fails in its function, organic disease of the kidneys is not the primary fault.

EXTRA-RENAL URÆMIA

ALKALOSIS (see also Alkalosis, pp. 405-408).—This is a toxic state characterised by malaise, gastro-intestinal disturbance, and a variety of nervous symptoms due to an increase of the CO_2 combining power of the blood plasma.

Ætiology.—Alkalosis is caused by giving too large doses of sodium bicarbonate or other alkali. The minimum normal tolerance of alkali by mouth seems to be the equivalent of 15 g. of sodium bicarbonate in 24 hours. If a patient develops alkalosis when taking this quantity of alkali or less, some predisposing factor will be found. Such factors are anæmia, kidney disease, or vomiting. Of these factors anæmia is important, because of the action of hæmoglobin as a buffer helping to maintain a constant pH in the blood. With a low hæmoglobin content in the blood an extra tax is thrown on the kidneys, so that in anæmia a smaller quantity of alkali given by the mouth may be the cause of alkalosis. Impaired renal function, such as results from kidney disease, is another factor predisposing to alkalosis, because the kidneys are unable to excrete the excess of alkali. As alkalosis may itself be responsible for kidney damage a vicious circle is set up. It has been suggested that chloride loss and the consequent hypochloræmia is the cause of the rise in blood urea. This explanation cannot, however, account for the whole condition, because there are other conditions in which there is a considerable loss of blood chlorides without uræmia. The uræmia which develops in repeated vomiting, however, is probably due to loss of hydrochloric acid in the vomit.

Symptoms.—The toxic symptoms caused by giving too large doses of sodium bicarbonate or other alkali appear from 4 days up to 4 weeks from the beginning of treatment (Cooke). The patient complains of malaise, dizziness, constipation and headache, which commonly takes the form of a sensation of pressure on the vertex. He becomes nervous and irritable, often resentful. There is loss of appetite, distaste for food, nausea and vomiting. The patient becomes drowsy in the day and sleepless at night. Respiration is slowed, the pulse rapid, the face flushed, and the body perspiring. There is aching or actual pain in the trunk and limbs, with tenderness of the muscles on pressure, and increased muscular irritability. In the severest cases there may be tetany or epileptiform convulsions, and if the condition is unrecognised

the patient may become comatose, with incontinence of urine and *fæces*, and die. In addition to the above symptoms, a dry furred tongue, thirst, and at a later stage diarrhoea are common. The urine is alkaline, except sometimes in the early stages. It contains a trace or cloud of albumin. The deposit may contain hyaline or granular casts, and a few red and white blood corpuscles. Renal function is impaired, as is shown in nearly all cases by urea retention, and in some by failure of the power of urine concentration, which results in the excretion of urine of a constantly low specific gravity. There may also be a considerable polyuria, amounting to 2 or 3 litres in 24 hours. In most cases reported the dose of alkali which has caused alkalosis has been the equivalent of 20 g. of sodium bicarbonate given daily for 4 days or more. More usually the toxic dose has been the equivalent of 30 to 60 g. of sodium bicarbonate daily, and larger doses than this are naturally all the more likely to produce the condition.

Diagnosis.—Alkalosis must be suspected whenever a patient taking alkali develops symptoms of malaise, headache, constipation, digestive disturbance, or change in the personality. The diagnosis is established by an examination of the blood. The blood urea is commonly raised to 60 or 80 mgm. per cent., and may reach the high figures found in renal uræmia. The alkali reserve, which normally varies between 50 and 75 c.c. CO_2 per 100 c.c. of plasma, increases to 90 c.c. or more. The chloride content of both blood and urine may be low. The CO_2 combining power of the plasma is the most important observation to make, because it precedes the rise of blood urea.

Treatment.—The administration of alkalis is immediately discontinued. Acid sodium phosphate, gr. 10, three times daily after food is administered. If the case is under close observation, and if repeated estimations can be made of the alkali reserve, larger doses may be prescribed, but on account of the danger which exists, owing to renal damage, of a rapid change from alkalosis to acidæmia, some authorities are opposed to giving acid in any form. In any case, it is unwise to prescribe ammonium chloride.

GASTRO-RENAL URÆMIA

Repeated vomiting from any cause may induce alkalosis and thus be responsible for uræmia. In acute cases with albuminuria the differential diagnosis from renal uræmia is made clinically on the high specific gravity and high urea content of the urine. In chronic cases a complete blood examination and the observation of the degree of alkalosis may be required to exclude renal uræmia.

Diarrhoea may be responsible for uræmia, both on account of the loss of fluid and the resultant loss of chloride. But in this case the uræmia is associated with the opposite condition of acidosis, as may be established by the estimation of the alkali reserve. In acute cases, treatment is by intravenous injection of 200 to 400 c.c. of a 2 per cent. sodium bicarbonate solution.

RENAL URÆMIA

This type of uræmia belongs to a different category from that of Extra-renal Uræmia, because it is primarily due to severe kidney disease of which it is a complication or a terminal phase. A raised blood urea is the distinguish-

ing feature of uræmia. Nevertheless, the term renal uræmia is used in a clinical sense, both because the diagnosis can be made without an estimation of the urea in the blood, and because, on occasion, the patient may suffer from uræmia before the blood urea is appreciably raised. Renal uræmia varies greatly in its symptomatology, but, as Clifford Allbutt pointed out, it is generally characterised by anæmia, headache, nausea, lethargy, retinitis, convulsions or coma.

Although there are many factors in support of the so-called retention theory of uræmia, its complete explanation in biochemical terms is not yet clear.

In view of the high blood urea in severe uræmia, urea retention might be regarded as the simplest explanation of the condition. It has been shown experimentally that the administration of massive doses of urea causes headaches, giddiness, apathy, drowsiness, bodily weakness, nausea and diarrhœa—a group of symptoms characteristic of chronic uræmia. But that this is not the whole explanation of uræmia is shown by the fact that symptoms of uræmia may be present before the blood urea has risen to a level proportionate to the symptoms. It has been suggested, therefore, that other renal excretory products, such as indican, uric acid and the salts of urine, may contribute to the uræmic state. When the hypobromite method is used for the estimation of urea in the blood or cerebro-spinal fluid other nitrogenous products than urea are estimated. The urease method gives the amount of urea only, so that the difference between the results obtained by these two methods is an indication of the quantity of amine bodies other than urea, and to these bodies some at least of the clinical syndrome is probably due. But even this expansion of the retention theory to include nitrogenous bodies other than urea does not explain uræmia, because in complete suppression of renal function, such as occurs when the ureter of a single kidney is completely destroyed by any cause (Ascoli's *urinæmia*), the clinical picture is entirely different from that of uræmia complicating acute and chronic Bright's disease. Ascoli says: "Severe *urinæmia* in man is chiefly manifested by bodily weakness and languor, which often appear before any other symptoms, but generally lead to progressive mental weakness and exhaustion, often terminating with great suddenness. The greater part of the most prominent symptoms of uræmia are, however, lacking, especially the severe and acute mental disturbances, the sudden amaurosis, and the epileptic phenomena in general. Only in occasional cases do the symptoms resemble uræmia." The name of *latent uræmia* is sometimes given to this condition, but it is hardly suitable. It has also been held in the past that uræmia may be due to some precursor or derivative of urea or other nitrogenous body normally secreted by the kidneys, but there is no convincing evidence of the accumulation of such bodies in the blood at least in such quantity as to cause uræmia.

It is evident that the retention theory and a failure of the excretory function of the kidneys do not provide a complete explanation of renal uræmia. But the kidneys have two other important functions, namely, the regulation of the osmotic pressure of the blood and the regulation of its hydrogen-ion concentration. Now, in uræmia the acid-base equilibrium is disturbed, and there is a fall in the CO_2 combining power of the blood plasma; that is to say, *acidæmia*. If this state of *acidæmia* is corrected by the administration of alkali in such dosage as to restore the CO_2 combining power of the

plasma to normal, the patient may be relieved of the more acute symptoms of uræmia, and may be maintained symptom-free for several weeks or months. Or, if the uræmic state is the complication of a recoverable disease, as in the uræmia of acute nephritis, or the uræmia complicating or following operations on the urinary tract, the restoration of the acid-base equilibrium in the blood may save the patient's life. The fact is that when diseased kidneys fail in their function of maintaining acid-base equilibrium, and even when the disease is not so severe as itself to have determined the onset of uræmia, the administration of alkali or acid in excess readily causes uræmia. Further, both acidæmia and alkalosis in themselves cause renal damage, and thus aggravate pre-existing renal disease. In this way a vicious circle is set up.

In order to realise fully the way in which a grave disturbance of the acid-base equilibrium of the blood caused by giving too much alkali to a patient with presumably previously healthy kidneys, the reader is referred to alkalosis. In a severe case of alkalosis the clinical syndrome may be indistinguishable from renal uræmia, including albuminuria, low specific gravity urine, cylindruria, high blood urea, polyuria, thirst, lethargy, loss of appetite, nausea, vomiting, constipation, diarrhœa, coma, convulsions and death.

There is another approach to the interpretation of some of the symptoms of uræmia. Symptoms similar to those occurring in the cerebral type of uræmia may be produced by a disturbance of the cerebral circulation due to œdema of the brain, or to spasm of one or more of the cerebral arteries without a rise in the blood urea. In either case a raised blood pressure is the determining factor. In the acute cases, headache, vomiting and bradycardia are the important clinical features, and because of the rise of blood pressure which characterises the attack, it is described as a hypertensive cerebral attack. Even in cases with a high blood urea the cerebral symptoms may be due to disturbance of the cerebral circulation, and treatment by lumbar puncture or venesection, according to the indications provided by the particular case, may abort or cut short the attack. (*Vide* Diagnosis.)

It may be concluded that some of the symptoms of renal uræmia are due to a toxæmia acting on the nervous system caused by the abnormal metabolic products resulting from inadequate excretion by a diseased kidney. An important part of the clinical syndrome is due to a failure of the kidneys to maintain the normal hydrogen-ion concentration of the blood. The acidæmia that results is in part due to a failure of the kidneys to excrete acid phosphates (Marriott and Howland). Other symptoms, such as increased nerve excitability and localised muscular twitchings, have been attributed to a fall in the blood calcium by de Wesselow, and Izod Bennett compares such twitchings with those of tetany. Lastly, the disturbance of cerebral circulation, whether due to cerebral œdema or vascular spasm, may play a part. On these several lines we are approaching an adequate explanation of the uræmic syndrome. Lastly, account must be taken of the possibility of the secretion of a pressor substance by ischæmic kidneys being a factor in the causation of the uræmic state.

Symptoms.—A convenient clinical classification of the types of uræmia is : (1) *Cerebral* in the fulminating and acute cases ; (2) *Respiratory* where acidæmia is predominant ; and (3) *Gastro-intestinal* in the chronic cases. The terms acute and chronic apply to the uræmia and not to the disease responsible for it. But each of these types is really nervous in origin. Usually the first

type begins with severe headache. Drowsiness and twitchings of the face and hands follow. The twitchings may become aggravated into epileptiform convulsions, and the drowsiness may deepen into coma, ending in death. But several important departures from this course may occur. Sudden loss of vision, amaurosis, is not infrequent, although the fundi may not show the changes characteristic of albuminuric retinitis. Local palsies, hemiplegia or monoplegia, may come on spontaneously or after a convulsion, and are frequently due to small vascular lesions. Intense itching of the skin, tingling and numbness of the extremities, muscular cramps or insomnia may usher in the more serious symptoms. Sudden mania or delusional insanity may be the first and a very misleading symptom. The cerebral type is often rapidly fatal, but convulsions and amaurosis, though more striking, are less grave than the other symptoms. In the epidemic of war nephritis we saw seven instances of uræmic convulsions with complete recovery from the nephritis.

The commonest respiratory symptom is dyspnoea, often paroxysmal, to which the name of uræmic asthma is given. It is associated with a fall in the CO_2 of the alveolar air from the normal 5 per cent. to 3 per cent. or lower. There is diminished alkalinity of the blood, from the presence of some non-volatile acid. Addison called attention to the hissing character of the respirations in this condition. In all types of uræmia there is a tendency to stomatitis, and this is perhaps particularly so in uræmic asthma. This combination of dyspnoea of a hissing character in a drowsy patient with bleeding gums often characterises the terminal phase of uræmia. At first there may be no signs in the chest except the ordinary cardiovascular signs of chronic nephritis, but as the attack proceeds there are usually abundant moist sounds from the onset of cedema of the lungs. The heart fails, the patient becomes steadily waterlogged, slipping down into the bed from the orthopnoæic position as he becomes more and more drowsy. The fatal issue may not occur in this way, however, but from development of some of the more acute nervous symptoms.

Less common than this type of dyspnoea is Cheyne-Stokes' respiration. The whole of the cerebral functions may then show a curious periodicity; thus the pulse quickens during the noisy breathing, the pupil dilates, the patient becomes more conscious and restless. As the apnoæic pause succeeds, the pulse slows down again, the pupil contracts and the patient becomes quieter or even comatose.

The gastro-intestinal symptoms are nausea, hiccough, vomiting and diarrhœa. The gastric part of these symptoms may be very chronic. Any practitioner who neglects systematic examination of the urine will sooner or later treat a case of uræmia as one of simple dyspepsia. Apart from the urine, there is, however, one significant point: the dyspepsia may improve under treatment while the vomiting persists. In simple dyspepsia vomiting is never the last symptom to clear up. It is stated that this vomiting has no relation to meals, but this is far from being invariably true. Vomiting may occur only then, and so the mistake is made. In severer cases the vomiting may be quite uncontrollable, when the prognosis becomes correspondingly grave.

Attacks of diarrhœa are not uncommon in chronic nephritis and are not in themselves significant of uræmia. The amount of nitrogenous excretion

occurring by the bowel, when urinary elimination is inadequate, irritates the intestine and leads to the so-called albuminuric ulceration. Another explanation of this condition is that hæmorrhages which occur here as elsewhere in chronic nephritis are the precursors of the ulceration. There may also be an intense catarrhal or even "diphtheritic" colitis. Here, therefore, there are local lesions sufficient to account for symptoms usually referred to uræmia, for such lesions are conspicuously absent at least in the asthmatic and gastric syndromes. It is accordingly inadvisable to call these symptoms uræmic, as is generally done. At any rate the term should be confined to those violent choleraic attacks which are out of all proportion to the local lesions. Both the vomiting and diarrhœa are sometimes regarded as an attempt at vicarious elimination of toxins. The fact that the vomit may contain a higher percentage of amines than the blood certainly suggests this, but it must not be forgotten that either vomiting or diarrhœa may so alter the pH of the blood as to aggravate existing renal damage. It is this alteration which may actually determine the onset of uræmia.

Diagnosis.—This brief account of the symptoms of uræmia will indicate also some of the pitfalls besetting diagnosis. When the patient is known to have had Bright's disease, or indeed any disease or injury to the urinary system, the possibility of uræmia will arise. The finding of hæmaturia, albuminuria, cylindruria, low specific gravity urine, bacilluria or pyuria, will indicate urinary disease. The estimation of the blood urea and CO_2 combining power of the plasma will often be necessary to establish the diagnosis.

The first differential diagnosis to make is between extra-renal and renal uræmia. The causes of extra-renal uræmia must be inquired into, particularly as to whether the patient is taking alkali. Anæmia is a predisposing factor. The importance of vomiting and diarrhœa in causing extra-renal uræmia must be taken into account. In acute extra-renal uræmia, such as may be due to vomiting, the concentration of urinary urea is high. In chronic cases, however, secondary kidney damage may lead to polyuria, low specific gravity urine, and low urinary urea even in extra-renal uræmia.

Certain conditions which clinically resemble uræmia are separated from it under the term *pseudo-uræmia*, because they have a different pathology and belong to a different order of clinical events. The commonest cause of pseudo-uræmia is cardiovascular disease. Under this heading are to be included the convulsive seizures and varied evidence of cerebral disturbance in arterio-sclerotic subjects in whom the symptoms are due to vascular lesions, sometimes limited to capillary areas, in the brain. Heart failure, when responsible for cerebral disturbance, nocturnal dyspnoea, Cheynes-Stokes' breathing, and on occasion psychosis, belongs to the same order of events, and has to be distinguished from true uræmia. It may be a matter of considerable difficulty to distinguish between cardiovascular disease in which the right side of the heart is failing, and in which there is renal congestion with albuminuria, microscopic hæmaturia and cylindruria—a difficulty which may be increased by the fact that the blood urea may rise to 80 or 100 mgm. per cent. in congestive heart failure. Further, in true or renal uræmia cardiac failure may be an important complicating factor. In simple congestive heart failure the urine is loaded with urates, its specific gravity is raised, twitches do not occur, and the blood urea is normal or inconsiderably raised. Disorder of the cerebral circulation, whether due to cerebral thrombosis, capillary

hæmorrhages, angio-spasm, or cerebral œdema, may be responsible for transient monoplegia or hemiplegia, convulsions and coma. In some cases these and other symptoms of central nervous origin are associated with a sudden rise in blood pressure and constitute hypertensive cerebral attacks. In other cases the intracranial disturbance is due to œdema of the brain, as was first suggested by Traube in 1860. In this type of attack the patient is usually under the age of 40. There is complaint of sudden severe headache. Drowsiness is common, and motor weakness with focal signs or loss of vision may be transient phenomena. Retinal hæmorrhages, exudates and papilloedema appear within a few hours. The blood pressure rises and the cerebro-spinal fluid pressure is also raised (McAlpine). The blood urica is normal unless the attack occurs as a complication of uræmia. Œdema of the brain is found post mortem.

Other diseases which may simulate uræmia are cerebral tumour and meningitis. Some cases of cerebral tumour without localising signs, but with the classical symptoms of headache, vomiting and optic neuritis, may be very difficult to distinguish from uræmia, if there is chronic nephritis as well—a not very uncommon complication in syphilitic tumours of the brain. But such cases are more chronic in their course than uræmia. If the cerebral type of uræmia be accompanied by pyrexia, as it sometimes is, the question of meningitis must be considered. Lumbar puncture may then throw light on the case by the cytology, bacteriology and urica content of the fluid. Lastly, when the uramic state has reached the stage of coma the differential diagnosis from alcoholic poisoning, status epilepticus, trauma, opium, diabetic coma, the apoplectiform onset of general paralysis and cerebral hæmorrhage must be made. In all such cases a careful examination of the urine is essential, because comparatively slight renal inadequacy may lead to toxic symptoms by the retention of some poison which would otherwise have been promptly eliminated. Thus salicylates, iodides, opium and mercury are badly excreted by the nephritic.

Treatment.—As in the case of extra-renal uræmia the maintenance of the normal acid-base balance of the blood is of first importance. In most cases of renal uræmia there is acidæmia, and this is treated by the cautious administration of alkali by mouth, or by the intravenous injection of a 2 per cent. solution of sodium bicarbonate 200 to 400 c.c. Anæmia renders the acid-base balance unstable. It is treated by the administration of iron, and 60 to 90 grains of iron and ammonium citrate are given in divided doses daily, provided the salt does not upset the digestion. Vomiting and diarrhœa should be checked, because they weaken the patient, prevent assimilation of food and cause dehydration. Vomiting causes alkalosis, and diarrhœa may cause acidæmia. A bismuth mixture or 3 minims of dilute hydrocyanic acid and 10 minims of solution of adrenaline hydrochloride 1 in 1000 in half an ounce of water given every 3 or 4 hours may afford relief.

The retention of urica is treated by a low nitrogen diet. The diet advised for acute nephritis may be given with advantage for a week or 10 days, and after this for 1 or 2 days in each week. If a low nitrogen diet is continued it must at least be adequate in its protein content to retain nitrogenous equilibrium, for which purpose 1 G. per kilo body-weight is required. Elimination is promoted by maintaining bowel function, avoiding constipation on the one hand, and diarrhœa on the other. According to von Noorden, 8 G. of

nitrogen can be excreted by the bowel in the day and 3 G. by the skin. Strong aperients and mercurial preparations should not be employed. A good evacuation may be secured by magnesium sulphate 30 to 60 grains repeated as necessary, 60 to 90 grains of pulv. jalapæ co., 1 oz. of mist. sennæ co., or 60 to 90 grains of compound liquorice powder. The function of the skin is promoted by suitable clothing, wearing wool or flannel next to the skin, and sleeping between blankets. Much sweating may have disadvantages that it causes dehydration, and gives the kidneys a more concentrated and therefore a more irritating urine to secrete. The vapour bath or hot pack is more trying to the heart than the hot-air bath. None of these measures should be continued more than a quarter of an hour after sweating has begun, and a careful watch must be kept on the pulse; the procedure should be stopped at once if there are any signs of collapse, and stimulants should be at hand. If the treatment is having a good effect, sweating will begin at a lower temperature with successive baths. A nightly hot bath, containing 4 tablespoonfuls of mustard, followed by wrapping in hot blankets until sweating has ceased, is useful in some chronic cases, even where there is no œdema. Pilocarpine is no longer advised to provoke sweating.

Headache and other symptoms of cerebral irritation may be relieved by lumbar puncture when accompanied by an increase in cerebro-spinal fluid pressure. It is safe to draw off 10 or 20 c.c. if the pressure is raised, but if with the drawing off of the fluid there is increase of headache the needle should be withdrawn immediately. If the headache is due to a hypertensive attack, particularly if there is evidence of congestive heart failure, venesection may give relief. The withdrawal by venesection of 10 to 20 oz. of blood may cure the headache, and it may stop convulsions in acute nephritis with an overburdened heart. Venesection is contra-indicated in chronic uræmia with anæmia. Bromide and chloral hydrate will often relieve headache. Twenty to 30 grains of sodium bromide with 15 to 20 grains of chloral hydrate are given by mouth. Aspirin, phenacetin and codeine are other useful drugs best prescribed in combination in severe cases. Morphine, papaveretum (omnupon) and dilaudid are reserved for intractable cases, and are to be used with caution.

LARDACEOUS DISEASE

Synonyms.—Amyloid or Waxy Kidney.

Definition.—A pathological condition in which the blood vessels of the kidney, in more advanced cases the tunica of the tubules and the interstitial tissue also, are the seat of waxy degeneration.

Ætiology.—This affection is now rarely met with. It attacks men more than women, and although occasionally seen in children it is more likely to occur in adolescence and earlier adult life, being uncommon after fifty years of age. It is usually due to chronic suppuration, especially in bone, chronic tuberculosis and syphilis. It rarely occurs in other chronic infections, but it has been described in severe rheumatic heart disease, and a certain amount of amyloid change has sometimes been found post mortem in patients suffering from chronic cardiovascular disease and chronic nephritis in the absence of chronic suppuration. As it is a degenerative change it has however more affinity with nephrosis than with nephritis.

Pathology.—Amyloid material or lardaccin is a product of protein degeneration, and consists of protein linked with chondroitin-sulphuric acid. The latter substance is a normal constituent of elastic tissue and cartilage. In uncomplicated cases, the affected kidney has the appearance of a large white kidney with a smooth surface and a capsule that strips easily. The organ is firmer than it otherwise would be. On section, the cortex is thicker than normal and has a yellowish white appearance; the glomeruli may be visible as minute translucent spots. The pyramids are dark red, in contrast to the pale cortex. If a solution of iodine in potassium iodide is poured over the surface, some of the glomeruli stand out as mahogany-brown spots and the vasa recta as brown streaks. In histological preparations stained with methyl-violet, amyloid material takes a pink colour. The disease tends to appear first in the capillaries of some glomeruli, while others are normal, and its incidence is often partial within a single glomerulus. The afferent arterioles, vasa recta and capillary plexus are next affected; in more advanced cases there is amyloid degeneration of the tunica propria of the tubules with amyloid deposits in the interstitial tissue. In most cases there is an associated nephritis, interstitial rather than parenchymatous. The kidney lesion is generally the most striking part of a widespread lardaceous degeneration which also involves the liver, spleen and intestine; less commonly the blood vessels of the thyroid, suprarenals, pancreas, heart and brain may be affected as well. Occasionally only the kidney is implicated.

Symptoms.—The onset is insidious and the symptoms are not likely to occur unless chronic suppuration has existed for at least 3 months.

The urine is copious, of low specific gravity (1003 to 1010). The amount of albumin is variable; when abundant there is probably coincident nephritis. The amount of urine and its specific gravity may also be affected by the presence and degree of coincident nephritis, and the state of the heart. Hyaline and granular casts are present in the urine; casts staining brown with iodine are not evidence of amyloid disease, and may occur in other diseases of the kidneys. True waxy casts are not found. In later stages there is oedema, with diminished excretion of urine. The blood pressure is not raised, nor is the left ventricle hypertrophied, unless there is coexistent chronic nephritis.

Diagnosis.—The diagnosis is indicated by the nature of the urine. It is made (1) when there is a sufficient cause in the past history or present condition, namely, chronic suppuration or syphilis; (2) on the general condition of the patient, namely, a secondary anaemia, which may reach an extreme grade, with a pale or "alabaster" facies and cachexia; (3) on signs of lardaceous disease in other organs, such as enlargement of the liver or spleen and diarrhoea.

Course and Prognosis.—This depends on that of the primary cause. If the latter is unchecked, the disease is slowly progressive and death occurs from exhaustion due to the original disease, less often from uræmia. Where the original disease can be cured, recovery may occur. Complete recovery of the kidneys is less likely than is recovery of the liver, spleen and intestines.

Treatment.—The treatment is that of the original disease. In suppuration of the bones or joints, empyema, etc., it is surgical; but it must be recognised that in advanced cases surgical treatment may be too late, even though it is successful in eradicating the septic focus. In all cases fresh air

and sunlight and a nourishing diet are essential. Iron, arsenic and cod-liver oil should be given. Cases of syphilitic origin should be treated with bismuth and arsenic, while mercury and iodides should rarely be given, and then only with caution since even therapeutic doses have produced serious reactions.

PYELITIS

Definition.—Pyelitis is inflammation of the renal pelvis. The changes in the renal parenchyma are those described under Toxæmic Kidney. Pyelitis may be complicated by nephritis, and the condition is then termed pyelo-nephritis.

Ætiology.—Most cases are due to a blood-borne infection of the renal pelvis, and it may be noted in this connection that it is a normal function of the kidney to excrete micro-organisms present in the blood stream; whether the renal parenchyma is, or is not, of necessity damaged in the process is a point on which there is not as yet exact information. The pelvis may also be involved by ascending infections—(a) via the lumen of the ureter when there is ureteral obstruction; it is probable that infection does not spread by this channel when the lumen is normally patent. (b) By way of the peri-ureteral lymphatics from local foci in lower parts of the urinary tract, such as the bladder, urethra, prostate, seminal vesicles and epididymis. Lastly, there is the possibility of direct spread of infection from the bowel, and by cross lymphatic channels from one kidney to the other. In those cases in which a pyelitis occurs secondary to appendicitis, cholecystitis, ulcerative colitis, etc., the spread of infection may be by the lymphatics or the blood stream.

Pyelitis is more common in females than in males. Its age incidence depends on the determining cause. Thus, it is common in female infants, as a result perhaps of urethral infection, to which they are more liable than male infants. It is not an uncommon complication of pregnancy, occurring especially in the fifth month of gestation. It is common in males at a later age, associated with enlarged prostate and cystitis.

In general terms any injury or disease of the renal pelvis, or any condition which interferes with the normal flow of urine, may be the determining cause of pyelitis. Thus it is a common complication of hydronephrosis from whatever cause. It often complicates stone in a kidney, tuberculosis of the kidney and new-growths of the renal pelvis. The frequency of pyelitis as a complication of intestinal catarrh (whether due to infection or the habitual use of laxatives) and ulceration is probably due to the increased virulence and excessive numbers of bacteria that reach the kidney in such conditions.

Pathology.—The mucous membrane of the pelvis is swollen, cedematous and hyperæmic, and the submucous venules are engorged. Where there is obstruction, the pelvis is dilated and contains a slightly turbid or opalescent fluid. In these circumstances the ureter above the obstruction is dilated and tortuous and its walls are thickened. The kidney is swollen and pale, from cloudy swelling, and in severe cases there may be multiple small abscesses in the renal parenchyma.

Bacillus coli is by far the most common infecting micro-organism. Streptococci, staphylococci, gonococci and bacilli of the proteus and typhoid

groups may be found. The infecting micro-organism is readily recovered from the urine.

Symptoms.—The clinical types of pyelitis differ greatly from one another, and the condition may be responsible for an acute fulminating illness or for chronic malaise of indefinite nature.

LOCAL SYMPTOMS.—Pain is the most important, especially as a diagnostic indication in acute cases. It is a dull ache in the loin or flank, at first slight and intermittent, later, or in other cases at once, constant and sometimes intense. Occasionally it takes the form of renal colic. At its onset the pain may be diffuse and abdominal. Increased frequency of micturition is a common symptom. There may be strangury.

GENERAL SYMPTOMS.—In acute cases there may be sudden onset with rigors, vomiting, headache and the general constitutional disturbance of profound toxæmia. These cases may simulate septicæmia (in fact there may be septicæmia), appendicitis, or, when associated with abdominal distension, constipation and vomiting may even simulate intestinal obstruction. In other cases, with cerebral symptoms, meningitis may at first be difficult to exclude.

In subacute cases, without marked pain or rigors, there is general malaise, fever, anorexia, wasting and a secondary anæmia associated with some degree of polymorphonuclear leucocytosis (W.B.C. = 10 to 15,000).

In relapsing cases there are periods of exacerbation with acute symptoms, and intervening periods of fair health or general malaise. Fever is commonly present; in acute cases with rigors it may rise to 105° or 106° F. In general the temperature is irregular, remittent or intermittent, varying between 102° and 104° F. in acute cases, and 100° and 102° F. in subacute cases. The pulse is raised in proportion to the temperature, and there is a corresponding slight increase in the respiration rate. Of other general symptoms constipation or diarrhœa frequently precedes the disease, and constipation generally accompanies it. Toxæmia is often marked.

Deep tenderness on palpation of the renal region is the most important sign to determine. There is some degree of abdominal rigidity, and it may be possible to determine enlargement and tenderness of the kidney. The urine is passed in small quantities at frequent intervals. It has the usual characters of febrile urine and is turbid. The turbidity or an opalescence is still present after filtration. When an appreciable quantity of pus is present it settles at the bottom of a specimen glass in a thick whitish deposit. Examination of the deposit (catheter specimen in women) shows pus cells and epithelial cells from the urinary tract. There may be hæmaturia.

Bacteriuria.—In this condition bacteria are present in the urine in such quantity as to make it hazy to the naked eye, but there is little or no inflammatory reaction in any part of the urinary tract. Hence there are no localising symptoms and few pus cells. The urine when freshly passed has a hazy appearance. In a test-tube, when the tube is rotated, the urine has a "satiny" appearance or shimmer. It is not cleared by filtration. It often has a fishy smell in *B. coli* infection, and is ammoniacal in smell in *B. proteus* infection. Its reaction is acid, unless due to staphylococcal or *B. proteus* infection. It generally contains a trace of albumin, and often may contain a few white blood corpuscles and epithelial cells. A catheter specimen grown in broth, in dilutions of 1 c.c., $\frac{1}{10}$ c.c. and $\frac{1}{100}$ c.c. urine in

10 c.c. broth, gives a growth in all dilutions, and in *B. coli* infections there is generally a growth in greater dilutions. Streptococcal and staphylococcal infections are less common.

There may be no other symptoms. On the other hand, there may be indefinite malaise, fever, gastro-intestinal disturbance, especially indigestion, constipation and abdominal pain; in other cases headaches, rigors and even meningism may occur. There may be local symptoms, such as enuresis in children and frequency of micturition in adults. When the symptoms point to inflammatory reaction in one part of the genito-urinary tract, such as pyelitis, cystitis, prostatitis, urethritis, or epididymitis, the condition is better diagnosed accordingly.

The recognition of bacilluria may be of great importance, not only because of the ill-health, acute disease or complications for which it may be responsible, but also because it may be a valuable pointer to other disease. For instance, a patient may complain of loss of energy and indefinite malaise. On clinical examination the only clinical finding may be some degree of secondary anæmia or bacilluria. A further examination of the urine bacteriologically, or X-ray examination of the urinary tract, may reveal previously unsuspected tuberculous disease, stone or neoplasm, even in the absence of urinary symptoms.

Diagnosis.—When there is fever and constitutional disturbance without localising signs or symptoms, the differential diagnosis is from those diseases which come in their early phases under the category of indeterminate fever. The diagnosis is established by examination of the urine. Pyonephrosis is diagnosed by the presence of a tumour. Calculus is recognised by its clinical features and by X-ray photograph. Perinephric abscess in its early stages is not accompanied by pyuria or frequency of micturition. Cystitis is generally afebrile; and it is accompanied by suprapubic discomfort and pain, particularly at the end of micturition; the diagnosis can be established by cystoscopy. Urethritis is recognised by local tenderness, urethral discharge and urethroscopy, and prostatitis by swelling and tenderness on rectal examination.

Prognosis.—The natural course in the majority of cases is to recovery in a few weeks. With modern remedies the urine can generally be sterilised in 7 to 14 days unless the urinary infection is a complication of some other condition or disease. The prognosis depends very largely on this sterilisation of the urine, because the persistence of even a minimal infection is likely to lead to relapse, or apparent recovery may be followed by a recurrence after a variable length of time. The disease may progress to pyelo-nephritis, ascending suppurative nephritis, pyonephrosis or perinephric abscess. A fatal termination is rare, except when the condition complicates other disease, such as paraplegia, or in elderly persons with obstruction to the outflow of urine.

Treatment.—Prophylaxis is important in nurseries and children's hospitals, since there is evidence of spread of infection via the urethra, at any rate in females. Here it is a question of cleanliness. In general terms exposure to cold, over-fatigue, and loose stools are to be avoided when there is susceptibility to coli infection of the urinary tract.

The treatment of an acute attack consists of absolute rest in bed, flushing out the kidneys with large quantities of fluid, and regulation of bowel

function. It is important, especially when there is fever, to avoid exposure to cold and any possibility of chill. Particularly when there is fever the patient should wear wool next to the skin, lie between blankets and be nursed in bed. Five to 8 pints of fluid are given in every 24 hours in the form of water, barley water, imperial drink, lemon drink, weak tea and thin soups. Milk as such is unsuitable, but junket, buttermilk, whey and cream are good. As the temperature subsides the diet is increased by the addition of carbohydrates, fruit, vegetables and fat. Cooked milk in the form of milk puddings is allowed. Alcohol is withheld. The bowels are emptied with an initial laxative, followed by an enema if necessary. After this the action of the bowels is regulated with paraffin, salts or mild laxatives, such as liquorice powder, senna pods or rhubarb, so that constipation is avoided on the one hand and loose stools on the other.

In the initial febrile stage when there is bacterial toxæmia, sufficient alkali is given by mouth to make the urine alkaline. A mixture containing 30 grains each of potassium citrate and sodium bicarbonate is given 3-hourly until the urine is alkaline. Every specimen of urine passed is tested with litmus paper. When the urine is alkaline the quantity of alkali by mouth is reduced by giving it 4- or 6-hourly, but always in sufficient quantity to keep every specimen of urine alkaline, until the temperature is normal. One of the sulphanilamide group of drugs is then given 4-hourly. A full dose is sulphanilamide 1 gramme 4-hourly (6 grammes in 24 hours). This may be reduced to 4 grammes in 24 hours after 3 or 4 days, and later to 3 grammes (*i.e.* 0.5 gramme 4-hourly). Alternatively sulphapyridine (M. & B. 693) may be prescribed, or rubiazol (Roussel) 12 to 8 tablets in 24 hours (one tablet contains 0.2 gramme of the azo-compound). In some cases, especially if these drugs are not well tolerated, a mandelic acid salt may be given. The usual dose is 45 grains of ammonium or calcium mandelate thrice daily.

Flushing out the kidneys with large draughts of water must always dilute the antiseptic drug. Flushing and antisepsis should therefore be attempted consecutively and not simultaneously.

In uncomplicated cases a sterile urine may be expected in 7 to 10 days. In some cases the drug may have to be administered for a longer period, or the course of treatment may have to be repeated after an interval of a fortnight or 3 weeks. Occasionally in resistant cases, or in those which continually relapse as soon as the drug is withheld, good results have been obtained by giving ammonium mandelate for several weeks, provided that the patient is under good observation, so that the occurrence of kidney damage and acidæmia can be prevented. In acute cases with fever it may be better to initiate treatment with a sulphanilamide drug without the initial course of alkali therapy, provided that a close observation of the case can be maintained, including estimation of the blood urea as required. During the acute phase of the disease the kidneys are more susceptible to the toxic effects of these drugs.

It is important to remember that, like simple bacilluria, infections of the urinary tract are often a complication of organic disease of either the urinary tract or bowel. A urinary infection, which at first sight appears to be a simple coli infection of the urinary tract, may be only a complication of tuberculosis of the kidney calculus, hydronephrosis or neoplasm. Equally it may be a complication of organic disease of the digestive tract, such as chronic appendicitis,

especially if the right ureter is involved, diverticulosis, or even cholecystitis. In any case of urinary infection that is resistant to treatment or presents any unusual symptom, a detailed investigation of both urinary and digestive tracts is required in order to determine or exclude a change in structure which may be the underlying and determining cause of the urinary infection.

In the uncommon fulminating cases with unilateral suppurative nephritis, nephrectomy may save the patient's life.

Chronic cases.—An initial course of treatment such as that outlined for acute infection with rest in bed is advisable. In some cases, perhaps on account of long-standing infection, or some other disease of the urinary tract, such as calculus diverticulum of the bladder, enlargement of the prostate, etc., it may be impossible to sterilise the urinary tract. Every effort should then be made to build up the patient's resistance by living in fresh air, avoiding chill and over-fatigue, a generous and nourishing diet, and by so arranging the diet that the bowels are open regularly without taking purgatives other than a simple saline in the morning, paraffin and agar, or other laxative which determines the evacuation of a formed stool. It is important so far as is possible to restore integrity of structure to the body as a whole by the cure of anæmia, for instance, and by the eradication of obvious sepsis elsewhere in the body. In incurable cases the infection may be controlled to a greater or less extent by long-continued use of hexamine, which may often be given with advantage with methylene blue.

PERINEPHRITIS AND PERINEPHRIC ABSCESS

1. Perinephritis without suppuration is really a part of some cases of chronic nephritis. Its clinical importance is not generally recognised, but it may be a cause of lumbar pain in that disease. The capsule of the kidney is thickened and adherent to the perirenal tissues, many of the adhesions being vascular.

2. Perinephritis proceeding to suppuration may be primary or secondary.

Ætiology and Pathology.—The primary form may follow injury, but more frequently it results from boils, carbuncles and tonsillitis, or complicates an acute specific fever. Soon after the War of 1914-1918, cases were so common as to be described under the name of *epidemic perinephric suppuration*. The infecting organism is *Staphylococcus pyogenes*. J. Koch has shown experimentally that intravenous injection of staphylococci is followed by their excretion in the urine after an interval of 4 to 6 hours. In the process of excretion, according to Koch, they may give rise to multiple cortical abscesses, cylindrical medullary abscesses, or, passing along the cortical lymphatics, may gain access to the perinephric tissues and there cause abscess formation. In these circumstances perinephric abscess is an example of the mildest form of staphylococcal pyæmia with single metastatic abscess formation. The secondary form may complicate suppuration in the neighbouring organs, such as the kidney, liver, gall-bladder or appendix. It may be secondary to caries of the spine. In other cases the infection may be carried by lymphatics from a focus in or around the bladder, rectum or female pelvic organs.

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Such is the ordinary terminology, but it will be observed that the "primary" form is really due to infection from a distant focus through the blood stream, while the "secondary" is due to direct extension or infection through the lymphatics from some focus in the neighbourhood of the kidney.

Symptoms.—The onset is generally gradual. It is characterised by fever and malaise as in typhoid fever. There may be no local symptoms for the first 7 to 14 days, and during this period there is increasing toxæmia, general abdominal discomfort or pain, slight fullness and resistance, with deep tenderness, in the affected loin. As the abscess forms, pain and tenderness increase, there is induration and, later, redness of the skin and œdema in the lumbar region. The tumour first tends to spread backwards, obliterating the normal hollow in the loin, and then as pus collects it may spread forwards, forming a tender tumour palpable from the front. In its relations to the colon it resembles a renal tumour, but does not move with respiration. There is resistance or rigidity of the abdominal wall on the affected side. There is an increasing polymorphonuclear leucocytosis up to 20,000 or even 40,000. The urine is febrile in character, containing a trace of albumin and perhaps a few white blood corpuscles; it does not contain pus, unless the kidney itself is involved, but hæmaturia may occur. In some cases the disease runs an acute course, and there may be rigors at an early stage.

Course.—When the condition is simply associated with chronic nephritis it has no separate significance. When it proceeds to suppuration the abscess may rupture into the peritoneum, colon or pleura, or on to the surface, unless the abscess is opened and drained.

Diagnosis.—Before localising signs appear the condition may be mistaken for typhoid fever, malaria or septic endocarditis. The blood examination is important for the purpose of excluding malarial parasites; leucocytosis is against typhoid fever, and when above 15,000 is in general against infective endocarditis. Absence of agglutination of micro-organisms of the typhoid group is further evidence.

When the tumour exists it has to be distinguished from a renal tumour or pyonephrosis. Renal and adrenal growths may be accompanied by fever, but do not usually give the general symptoms of suppuration; they tend to extend forwards rather than backwards, and induration of the tissues is absent. Pyonephrosis causes symptoms of suppuration and a tender swelling, but the tumour is circumscribed, moves with respiration, and does not cause any bulging in the lumbar region. Pyuria is usually present.

The diagnosis of caries of the spine, hip disease, and even of myositis as distinct from perinephritis may be difficult. Since perinephritis in itself induces lumbar rigidity and some degree of scoliosis, X-ray examination may be required to exclude caries of the spine. Hip-joint disease is excluded by absence of local tenderness and by the freedom of flexion and rotation of the thigh.

Treatment.—In the early stages, before there is evidence of suppuration, and when the chief symptom is lumbar pain, the treatment is that of a patient acutely ill with a general toxæmia. The bowels should be kept well open, and fomentations or poultices applied to the lumbar region. Aspirin

may be given to relieve pain. An operation should be performed and the abscess evacuated as soon as the diagnosis is definitely established.

TUBERCULOSIS OF THE KIDNEY

Small grey tubercles are frequently found scattered through the kidneys in persons who die of acute miliary tuberculosis; the kidney disease, however, scarcely affects the clinical aspect of the case, and this form of renal tuberculosis will not be considered here. Further, in patients who die of pulmonary tuberculosis it is not uncommon to find tuberculous foci in the kidneys post mortem, although there was no indication of their presence during life.

Clinical renal tuberculosis is either the fibro-caseating form of the disease, or it is tuberculous hydronephrosis. In either case, the tuberculous infection is generally primary in the kidney in so far as its clinical expression is concerned.

Ætiology.—It is more common in women than men. The maximum age incidence is in the third and fourth decades; the disease is uncommon in the young and rare in the old. At an early stage the disease is unilateral. In the majority of cases the tubercle bacilli are carried to the kidney by the blood stream from a tuberculous focus, such as a caseating lymph gland. Recent experimental work has shown that bacteria do not ascend in the lumen of the ureter unless it is diseased, when the infection may spread by direct extension in its walls. Infection may also reach the kidney via the lymphatics in a proportion of cases. The path of infection is by way of the ureteric lymphatics, and it is probable that in pelvic tuberculosis, for example, tuberculous prostatitis, may spread to the kidney by this route. There is also reason to think that tubercle bacilli from a diseased kidney may infect the opposite healthy kidney by the same lymphatic path, the bacilli first travelling in the urine and walls of the ureter from the diseased kidney and causing disease of the bladder, and then travelling from the bladder by way of the ureteric lymphatics to the sound kidney. On the other hand, there is a shorter path for infection from one kidney to another by the para-aortic lymphatic system. Since the disease in the other kidney takes the same anatomical form as it originally had in the kidney first affected, it is probable that, if the first is due to a blood-borne infection, so is the second. Vesical tuberculosis is, as a rule, secondary to infection elsewhere in the urogenital system commonly in the kidneys.

Pathology.—The initial lesion is in the cortex, or one of the pyramids, and it consists of one or more tubercles. The morbid process spreads by destruction of kidney tissue; there is caseation in the centre of the lesion, inflammatory reaction, with intense small-cell infiltration, giant-cell formation and more or less fibrosis at the periphery. The lesion also spreads by the deposition of tubercles at a distance; these are scattered through the cortex, singly or in groups. Extension through the capsule is uncommon, but extension to the renal pelvis is frequent. Complete destruction of one or more pyramids may occur, or the disease may spread and involve one or more calices or the entire pelvis. The resulting infiltration and cicatricial contraction may lead to hydro- or pyo-nephrosis. The disease tends to extend

down the ureter, and the bladder is commonly infected at an early stage. Secondary infections may lead to metastatic abscesses in the kidneys and ultimately to destruction of the whole organ.

Symptoms.—Frequency of micturition is often the earliest symptom; it is first noticed by day and later at night. Urgency and painful micturition develop next. The urine may show no other abnormality than a trace of albumin at an early stage; characteristically it is pale and a little turbid from the presence of pus; it is acid in reaction, it may contain a few renal cells, and it is sterile on culture. By appropriate staining tubercle bacilli may be demonstrated in the centrifuged deposit. Hæmaturia may be the first symptom, or the disease may develop insidiously with lumbar pain. On examination, the kidney is sometimes enlarged, and it may be hard and irregular; it is often tender. Tenderness along the course of the ureter or thickening of the ureter, as determined by abdominal or rectal examination, is of great importance. The rest of the urino-genital system requires close examination; this should include cystoscopy and in some cases ureteral catheterisation. X-ray examination of the abdomen may reveal calcified tuberculosis of the kidneys or lymph glands, and it may be required in the differential diagnosis from renal calculus. Finally, a careful review of the patient's history and present condition for evidence of a chronic bacterial toxæmia or of tuberculous infection elsewhere must be made.

Diagnosis.—The presence of tubercle bacilli in the urine, whether determined by microscopic examination of the stained deposit or by guinea-pig inoculation, is not absolute proof of renal tuberculosis, because the bacilli may be excreted by a healthy kidney or they may come from some other part of the urinary tract. Nevertheless, the demonstration of tubercle bacilli in the urine is of the first importance in a doubtful case, and the diagnosis may be established by cystoscopy. The cases which require most careful examination are those with an atypical onset, such as massive hæmaturia, and those in which there is a gross secondary infection when first seen. The possibility of renal tuberculosis must always be borne in mind in hydro- and pyo-nephrosis. The differential diagnosis from simple albuminuria and the several forms of Bright's disease is made on the presence of pyuria and the absence of signs and symptoms of chronic nephritis. Patients with pulmonary tuberculosis are perhaps more prone than others to chronic nephritis on account of the secondary infections which complicate their disease.

Course and Prognosis.—The onset is insidious and the course progressive. Natural recovery is hardly known, though occasionally an unsuspected caseous kidney may be found at autopsy in patients dying of other diseases. The disease runs an uncertain course, having a duration of a few years up to ten or even twelve years from the date of diagnosis. Death results from tuberculous toxæmia, secondary infection, or failure of renal function.

Treatment.—When the disease is unilateral the kidney should be removed but nephrectomy is rarely justified if the other kidney is involved. In any case the patient's health and resistance should be raised to the utmost by rest, fresh air and good food, on the general lines of treatment of tuberculosis of the lungs. Cautious tuberculin treatment may be indicated when the disease cannot be treated surgically.

RENAL CALCULI (NEPHROLITHIASIS)

Renal calculi may be composed of calcium oxalate or carbonate, uric acid, urates, phosphates, cystin, or of a mixture of these.

Ætiology.—All these materials are sparingly soluble in water and their solubility in urine is dependent on (i) its pH. If this stands at 5 uric acid is precipitated, while phosphates and carbonates are deposited at pH about 8; the others at some intermediate point. (ii) On the presence of urea, which renders both uric acid and oxalates more soluble. (iii) The protective action of certain non-albuminous colloids. If these become coagulated their protective influence is lost. Thus two factors are required to form a renal calculus: crystals derived from the urine and some colloidal material to bind them together. Hence, as Benjamin Moore pointed out, the commonest nucleus of a stone is calcium oxalate, since oxaluria excites albuminuria and even hæmaturia, thus providing the necessary colloid. Prolonged recumbency, as after fracture of the femur, provides opportunity for calculus formation apparently from stagnation in the dorsal portion of the calices. Infection of the urinary tract such as pyelitis is not considered so important as formerly, and indeed may be merely secondary to the calculus; but it is a factor in cystinuria, which will not lead to a calculus unless the urine becomes infected. Pure uric-acid stones may occur in quite young children, but the definite deposit of uric-acid crystals in the pyramids and pelvis of the kidney which is almost a normal event does not seem to lead to calculus formation and milk is usually sufficiently diuretic to remove them. The most important single factor in the prophylaxis of stone is adequate diet. Lack of vitamin A is especially prone to excite stone. In a recent investigation 96 per cent. of cases of renal calculi showed evidence of its deficiency. It is necessary for the maintenance of the proper nutrition of epithelial linings everywhere. The former comparative frequency of uric-acid stones in the children of the poor in London was probably related to the scarcity of fresh vegetables in the diet. Such stones were also common in Norfolk and the neighbouring fens. Chalk in the soil or in the drinking water does not predispose to stone. The factors leading to the deposits of various crystals in the urine have already been discussed (see Abnormalities of the Urinary Secretion).

Calculi may occur at any age, but are very rare in the old. They are commoner in males than in females. Those of sedentary habit are more liable to them. Alcohol and lead are said to predispose to renal calculi. A high blood calcium, whether due to excess of parathormone (as in parathyroid tumours) or of vitamin D, can be an important factor in producing calculi of calcium phosphate.

Pathology.—The pure oxalate stone is very hard, mulberry-shaped, stained by altered blood, and varies in size from that of a mere granule to that of a walnut. If it is encrusted with uric acid it becomes brown, and in form a coral-shaped mass, representing a cast of the renal pelvis and calices. Phosphatic stones are generally smooth and white. A cystin stone is hard, oval, light amber or greenish in colour, with a glistening surface. Other forms are rare. If the stone remains in the renal pelvis it may (1) by gradually increasing in size lead to the atrophy of the renal tissue; (2) by eroding the

capsule of the kidney produce a fistula into the perinephric tissues, resulting in a perinephric abscess; (3) by obstructing the outflow of urine cause hydronephrosis or, more frequently, pyonephrosis. If it passes into the ureter it may become impacted, in this way again exciting hydronephrosis or pyonephrosis, or if it obstructs the ureter completely, may produce atrophy of the kidney. If it causes ulceration of the ureter, this may be followed by stenosis. If it passes into the bladder it is very likely to excite ammoniacal decomposition, and thus become encrusted with phosphates.

Symptoms.—A stone may remain latent in the kidney without causing any symptoms. More usually it causes pain, particularly on any jolting movement. This is occasionally referred to the opposite side, a point to be borne in mind when considering operation. A bout of pain may be accompanied by hæmaturia, and there may be albuminuria for some days afterwards. A small oxalate stone may produce more pain than a large uratic stone, because of its hardness and roughness. A large, branched uratic stone occasionally causes profuse hæmaturia without any pain. The results of renal calculi may be classified as (a) mechanical, (b) septic. Under the first heading come colic, hæmaturia, anuria, hydronephrosis; under the second, pyelitis, perinephric abscess, pyonephrosis.

Renal colic is the most severe and distressing manifestation of calculus. It is particularly likely to be started by riding on a horse or in a train or omnibus, which causes the calculus to engage in the entrance to the ureter. Violent paroxysms of pain then occur, radiating along the course of the genito-crural nerve down into the groin and testis, which becomes retracted in the scrotum. The pain is also felt in the loin, and the muscles overlying the kidney become rigid. Vomiting and sweating are common. The patient is unable to keep still, and rolls about or gets on to his hands and knees, calling out with each paroxysm. He becomes pale and his pulse increases in frequency, and the temperature is apt to rise. During or after the attack there is usually some hæmaturia, and crystals may be found in the urine. The attack may last several hours and then end as abruptly as it began. Anuria is a serious symptom and implies that the ureter is completely blocked, and the other kidney is either diseased or its secretion reflexly inhibited. Occasionally both ureters may be blocked by calculi. Symptoms referred to the bladder, prostate or seminal vesicles do not occur until the stone reaches the bladder or the lower end of the ureter.

Diagnosis.—The occurrence of renal colic and hæmaturia suggests stone, but these symptoms may be produced by the passage of a blood clot from renal neoplasm or by acute pyelitis, especially in a movable kidney. Ordinary examination of the abdomen reveals nothing beyond lumbar tenderness in uncomplicated cases. X-ray examination is of great value. Oxalate stones are the easiest to detect by that method, as even when small they throw a dense shadow. This is fortunate, since oxalate stones are the commonest. Pure uratic stones may not be detected unless they are large. Cystin stones throw very little shadow. Calcareous abdominal glands and phleboliths may be mistaken for calculi on X-ray examination. In doubtful cases, pyelography, intravenous or instrumental, should be done. A skiagram of the pelvis should never be omitted, since a stone may have passed down to this region. Attacks of pain and hæmaturia with the presence of calcium oxalate crystals in the urine, but with a negative X-ray examination, are probably

due to crises of oxaluria (see p. 1296). Appendicular colic may simulate renal colic, but the point of maximum tenderness is different.

Prognosis.—As long as there is no serious destruction of kidney substance or septic complication the outlook as to life is good, if treatment be adequate. Attacks of renal colic may occur from time to time, with great suffering, and even after stones have been removed by operation they may form again, though this is exceptional. Occasionally stones may be followed by a true chronic nephritis with its usual consequences.

Treatment.—The methods which should be employed when crystals likely to form stones are found in the urine have been described under urinary deposits. Careful attention must be given to the diet, especially to its vitamin content. Disinfection of the urine should be carried out as described under bacilluria and pyelitis. It is well, however, not to render the urine alkaline when a stone is suspected, since this would lead to a deposit of phosphates upon it. A book of litmus papers should be given to the patient with instructions to place blue and red strips in the morning urine, which is likely to be the most acid. Enough citrate of potash should be given to render the urine amphoteric but not alkaline. Probably 20 grains at night will be sufficient for this purpose. The urine should be kept dilute by taking water freely. Mineral waters, such as Contrexéville and Evian, are helpful, the former particularly for uric acid, the latter for oxalates. Whey is also helpful when uric acid crystals are present. If a renal calculus is present, and this is confirmed by X-rays, removal by operation is indicated. The following points, however, are generally contra-indications for operation: (i) large bilateral stones; (ii) stones which are only the size of a pea or smaller, unless there is severe pain, extensive absorption of renal substance causing toxic symptoms, or obstruction to the outflow of urine. If a small stone is not passed as a result of medical treatment, its removal by operation should be seriously considered; (iii) in some patients small calculi are repeatedly formed and passed. In these cases operation is better postponed because of the likelihood of recurrence. If the diagnosis is uncertain, or operation is refused or postponed or considered inadvisable because of the patient's general condition, the treatment appropriate to the deposit found in the urine should be continued. Violent exercise and jolting movements should be avoided. Small stones can often be got rid of by giving the patient 5 to 10 minims of tincture of belladonna with 10 grains of potassium citrate every 4 hours for a few days, and directing that 5 pints of water should be taken in the 24 hours. For the symptomatic relief of pain, aspirin in 10-grain doses, hot baths and kaolin poultice (antiphlogistine) may be of service. Morphine should be avoided in the treatment of chronic renal pain, on account of the danger of establishing a habit.

For an attack of renal colic, $\frac{1}{4}$ th to $\frac{1}{2}$ rd of a grain of morphine tartrate, together with $\frac{1}{100}$ th of a grain of atropine sulphate, should be given hypodermically. The anti-spasmodic effect of the atropine aids the onward passage of the stone, while the morphine relieves the pain. If morphine be given alone, the pain is apt to recur as soon as its anodyne effect passes off. Ten minims of tincture of belladonna should then be given in an ounce of water every 3 or 4 hours, with abundant fluids, as described above, until the pupils are dilated and the face rather flushed. Inhalations of chloroform may be necessary at the onset, until the drugs have had time to act. Hot applications

to the loins or hot baths may help to relax spasm. Inversion of the patient has been advised, to attempt to disengage the stone from the ureter. After the paroxysm is over, the aid of X-rays should again be invoked to locate the stone if it has not been passed.

HYDRONEPHROSIS

Definition.—A condition in which the pelvis and calices of the kidney are distended by the accumulation of non-infected urine due to ureteral or urethral obstruction.

Ætiology.—**CONGENITAL.**—The condition may be congenital, due to an abnormality of the ureter or urethra; other congenital defects may be present. The ureteral stricture is commonly found at the exit of the ureter from the pelvis of the kidney, or near its entrance into the bladder. Other congenital causes are a faulty connection of the ureter to the pelvis of the kidney, or an aberrant renal artery. Hydronephrosis is sometimes found post mortem in infants and children without evidence of obstruction to the outflow of urine. In these cases the condition is presumed to be due to a neuro-muscular inco-ordination comparable to congenital hypertrophic stenosis of the pylorus.

ACQUIRED.—It is more common in females than in males, and the maximum age incidence in 74 cases collected by Herringham was between the third and sixth decade.

(a) *Bilateral* hydronephrosis results from stricture of the urethra, phimosis, enlarged prostate, obstruction within the bladder, or from a pelvic tumour; the last named is the commonest cause.

(b) *Unilateral* hydronephrosis is due to ureteral obstruction from—

1. Obstruction of the lumen by a stone, growth or blood clot.

2. Stricture of the ureter following ureteritis.

3. Pressure from without due to growths.

4. Torsion of the ureter by displacement of a movable kidney. It is also thought that chronic prostatitis or cervicitis may cause sufficient inflammation to produce some dilatation of the kidney pelvis and upper ureter which lengthens and thus kinks the latter.

Pathology.—Two types of hydronephrosis are recognised, namely, the pelvic type due to upper urinary tract obstruction and the renal type from obstruction to the lower tract. In the former the pelvis of the kidney is dilated and there is less absorption of renal paracachyma in the calices. In the latter the calices are more dilated and there is considerable destruction of kidney substance.

It is generally held that hydronephrosis results from intermittent obstruction. It has been produced experimentally, however, by ligature of the ureter causing complete obstruction. But complete obstruction is more usually followed by atrophy of the kidney.

Symptoms.—Many cases are latent, and give rise to no symptoms. The tumour may be discovered accidentally, or there may be complaint of pain in the flank or back. The onset is insidious.

The symptoms by which a hydronephrosis is indicated are the presence of a renal tumour and complaint of an aching pain in the flank or back, and

sometimes polyuria or hæmaturia. In intermittent hydronephrosis, the tumour suddenly disappears with the passage of a large quantity of watery fluid; after an interval the tumour gradually reappears and finally empties suddenly as before. This sequence may be repeated at intervals. Where true polyuria or hæmaturia occurs it is due to a coincident nephritis or pyelitis. There may be acute exacerbations of the chronic pain, with vomiting and collapse; such attacks may accompany emptying of the hydronephrotic sac.

Course.—When unilateral, hydronephrosis may never cause serious trouble, and intermittent cases may persist for years and finally disappear. In bilateral cases uræmia may supervene. Infection of the kidney is not uncommon, and may lead to acute pyonephrosis. The sac may discharge spontaneously through the ureter, and the fluid never reaccumulate. The sac may rupture into the peritoneum, or rarely through the diaphragm into the lung. Cases have occurred in which the ureter of the sound kidney has been blocked by a calculus.

Diagnosis.—The condition, especially when bilateral and unaccompanied by symptoms, is generally overlooked. In its most characteristic form, where the hydronephrosis is intermittent, the diagnosis is readily made. When the condition is apparent simply as a renal tumour the diagnosis from renal neoplasm (or retro-peritoneal glands in a child) is difficult. When the tumour is large it may be mistaken for an ovarian tumour. The diagnosis can be established by intravenous pyelography supplemented, if necessary, by instrumental pyelography. Aspiration of the sac has been occasionally done for diagnostic purposes; but surgical exploration is a safer measure. Fluid from a hydronephrotic kidney is clear or slightly turbid; it contains albumin, and traces of urea and other urinary constituents; in the deposits are epithelial cells.

Prognosis.—This depends on the cause of the hydronephrosis and the condition of the opposite kidney.

Treatment.—The first indication is to remove the cause. Cases of intermittent hydronephrosis that do not cause serious symptoms should be treated on general lines. An abdominal belt to support a hydronephrotic mobile kidney may be of service.

In unilateral hydronephrosis causing serious symptoms, or of large size, von Lichtenberg's plastic operation or nephrectomy is advisable. Since the state and function of the opposite kidney can be fairly accurately ascertained by pyelography and examination of a sample of urine obtained by ureteral catheterisation, nephrectomy is a less serious risk than it was before these exact methods of diagnosis were available. Sympathectomy has been recommended, but it is not clear how this can produce the desired effect.

In bilateral hydronephrosis the main indication is to remove the cause when possible, and to adopt every measure that may aid in preventing infection of the urinary tract.

PYONEPHROSIS

Definition.—Distension of the renal pelvis with pus, to an extent sufficient to cause a renal tumour.

Ætiology.—The affection is a sequela of pyelitis or hydronephrosis.

There are two main types, namely, tuberculous and pyogenic pyonephrosis. The latter, which is the commoner, is most frequently due to an impacted calculus.

Symptoms.—The patient is wasted, toxic and febrile. Rigors are common. There is a renal tumour, which is tender on palpation, and moves to some extent with respiration. Pyuria is present, unless the ureter is completely obstructed.

Diagnosis.—The differential diagnosis from hydronephrosis is made from the presence of pyuria and of local and general symptoms of bacterial infection. Perinephric abscess gives signs of a more diffuse swelling, usually with œdema and redness of the surrounding skin, and does not move with respiration.

Treatment.—In bilateral cases the treatment is palliative. In unilateral cases nephrectomy is indicated, if tests show that the other kidney is adequate.

TUMOURS OF THE KIDNEY

BENIGN GROWTHS

These are of relatively slight importance.

ADENOMATA are the most common, occurring in the cortex or under the capsule. They may be single or multiple; multiple nodules commonly occur in sclerotic kidneys in old age. They seldom attain any size.

FIBROMATA are not uncommon as nodules, sometimes multiple, in the cortex or medulla. **LIPOMATA** and **ANGIOMATA** are rare.

MALIGNANT TUMOURS

DYSEMBRYOMATA.—These tumours are found most commonly in children under 3 years, and almost always under the age of 11 (Hadfield). They are more often bilateral than carcinoma. They consist of cells remaining at the embryonic level and failing to differentiate in any direction ("Round-celled Sarcoma"). There is a stroma of undifferentiated foetal connective tissue which resembles spindle-celled sarcoma. In some tumours some degree of differentiation may take place. Thus these tumours may contain embryonic striped muscle, primitive cartilage or nervous tissue, and primitive poorly-formed tubules can usually be found. They are yellow and homogeneous on section.

ADENOCARCINOMA OF RENAL TUBULES.—As a result of an examination of the 74 specimens of tumours of the kidney in the St. Bartholomew's Hospital Museum, Hadfield has come to the conclusion that renal tumours previously known as Hypernephromata are in fact Adenocarcinomata. These tumours are single, large, well circumscribed, and often surrounded by a capsule of compressed kidney tissue which is destroyed by pressure rather than by infiltration. These tumours consist of ^{small} of splintering columns of cells. Their blood supply consists of irregularly ^{small} lobed, lake-like sinusoids which lie between the tubules of the growth in contradistinction to adenomata, which are composed of well-formed tubules having well-defined lumina and a simple capillary circulation. Both in adenomata and

adenocarcinomata, and especially in the latter, the cells are infiltrated with a lipoid-fat-glycogen complex ("lipoid infiltration"), which gives these tumours their peculiar yellow colour. Recent and old hæmorrhage is commonly seen. Cystic degeneration often occurs. On section there is fine and coarse lobulation. These tumours may spread along the renal veins into the inferior vena cava, and to the pelvis of the kidney and perinephric tissues.

Symptoms.—1. Hæmaturia is the first symptom in more than 70 per cent. of the cases. It is much less frequent in children. The blood is fluid or clotted, and moulds of the pelvis or ureter may be passed. The hæmaturia is spontaneous, profuse and intermittent; it is little influenced by rest, nor is it provoked by exertion. It may be the only evidence of a neoplasm, and after lasting for a week or 14 days may cease, leaving no further evidence of the growth until at some later date a tumour is felt. The urine frequently contains albumin at intervals.

2. Pain is uncertain. It may be a dragging feeling, or a constant ache. The passage of clots may give rise to renal colic; otherwise the hæmaturia is not accompanied by pain.

3. The presence of a tumour is a most important sign. It is felt on deep palpation bimanually. It is first palpable below the ribs, outside the rectus muscle, as a solid swelling, with rounded borders, that moves with respiration. It may be possible to define its upper border. As the tumour increases, it tends to go forward. It may fill the hollow below the twelfth rib behind, but does not cause a swelling in the back. Large renal tumours cause asymmetry and bulging of the abdominal wall and marked displacement of neighbouring abdominal viscera. On the right side, the ascending colon lies in front, on the left the last part of the transverse colon and the upper part of the descending colon; the tumour is, therefore, resonant on percussion in front. When the tumour is highly vascular, pulsation is felt in it, and a systolic bruit may be heard over it. In later stages, the tumour is liable to become fixed by adhesions.

4. Progressive emaciation is generally late. It may be absent although the tumour is large.

5. Metastases are sometimes the first sign of a renal neoplasm, occurring in the lungs, bones or brain. Secondary deposits in the para-aortic lymph glands may cause obstruction to the inferior vena cava, or this may result from pressure of the tumour itself.

Diagnosis.—Diagnosis is made on the presence of hæmaturia, with a tumour. When hæmaturia occurs alone, and other causes have been excluded by careful clinical, bacteriological and X-ray examination, then a more detailed investigation of the urinary tract must be undertaken immediately. This entails cystoscopy, intravenous pyelography and on occasion retrograde pyelography. When a tumour is the only sign an exploratory laparotomy is advised. The tumour requires to be distinguished from splenomegaly, hepatomegaly and Riedel's lobe. A renal tumour has not the definite edge characteristic of splenomegaly and enlargement of the liver. Enlargement of the liver is often a source of difficulty. A Riedel's lobe is continuous with the liver, does not extend back into the loin, and is dull on percussion. Splenic tumours are recognised by the fact that they tend to occupy an oblique position in the abdominal wall, by the presence of a notch and of a

sharp inner margin, free movement with respiration, and dullness to percussion.

A differential diagnosis from retroperitoneal tumours, including those of the suprarenal, is not always possible, though the suprarenal growths may sometimes be recognised by certain characteristic features. Thus, there is the medullary sarcoma type described by Hutchison, generally occurring in children, characterised by metastases in the skull, ecchymotic swelling of the eyelids, papilloedema and severe anæmia, and the "infant Hercules" type of tumour of the adrenal cortex.

Prognosis.—The disease is almost invariably fatal. Many die within 2 years, and the majority within 4 years, though exceptional cases of survival for 5 to 10 years after operation have been recorded.

Treatment.—Surgical treatment alone holds out a prospect of cure. Symptomatic treatment includes the use of drugs for the relief of pain and the control of hæmaturia.

CYSTS OF THE KIDNEY

SOLITARY CYSTS

These may occur in an otherwise normal organ. They vary in size from very small cysts to tumours of considerable bulk. They result from dilatation of an obstructed tubule, and they may be congenital.

MULTIPLE CYSTS

Multiple cysts of small size are commonly met with in sclerotic kidneys. They result from chronic inflammatory changes that lead to obstruction of the tubules with subsequent dilatation. There are also rare cases of multiple cysts, of large size, whose ætiology and course are little known.

POLYCYSTIC DISEASE OF THE KIDNEYS

Definition.—Polycystic kidneys appear as a massive conglomeration of cysts, varying in size from a pin's head to a marble, separated by dense strands of fibrous tissue, in which little or no renal tissue is evident on naked-eye examination.

Ætiology and Pathology.—The commonest age incidence is between 40 and 50 years; they are relatively common in the decades preceding and following; they may occur in infancy and childhood, and of these a large proportion are in still-born infants. Those occurring in infants are congenital, and other congenital abnormalities may be present. The disease in adults is probably also congenital in origin. In this case it must be progressive, because the renal damage in the later stages is too severe to have been compatible with many years of active life. In this connection it is noted that the disease is often found in more than one member of a family and in successive generations. Its familial incidence, congenital origin, and association with cysts in other organs, especially the liver, all suggest that this disease belongs to the group of congenital-developmental errors.

The organs are enlarged in size, and weigh 20 to 30 ounces each, or even 3 to 4 lb. They have been compared to a bunch of grapes in appearance. The cysts project from the surface and form the mass of the organ. They are lined by a layer of flattened cells, and are filled with fluid. This fluid is clear or turbid, limpid or viscid, colourless or yellowish; it is sometimes blood-stained, giving it a red, purple or green colour. Urea has been found in the fluid, which may also contain fat globules, cellular débris, cholesterol and triple phosphate crystals. On microscopic examination more or less renal parenchyma is found in the septa between the cysts; the tubules are distorted, and exhibit varying degrees of atrophy, degeneration and dilatation, while the glomeruli show changes characteristic of chronic interstitial nephritis. The blood vessels of the kidney undergo sclerotic changes; there is increased fibrous connective tissue and small cell infiltration. In some cases cysts are also found in the liver, ovaries, broad ligament, uterus, pancreas and spleen; but they are rare in any other organ than the liver.

Symptoms.—The affection is nearly always bilateral. When the tumours develop to large size in the foetus, difficulty in labour may result. In the adult there may be no symptoms, or any of the symptoms of chronic nephritis may develop and may terminate in uræmia, cerebral hæmorrhage or cardiac failure. General arterial disease, with raised blood pressure and cardiac hypertrophy, is commonly present; on the other hand, the condition may reach an advanced stage and fatal termination without appreciable cardiac hypertrophy. In a third group the bilateral renal tumours are the most striking features, associated with general malaise, dull aching pain in the loins, and recurrent hæmaturia. The tumours are not tender, and present the ordinary signs of renal tumours (*q.v.*). The urine is of low specific gravity, and commonly contains a trace of albumin; there may be polyuria.

Course.—This usually follows that of “chronic interstitial nephritis.”

Diagnosis.—A condition of “chronic interstitial nephritis” with large palpable kidneys should suggest polycystic disease. Renal neoplasms other than sarcomata are nearly always unilateral. The absence of fever and pyuria excludes bilateral pyonephrosis.

Treatment.—The treatment is that of chronic nephritis. Operation is contra-indicated, since both kidneys are nearly always equally affected.

OTHER FORMS OF CYSTIC DISEASE

Echinococcus cysts may occur in the kidney, and the discharge of the daughter cysts has produced attacks of renal colic. *Cystic degeneration of renal neoplasms* is described elsewhere.

MOVABLE KIDNEY

The kidney is normally held in place by the perirenal fat, the renal vessels and the peritoneum stretched over it. But this does not prevent a certain amount of respiratory excursion, as may be seen either by X-ray examination or in the operating theatre. The range of movement varies between 1 and 2 inches, and is more marked on the right than the left side. The term movable kidney should therefore only be applied to cases where there

is an excessive respiratory descent, so that the upper as well as the lower pole can be felt, or where the kidney can be moved about by external manipulation. As the kidney slips downwards, the lower pole gradually passes towards the middle line, while the organ rotates slightly, causing the hilum to look somewhat upwards.

Ætiology.—Movable kidney is about seven times more common on the right than on the left side. The ascending colon and the hepatic flexure lie on the inner aspect of the right kidney, thus tending to drag it down when the bowel is loaded or dropped. On the left side, on the other hand, the strong costo-colic fold suspends the splenic flexure much more securely, while the descending colon lies to the outer side of the left kidney.

The condition is much commoner in women than in men. In men the kidney pouches are deep, narrow and rapidly diminish in breadth from above downwards, while in women they are much shallower and broader, and diminish only slightly in breadth from above downwards. This natural difference is accentuated in the spare long-waisted women with narrow loins, who are recognised as specially liable to floating kidney. The greater liability of women to chronic constipation further helps to induce dropping on the right side.

Pathology.—Many reasons have been given for the occurrence of movable kidney; but few will stand investigation. Wasting with loss of perirenal fat, or loss of tone in the muscles of the abdominal wall, have been held responsible, but movable kidney is so common apart from such conditions that their importance is doubtful. Glénard emphasised the frequency with which movable kidney is associated with a general visceroptosis; indeed it is rare to find a movable kidney without coloptosis. Naturally, if there is general visceroptosis, the kidney is its most obvious sign. It is a firm organ which can be readily grasped, while the other dropped viscera would elude palpation. As Landau says, "Pleased with his discovery, the physician may impute all subsequent symptoms to the movable kidney." Most of these are really due to visceroptosis.

A serious sequel is the occasional occurrence of hydronephrosis produced by torsion of the ureter during the forward rotation of the organ or by its becoming kinked over the renal vessels. If hydronephrosis occurs, a subsequent infection may convert it into a pyonephrosis.

Symptoms.—There may be no symptoms at all and, if the movable kidney is only discovered in the course of routine examination, it is better not to tell the patient of its existence. It may be well, however, to inform a reliable relation, if such can be found, in order to protect oneself against a less discreet medical attendant subsequently revealing the fact to the patient. The commonest symptom is a constant dragging pain owing to traction on the renal plexus. This most frequently first declares itself between 25 and 35 years of age. A zone of hyperæsthesia corresponding to the distribution of the tenth thoracic segment may also be present. More serious symptoms directly due to movable kidney are Dietl's crises; but these are not common. The attacks are characterised by intense pain radiating down the ureter and through the back, shivering, nausea, vomiting, fever and collapse. The urine is scanty, and may contain blood. Sometimes the pelvis of the kidney may become distended, giving rise to an obvious increase in the size of the organ. This may pass off later, with abundant

discharge of urine, showing that the crises are due to kinking and consequent partial obstruction of the ureter. If repeated, they may lead to hydronephrosis.

The other symptoms which have been attributed to floating kidney are really due to the associated visceroptosis (*q.v.*). But there is no reason to attribute far-reaching nervous consequences to movable kidney, yet, for some enthusiasts, hysteria in women, hypochondriasis in men, and even insanity, are common outcomes. There is little doubt that far too much stress has been laid upon this condition as a cause of manifold complaints.

To detect a movable kidney on the right side, the left hand should be placed under the loin while the patient is recumbent, though some authorities prefer a semi-recumbent posture. The patient should then be told to take a deep breath while the right hand is placed just under the edge of the liver in the nipple line. The kidney may then be felt to slip between the fingers. Usually, this does not cause the patient a definite pain, but a dull, sickening sensation. In the more advanced degree of the condition, the organ may be felt far from its normal position, even to the left of the middle line or nearly down to Poupart's ligament. In examining on the left side, the observer should stand on the patient's left, placing his right hand behind the loin and palpating in front with his left.

A movable kidney usually feels larger than the normal excised organ. This is because of the surrounding investments through which it is felt.

Diagnosis.—Usually this is obvious, as the shape and mobility of the organ are so characteristic. Occasionally, a Riedel's lobe has been taken for movable kidney; but the continuity of the former with the liver should prevent this mistake being made. In the same way, a distended gall-bladder is continuous with the liver, and cannot be separated from it. Moreover, it is not nearly so movable, and curves characteristically towards the umbilicus. Carcinoma of the pylorus has offered difficulties in some cases; examination of the stools for occult blood, a test-meal and X-ray examination would clear up the diagnosis. Scybala near the flexures of the colon may be mistaken for floating kidney; but their indefinite shape and inelasticity generally help to distinguish them. Their disappearance after a series of enemata would settle the question. In one case a mesenteric cyst appeared closely to resemble a floating kidney.

Prognosis.—Apart from the development of hydronephrosis, movable kidney does not tend to shorten life in any way. It is doubtful whether a kidney once prolapsed can ever maintain the normal position unaided.

Treatment.—Some cases call for no local treatment, though the associated visceroptosis and neurasthenia will require attention. If pain is felt, the adoption, for a short time, of the knee-elbow position will help to replace the kidney and relieve the tension on the renal plexus. If pain is at all frequent, some form of abdominal support, such as a specially designed corset, should be worn. Hurst has urged that the support should be designed to increase the general intra-abdominal pressure, and not to replace any one viscus. We are convinced that this is sound and that, in many cases, a "kidney belt" is worse than useless, while the addition of ingeniously placed pads only increases the discomfort. Whatever the form of the support, it need only be worn while the patient is in the erect posture, and it is best fitted while she is recumbent, preferably with the pelvis raised on a pillow so as to

aid the replacement of the kidney. Often, when a support of this kind has been worn for a year or two, it is possible to give it up without recurrence of symptoms. Breathing exercises to develop the expansion of the lower thorax, with exercises to improve the tone of the abdominal wall have in many cases proved more efficacious than a passive support. Operation should not be advised except for recurrent Dietl's crises or when there is evidence of hydronephrosis, when nephropexy may be done. But even this may not be successful, and ultimately nephrectomy may be required for the hydronephrosis.

Treatment of Dietl's crises.—The patient must be put to bed and hot fomentations or antiphlogistine applied to the affected side. A hypodermic injection of a quarter to one-third of a grain of morphine may be required if the pain is severe. Usually this is sufficient but, should the attack last more than a few hours, an attempt must be made, under an anæsthetic, to rectify the position of the kidney by manipulation. Naturally, conditions are unfavourable for nephropexy during or immediately after a crisis, because of the congested state of the organ.

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SECTION XVII

DISEASES OF THE JOINTS AND INFLAMMATORY DISEASES OF THE FIBROUS TISSUES AND MUSCLES

ARTHRITIS

The diagnosis of "arthritis" should be reserved for cases in which there are pathological changes at the surface of a joint. In the past there has been a tendency to employ this term too loosely.

The clinical conception of arthritis is much simplified by modern classification, which divides it into two clear-cut *clinical* types, each of which presents distinctive features. These are the rheumatoid arthritic type and the osteo-arthritic type. Consequently such terms as "arthritis deformans" should no longer be used.

The features of the rheumatoid and osteo-arthritic types of arthritis will be found under their respective headings below, and since the criteria of these types are clinical, this terminology can be correctly employed in those cases in which the ætiology remains obscure.

The most basic difference between these two types is that rheumatoid arthritis is a generalized disease in which the most obvious local effects fall upon the locomotor system; while osteo-arthritis is a degenerative type of condition, which, without affecting the patient's general health, for various reasons becomes localised in certain joints.

There are, in addition, certain cases which are referred to as "Mixed arthritis," in which the degenerative lesions of osteo-arthritis become superimposed upon those of an inflammatory arthritis of the rheumatoid type.

1. RHEUMATOID ARTHRITIS

Synonyms.-- Atrophic Arthritis; Infective Polyarthritis.

Rheumatoid arthritis has generally been described as being of two types, namely, the "classical" or idiopathic type, of which the causation is unknown; and the infective type, in which a discoverable infective agent is causative. It was the opinion of the committee appointed by the Royal College of Physicians (1934) that the term "rheumatoid arthritis" is best reserved for the first type, and "infective arthritis" for the second. It is, however, generally impossible to distinguish between the two types on purely clinical grounds, and the distinction should therefore depend on the pathological findings. Either type may, however, be correctly referred to as the "rheumatoid type" of arthritis.

Rheumatoid arthritis is a generalised progressive disease affecting principally the joints, which are swollen and painful. If unchecked great destruction and deformity results.

Ætiology.—The malady is said to occur at least five times as frequently among females. The type of patient most commonly affected by the classical form of the disease is a young woman between 20-40 years of age, and of slender delicate build, with a somewhat narrow back and acute costal angle. The affection seldom commences after the menopause. Predisposing factors exist in many cases and include the climateric, chronic gout, malnutrition, emotional shock, and minor focal infection which is not in itself causative of the condition.

Pathology.—The pathological processes are inflammatory in nature. The soft tissues and the white fibrous structures around the joints are the first to be affected. The inflammation then spreads to the capsule and synovium, and granulation tissue forms in the angle made by the articular cartilage with the synovium. The latter then gradually extends inwards as a ring of "pannus," covering and eventually replacing the articular cartilage. As this happens on both articular surfaces the tendency is for them to adhere, especially if the joint is immobilised, and so fibrous ankylosis occurs, which in some cases progresses to a bony ankylosis.

—There are atrophic changes affecting the skin, subcutaneous tissues, muscles, ligaments, joints and bones. This latter condition of generalised osteoporosis shows as the first *X-ray* evidence of the disease. The peri-articular swelling can also be seen in outline, but actual joint changes do not occur until considerably later. Some degree of patchy recalcification may be observed when the progress of the disease is checked. Osteophytes are never found in rheumatoid arthritis, but in late cases very considerable disorganisation of the joints take place, and in these areas the bone sometimes gives the appearance of having been dissolved away.

The chief pathological change in the blood is an increase in the sedimentation rate of the red blood cells. This is an important index of activity, and the response of a patient to treatment over a considerable period can be estimated with some accuracy by means of this together with clinical observation. A secondary anæmia is usually present in the pre-arthritis phase. The glucose tolerance of the patient is generally found to be reduced in the active stages of the disease.

Symptoms.—There is in most cases a prodromal period, during which the patient loses a considerable amount of weight; and fatigue, both mental and physical, is a marked feature in nearly all cases. There may be other symptoms, such as paræsthesia, Raynaud-like phenomena, irregular menstruation, tachycardia, sweating, and a secondary anæmia.

The onset of the arthritic phase is often announced by a swelling of the mid-phalangeal joints of the second and third fingers of both hands. It is usually insidious, but is acute in about 10 per cent. of cases. In the case of the former, it is not uncommon for the disease to be marked by long periods of low and intermittent fever. The cause of this is not known. The thyroid gland is also sometimes enlarged, and fibrositic pains may be complained of.

Wasting of the small muscles of the hands is generally the next event, while the uncompensated pull of the interossei muscles, which appear to be affected a little later in the disease, tends, in combination with trauma, to drag the fingers into the typical position of ulnar flexion, in which they often become ultimately fixed. The affection then spreads centripetally

towards the trunk, involving in turn the wrists (which often become the seat of ankylosis), ankles, elbows, knees, shoulders, hips and jaw. The bilateral and symmetrical way in which all the joints are affected is a striking feature of the disease. This point, however, is not so well marked in cases of infective arthritis as in the idiopathic type of rheumatoid arthritis. In some cases the spine itself in due course becomes affected. (See p. 1362).

Whenever a joint becomes involved it will be noticed that the muscles which control it, particularly the extensors, waste rapidly, giving rise to the varying flexion deformities seen in the later stages. These may be perpetuated by a fibrous ankylosis of the affected joints, and contraction of the joint capsule. Bony ankylosis may follow this stage, and when it does so it generally occurs in the wrists and the bones of the carpus in the first place.

In certain cases enlargement of the lymphatic glands occurs, and even the spleen may become palpable. Sufferers with rheumatoid arthritis generally experience considerable pain which interferes with their sleep, and this adds progressively to the severity of the condition.

Symptoms often clear up during pregnancy occurring during the course of the disease, but in most cases they return with renewed vigour after parturition. It should not be forgotten that periodical intermissions are a well-observed occasional feature of the disease, which sometimes results in undeserved credit to any treatment which is being undertaken at the time.

Prognosis.—Under properly planned and supervised treatment, about 20 per cent. of patients should prove completely amenable to therapy, a further 50 per cent. should show great or moderate improvement, and an additional 20 per cent. are improved to some extent; leaving 5–10 per cent. of cases which appear to be entirely resistant to treatment of all kinds. With modern methods of splintage, gross deformity should very seldom occur, even when ankylosis takes place. It should be remembered, however, that in many cases treatment of some sort for months or even years is required, and that relapse may occur after apparent cure. Enlargement of the lymphatic glands has often seemed to be of bad prognostic significance, especially when the white cell count is profoundly altered in addition, and subcutaneous nodules appear. If the disease is not checked, the end-result is complete and painful crippledom. This often takes place within a very short period, particularly when the patient is young. Cases which occur later in life tend on the whole to be less virulent in their course.

Treatment.—As yet there is no specific form of therapy. It is by a careful and intelligent selection and combination of methods suited to the individual patient that success will be achieved.

Diet.—This should be rich in vitamins, especially B and C, and should be of the high caloric type, except in the rare cases in which the patient is overweight. When the sufferer is much underweight and does not return to normal by dietetic means alone, a small dose of insulin (5–10 units) may be administered 15 to 20 minutes before two meals in the day, which should be rich in carbohydrates.

Physical Therapy.—In the acute phase, most methods of external treatment will prove too exhausting and are therefore undesirable. An exception is progressively graded ultra-violet rays, which stimulate the skin, and to some extent the patient's powers of resistance. Later, massage and heat

will help to relax the muscle spasm, which is often a cause of pain and ultimate deformity.

Drug Therapy.—This has only a limited scope in arthritis of all types, and should never be the sole method of treatment employed. On general principles such patients need iron for anæmia, laxatives for constipation, and analgesics—especially aspirin, phenacetin, and, when necessary, in addition Dover's powder (gr. 10–15), or codeine phosphate (gr. $\frac{1}{4}$ – $\frac{1}{2}$)—for pain and sleeplessness. A valuable remedy is guaiacol carbonate, which is an intestinal antiseptic and analgesic. It may be given in doses of 5 to 10 grs. three times daily after food. A useful combination is guaiacol carbonate, grs. 8, aspirin, grs. 4, in cachet form, three times daily after food. Guaiacol carbonate is free from any danger of toxic symptoms. Arsenic is of considerable tonic value, especially if combined with nux vomica and taken before meals. Cod-liver oil also has great value and, if preferred, may be combined with malt extract. It should be given in full doses of one ounce two or three times daily if such can be tolerated. Thyroid is useful in cases occurring about the menopause. Iodine in the form of liquor iodi simplex once daily is useful in some cases.

Injections of gold salts have recently been found helpful in many cases suffering from the idiopathic type of the disease, especially when the onset is fairly recent and the blood sedimentation is high. When the malady is known definitely to be the result of an infection their employment is inadvisable until this has been eradicated.

This method of treatment is contra-indicated with renal or hepatic damage, diabetes mellitus, eczema, severe anæmia, colitis, pregnancy, hæmophilia, or a history of having suffered from any severe alteration in the white-cell count in the past, or purpura. With regard to other cases, the dangers of reaction, complications and mortality should be taken into consideration. There should be a complete blood count, blood sedimentation test, and an examination of the urine for albumin. The chief complications are purpura, rashes, boils, exfoliative dermatitis, gastro-enteritis and colitis, nephritis, jaundice, aplastic anæmia and stomatitis. There is a mortality of about 1 per cent. attendant upon this form of therapy. The patient herself should decide after being informed of the danger of reaction, complications and mortality. She should not be allowed to expose herself to strong sunshine or ultra-violet light, for fear of pigmentation.

It is well not to administer gold near the period of the menses, as skin eruptions are said to be more liable to occur then. There are several preparations of gold salts on the market. It is wise always to employ those which are administered intramuscularly. Whether they are suspended in an oily or in an aqueous solution appears to be immaterial. The initial dose should be 0.01 gm., and subsequent doses may be 0.05 and 0.1. This latter dose should not be exceeded, nor should the injections generally be given more frequently than once weekly. The total amount in a complete course should in most cases be limited to 1–1.5 gm. An interval of at least six weeks should then elapse before a further course is commenced. The dosage should be adjusted according to the patient's condition and response to previous injections. Patients of very light weight probably require smaller initial doses and more careful subsequent grading. A complete blood count, blood sedimentation rate test, and urine examination for albumin should be

repeated at regular intervals during the treatment. Great care should be exercised in watching for the first signs of reaction, or any other complication. In the first instance, no further injections should be given until 48 hours after the reaction has entirely subsided. In the second, the injections should be stopped immediately, and not resumed, if at all, until the patient has been free for two to three weeks. If complications appear treatment is palliative, while if the skin is involved also, calamine lotion, with 1 per cent. phenol, should be applied, and a mixture containing bromide and phenobarbitone (gr. $\frac{1}{2}$) should be administered thrice daily until the complication has disappeared. Patients with jaundice should be put to bed and treated as if suffering with catarrhal jaundice. For the other complications 10 c.c. of a freshly prepared 20 per cent. solution of sodium thiosulphate should be given intravenously every day, and 10 c.c. of 10 per cent. calcium gluconate may be administered intramuscularly at the same time.

Vaccine Treatment.—In infective arthritis a course of vaccine injections is a rational form of therapy. In certain cases it is very successful in reducing the activity of the disease. But it should, however, not be expected to take the place of the simple orthopædic and other measures necessary to prevent contracture of the joints. When the patient is suffering from idiopathic rheumatoid arthritis the results of vaccine therapy are less certain. But it is often worth a trial, either previous to the employment of gold therapy or in the interval between the courses. In cases in which gold is contra-indicated or is not well tolerated, it may be the method of choice, except in those who are febrile and much exhausted, when the powers of reaction are very low and harm may result. The right dose is the lowest which is found to provoke a favourable reaction, and not the highest which can be tolerated, as is believed by some.

Protein shock may be given in the form of intravenous T.A.B. vaccine injections. This is a non-specific procedure designed to raise the patient's temperature temporarily in the hope of benefiting him subsequently. Such treatment should never be undertaken when the patient is in an active phase of the disease. Once improvement has started, however, it may be justifiable to endeavour to speed its tempo by this means. Three to five injections should be administered, the dose varying according to the age and weight of the patient. At least 24 hours of normal temperature should be allowed between the injections, which should not in any case be given more frequently than twice weekly.

Additional methods of treatment useful in certain cases include blood transfusions in those in which improvement by other means is long delayed; colonic lavage when there is reason to believe that the lower bowel is implicated; and sulphur injections, which have a similar object to protein shock.

Finally, certain considerations regarding *focal sepsis*: It is a safe rule in such a disease such as rheumatoid arthritis, in which the exact ætiology is often obscure, that "whatever is found wrong—put it right!" This should extend to the discovery of foci of infection. It is, however, unwise to embark upon operative procedures while the patient is in a condition of debility, or while the disease is running an acute febrile course, with marked joint pain and swelling. In such patients an endeavour should previously be made to build up the general health. If after 4–8 weeks no improvement has occurred and the focus is still believed to be of importance, cautious

measures for its removal may be initiated. The patient should in such cases be warned that it is unlikely that the removal of such a focus will cure the arthritis, but that his general health, and the powers of active resistance will be stimulated thereby. The foci of infection which are of particular importance and should always be investigated are situated in the tonsils, the sinuses, and teeth—in that order. Less important foci include the colon, the appendix, gall-bladder, cervix, tubes, prostate and bladder. If more than one focus of infection is found the most apparently active should be treated first. If this cannot be determined the matter should be dealt with in the order mentioned above. In cases in which the sinuses and the tonsils both require surgical attention it is important to allow a period of several weeks to elapse between the two operations.

2. SPECIFIC INFECTIVE ARTHRITIS (OF RHEUMATOID TYPE)

GONOCOCCAL ARTHRITIS

From 1 to 5 per cent. of those infected with gonorrhœa develop gonococcal arthritis. The latter is also found in babies, whose infection occurs at birth. In view of the fact that infection due to this cause frequently ends in great crippling and bony ankylosis of the joints, it is important to make the diagnosis at the earliest possible stage. If this is done the prognosis is now good.

The clinical appearance and course of gonococcal arthritis is similar to that of the idiopathic type of rheumatoid arthritis when the onset of the latter is acute. A differential diagnosis may be made from the following points: (1) A recent history of gonococcal infection or urethritis. Unless specifically questioned, patients often omit to mention this. (2) The onset of joint symptoms within three weeks of such an infection. (3) There is a predominance of 3 : 1 in males, unlike true rheumatoid arthritis. As in the latter disease, however, the malady is usually a polyarthritis from the onset. (4) The knees, wrists and ankles are generally the seat of the most virulent attack; while a painful teno-synovitis around the wrists and ankles is a common forerunner of actual arthritis. (5) Conjunctivitis and irido-cyclitis is not uncommonly associated with gonococcal arthritis.

If pathological aid is available the complement fixation test of the blood is found to be positive in about 80 per cent. of cases after the first month. Gonococci may in many cases also be grown by special methods from samples of the joint fluid, which affords an immediate confirmation of the diagnosis.

The main points in treatment are (1) that of the primary focus and prostate; (2) the sulphanilamide group of drugs; and (3) artificial fever therapy (hyperpyrexia). The last, whenever it is available, is considered by American observers to be "specific" for gonorrhœal polyarthritis.

In the chronic stages cases should be treated on the same lines as rheumatoid arthritis, except that gold salts are contra-indicated. (See also p. 23).

PNEUMOCOCCAL ARTHRITIS

A polyarthritis clinically of the rheumatoid type is a rare sequel of lobar pneumonia. It affects children more commonly than adults. An arthritis

affecting one or more of the larger joints is somewhat commoner. These conditions will generally occur subsequent to the stage of pneumonic resolution. Primary pneumococcal arthritis of either type is uncommon.

Pathology.—The joint fluid is in most cases purulent, and pneumococci can be found in it, except in cases which have received sulphapyridine in large doses.

Prognosis.—If the patient survives the pulmonary infection his resistance to the organism should be good, and joint function is in most cases preserved provided erosion of the cartilage has not taken place.

Treatment.—Joints affected in this way should be aspirated early and irrigated with isotonic saline solution, although drainage is not advisable. They should be immobilised in light plaster splints, which should be removed daily to permit of gentle movement. Sulphapyridine should be given in full doses.

ACUTE SUPPURATIVE ARTHRITIS

This is often polyarticular in its distribution, and may be mistaken at first for acute rheumatic fever or rheumatoid arthritis. It is commoner in children than in adults.

Ætiology.—The condition may be a blood-borne infection (metastatic), or may arise as an extension from neighbouring areas of osteomyelitis, or other infection. The former is the more common and may be secondary to a focus of infection in the middle ear, throat, sinuses or prostate. It may also follow the acute specific fevers, particularly scarlet fever and septic tonsillitis. It has also been reported as following meningitis, septic endocarditis, infected varicosities and burns, pyelitis and furunculosis. An arthritis following typhoid fever is not generally suppurative, but may become so.

The organisms which are chiefly responsible are the hæmolytic streptococcus, the staphylococcus, the pneumococcus and the gonococcus.

Symptoms.—These include an acute onset of chills and sweats, pyrexia, local pain and tenderness in the joints, with redness, swelling and limitation of movement. There is in most cases a high degree of polymorphonuclear leucocytosis.

Course.—The joint fluid rapidly becomes purulent and extensive damage to the joints will occur if treatment is not instituted rapidly. Badly damaged joints generally ankylose ultimately. The mortality amongst such cases is in the neighbourhood of 20 per cent.

Treatment.—This should be directed towards the primary source of the infection as well as the affected joints. If the organism is known to be a streptococcus, pneumococcus, gonococcus or meningococcus, drugs of the sulphonamide or sulphapyridine group will be of value. But they are ineffective in cases of staphylococcal arthritis, in which condition sulphathiazole may be of benefit.

Immediate aspiration of the affected joints is essential, both for diagnosis and therapeutically, and lavage with normal saline should be done. In some cases it is necessary to open the joint for this purpose. Blood transfusion is valuable in these cases; and orthopædic care is required if ankylosis appears probable. Hyperpyrexia treatment is useless in the majority of cases.

TUBERCULOUS ARTHRITIS

Tuberculous arthritis usually occurs in young patients, and is an infection from a primary tuberculous focus elsewhere in the body, and frequently signs of tuberculosis in other parts of the body are present. The possibility of an arthritis in a young subject being tuberculous should always be borne in mind, and an X-ray examination is of great value in differentiating this type from other varieties. The subject is dealt with fully in surgical books, to which the reader is referred.

DYSENTERIC ARTHRITIS

A polyarthritis of the rheumatoid type follows bacillary dysentery in about 3 per cent. of cases, at an interval varying from three weeks to several months after the cessation of acute symptoms. It may also occur in the course of a chronic ulcerative colitis. In some cases the process will only affect one joint, but in either event the process commences as an inflammation of the periarticular tissues and progressively invades the joint surfaces. Suppuration is very rare.

Treatment will be directed to the dysenteric condition, and should be palliative so far as the joints are concerned.

UNDULANT FEVER OR BRUCELLIASIS

A mild polyarthritis due to the organism of this disease is probably more frequent than is usually believed. It is generally associated with myalgia, and sometimes with intermittent hydrarthrosis. The onset may be acute or chronic, and the clinical picture may closely resemble rheumatoid arthritis or rheumatic fever. Cases have also been confused with pulmonary tuberculosis and "neurasthenia."

The general symptoms are indefinite and multiple, and include malaise, long-continued low-grade pyrexia, which "undulates," loss of weight, sweating and depression. The blood may show a secondary anæmia and a leucopenia with relative lymphocytosis. The agglutination tests will probably also be positive if the disease is of some weeks duration.

DENGUE

This disease gives rise to a very acute form of peri-arthritis. Intense pain and sometimes swelling occur in the tendons and muscles around the joints. These usually disappear when the fever subsides, but in the stage of convalescence may recur and last for weeks or months. The condition should be differentiated from rheumatic fever, from which it differs in being epidemic and in not responding to salicylates.

MENINGOCOCCAL ARTHRITIS

This is not uncommon in the second week of cerebro-spinal fever, and may be polyarticular or monarticular. It sometimes becomes purulent. It resembles gonococcal arthritis closely, except that it is generally less severe. There is also a sporadic form—chronic meningococcal septicæmia—

which occurs in the absence of meningitis. The diagnosis in these cases rests upon the presence of intermittent fever, a rash, which may be purpuric, and a positive blood culture. The fluid from the joints affected may contain the organism. The patients often seem surprisingly well, and complain of little except joint pains. Both these types respond well and rapidly to sulphapyridine therapy.

3. OSTEO-ARTHRITIS

Synonyms.—Hypertrophic Arthritis; Arthrosis; Arthritis Deformans; Morbus Coxæ Senilis (of hip).

Osteo-arthritis is a degenerative condition which affects the articular cartilages and weight-bearing surfaces of the larger joints.

Ætiology.—The known factors include trauma, certain disorders of metabolism, and nervous diseases, *e.g.* Charcot's joints. Senility is also a cause, as may occasionally be prolonged toxæmia. There is, however, no evidence that focal sepsis is primarily concerned in the ætiology of this type of arthritis.

The malady tends to occur principally in men over middle life who have led a strenuous existence; while in women it mainly affects the knees and is secondary to the proliferative synovitis which is often a distressing feature of the menopausal period.

Pathology.—The changes which occur in the joints affect primarily the articular cartilage, which in the early stages will show grooving and "fibrillation," starting at the points where the pressure of the opposing surfaces is greatest. Later, the cartilage may actually wear through at these points, and the two bony surfaces will come into contact. When this happens, the constant rubbing of bone on bone will gradually polish and "eburnate" these areas. At the same time a gradually progressive enlargement of the articular surfaces will occur, which culminates in the production of "lipping" and of bony outgrowths from the joint margins called osteophytes. These excrescences may be easily palpable at the joint margins, and are the typical lesions of osteo-arthritis. No constant changes are found in the synovium.

Symptoms.—The onset of the disease is insidious. The first symptom to be complained of is usually stiffness, often accompanied by some pain after exertion. The site is generally one or more of the larger joints; or it may be any joint which is subjected to particular stresses as the result of the patient's occupation or sport. In the course of time considerable wasting of the muscles controlling the affected joints supervenes. These therefore tend to become unstable and so liable to further trauma. The coarse grating which can be elicited in joints affected with the disease is due to an accompanying teno-synovitis, and is no measure of the actual damage to joints. When extrinsic joint changes have occurred, the patient usually experiences considerable pain, particularly on bearing weight. The movements of the joint also become much limited on account of spasm of the surrounding muscles, which may in itself be a cause of pain. There is generally not much effusion present. Occasionally new bone formation may limit the movements of the joint, although this is not very common. Small rounded bony swellings on the terminal phalanges of the fingers and thumbs, termed

Heberden's nodes, not infrequently develop during the course of the disease. These may be the cause of considerable pain in their early stages.

The examination of a hip joint affected with early osteo-arthritis will reveal some limitation and pain on rotation and often also of abduction of the joint, long before the movements of flexion and extension are appreciably interfered with. In addition, it may be found that such a patient when standing will not support his weight equally on both hips, in order to avoid pain. Some wasting of the gluteus muscle on the affected side will also be evident fairly early; while in advanced cases actual shortening of the affected limb will occur, either the result of absorption of the femoral head or from its dislocation upwards. Another point to remember is the possibility that even severe pain complained of in the knee may in reality be referred from a diseased hip; and in such cases if a full examination of the patient is omitted treatment may be directed to the wrong joint. "Sciatica" is often found to be the result of osteo-arthritis of the hip, or of the lumbar spine, and is sometimes the initial symptom.

Osteo-arthritis of the lumbar spine is frequently present without giving rise to symptoms. Such cases are often discovered radiologically in the course of an examination for another purpose. This sometimes gives rise to difficulty in compensation cases when existing symptoms may be attributed to this cause *post hoc*. The sacro-iliac joints are in the same way frequently reported as being the seat of osteo-arthritis. In many cases, however, there is an absence of symptoms and so no treatment is required, unless low backache or sciatica supervenes.

The osteo-arthritic joint does not ankylose, but may become locked as the result of excessive osteophyte formation. In other cases it becomes unstable, owing to continued use in the presence of insufficient muscular support, due to muscular wasting; in these cases the joint surfaces may ultimately become very disorganised.

When a weight-bearing joint is affected, the patient suffers great pain on standing, and a certain amount of absorption of articular bone may occur, resulting in some shortening of the limb.

In some cases the articular cartilage may become fragmented, or osteophytes may break off into the joint cavity. In both these circumstances they form loose bodies which give rise to all the symptoms usual in that condition, in addition to those of the arthritis.

The results of radiological examination and blood sedimentation rate and glucose tolerance are described under Diagnosis.

The general health is not affected, unless the result of unaccustomed inactivity necessitated by the affection.

Diagnosis.—Osteo-arthritis should never be confused with rheumatoid arthritis, the two conditions being entirely different. The former is a degenerative condition affecting one or two joints, usually the larger ones. There is generally a history of trauma in the past, or of continued trauma of a minor nature, such as some occupational or sporting stress or strain; or a postural defect; or in some cases a sudden increase of weight, with resultant strain on the ankles, knees, or hips, as may occur at the menopause. Apart from the menopausal group of cases, the patient is more often a male, and his general health is not directly affected by the disease. Again, the pain is generally relieved by rest.

In rheumatoid arthritis, on the other hand, the joint changes are inflammatory in nature, and trauma is not a marked feature. The onset is in the smaller joints, many of which are generally affected, symmetrically and bilaterally. Finally, the patient is usually a woman, and there are indications of general ill-health and also loss of weight, which often preceded the joint manifestations.

The *radiographic* appearance of a joint affected with osteo-arthritis is generally typical. The bone density is unaffected and the joint space is narrowed; this depends upon the amount of erosion which has occurred in the cartilage. Osteophytes will be seen at the joint margins, and there is frequently also a deposition of calcium in the attached ends of certain tendons, such as those of the patella and ligaments, *e.g.* the cruciates, which result in an appearance of "spiking." Such, however, should not be confused with osteophyte formation, as it will sometimes be found independently of the existence of osteo-arthritis. In osteo-arthritis of the hip considerable deformity, both of the head of the femur and of the acetabulum, may be seen, and, in addition, small degenerative cystic areas adjacent to the joint are not infrequently noticed.

The blood sedimentation rate and the glucose tolerance are normal.

Differential Diagnosis.—Conditions which are liable to be confused with osteo-arthritis are Paget's disease, osteochondrosis desiccans, and occasionally neoplastic growths of the articular ends of bones.

Prognosis.—If not treated the course of osteo-arthritis is progressive and generally ends in disablement. Much, however, can be done in the early stage to prevent this, by relieving the affected joint of all possible strain, and by support and correct treatment. The outlook is perhaps best in that form known as "menopausal arthritis," provided full and adequate measures are taken before the malady becomes established. In such cases a return to the normal may be anticipated in the course of time.

Treatment.—As in osteo-arthritis there is an absence of general symptoms, there is no indication for general treatment other than seeing that the patients receive an adequate supply of vitamins and that their bowels act regularly, while definite periods of rest are advisable. If the patient is obese, a diet low in fats and carbohydrates should be insisted upon if success from other measures is to be achieved. The caloric value should not exceed 900–1600 calories, and fluids should not be taken at the same time as food. The patients should be reassured that they will not become extensively and hopelessly crippled, as may occur in rheumatoid arthritis.

It is the experience of most observers that little benefit is to be anticipated as the result of removing foci of infection. If, however, these are found they should be dealt with on their merits. If varicose veins are present, as is often the case when the malady is situated in the knees, treatment directed towards these will frequently result in improvement in the joints.

The drugs chiefly valuable in this condition are those of the analgesic group, such as aspirin, guaiacol carbonate, phenacetin and occasionally codeine. They should not be prescribed as a routine and should only be used when pain is severe. The iodides are also employed on general principles, and appear to be of value in some cases, as are sulphur (by mouth) and guaiacum. Gold salts should not be used in this type of arthritis.

In cases which arise about the time of the menopause small doses of thyroid are often of value, but should be combined with the other measures outlined, particularly those for reduction in weight, muscular re-education and joint support by means of elastoplast.

Physical therapy of some sort is essential in treatment of osteo-arthritis. The desiderata are: heat, to stimulate the failing circulation locally and to relieve muscle spasm and consequently pain; massage, to maintain and stimulate the nutrition and drainage of the skin and underlying tissues; and movement, in order to maintain the mobility of the joint, to prevent or repair muscle wasting, and discourage the formation of adhesions.

Movement should, if possible, be active, *i.e.* special exercises, or if this is not feasible at the outset, electrical stimulation by means of the surging faradic current, or hydrotherapy, if available, may be substituted. Movement of the affected joint should, however, as far as possible be disassociated from weight-bearing through the affected joint, in order to allow of repair in the cartilage.

A further method of stimulating the circulation reflexly is by means of histamine ionisation. Sufficient current should be used to produce a temporary urticarial reaction in the skin under the pad. When dealing with the extremities, paraffin wax applied at a high temperature is also useful as a means of applying heat.

The question of posture, or "body mechanics," is an important and neglected aspect of many cases of osteo-arthritis. For instance, pronated feet may cause strain and, later, arthritis in both knees and lumbar spine; as may a pendulous abdomen. Proper postural exercises should be taught, which the patient should continue until the correct posture is maintained reflexly.

When after a period of rest the patient begins to put weight on to the affected joint again, the latter should always be adequately supported. For this purpose a crêpe bandage, or better still elastoplast, is of great value. In some cases special appliances, such as the Howard-Marsh splint for the knees, or a back support when the spine is affected, may be needed for a period in order to protect the joint. For the feet proper arch supports may be necessary, while in cases of severe arthritis of the hip or knee, when weight-bearing continues to give great pain, some form of walking caliper, whereby the weight of the body is "by-passed" from the ischium down to the heel of the shoe by a light metal rod, is indicated.

In very advanced cases operative procedures will sometimes prove necessary. Chief amongst these are synovectomy; in those cases in which soft tissue proliferation is not responsive to other treatment. When in the hip-joint a small degree of painful movement is all that remains possible of achievement, arthrodesis, either by open operation or by means of the Smith-Petersen pin, will often be the best method of treatment. Multiple bone puncture has recently been advocated (empirically), but no controlled series of the effects of this has yet appeared.

Recently X-ray treatment has been considerably employed as an anæsthetic procedure in painful osteo-arthritis. The effects are very variable but on the whole appear to be more successful when it is the superficially placed joints which are to be treated.

Finally, the question of climate should be considered. If the economic

status of the patient will allow him to live in a warm climate, this will in most cases prove to be very beneficial.

4. HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY (see p. 1375)

5. ARTHRITIS DUE TO GOUT (see pp. 432, 434)

6. SPONDYLITIS

Spondylitis is arthritis of the spine. Most cases fall into one of three types: (1) the ankylosing type—spondylitis ankylopoietica; (2) rheumatoid arthritis, in which the spine is involved as a secondary spondylitis; and (3) osteo-arthritic type of spondylitis.

1. ANKYLOSING TYPE OF SPONDYLITIS

Synonyms.—Ankylosing Spondylitis; Spondylitis Ankylopoietica; Atrophic type of Spondylitis; Spondylose Rhizomélisque; Von Bechterew's Disease; Marie-Strumpell Disease. It is now realised that all these terms apply to the rheumatoid type of spondylitis or sub-varieties of this.

Ætiology.—As the condition is a form of rheumatoid arthritis with a special localisation to the spine, the predisposing and exciting factors are the same as in that disease (*q.v.*). The age of onset is also similar, occurring chiefly in the young. The malady differs, however, in chiefly affecting males. It is considerably less common than the classical form of rheumatoid arthritis.

Pathology.—The only true joints in the spinal column are those which permit of movement between the intervertebral articular facets and the costo-vertebral joints. This type of arthritis commences as a synovitis of these joints, together with some osteoporosis of the vertebral bodies. In the vast majority of cases radiological evidence of an infective process will be found in the sacro-iliac joints, concurrently with these changes. The nature of this is unknown. The ligaments surrounding the affected joints subsequently calcify, and those portions of the spinal column become rigid. When this process is complete the lateral borders of the intervertebral discs also calcify, as do the anterior and lateral longitudinal ligaments, the whole process resulting in a bamboo-like appearance which is typical of the condition.

Symptoms.—The early symptoms of the rheumatoid type of spondylitis are usually of a diffuse and insignificant nature, and are therefore generally overlooked until they are localised in the spinal region, which may be comparatively late in the disease. The principal complaint will often be of a diffuse fibrositis which chiefly affects the upper part of the body. This syndrome, if it persists, should always arouse a suspicion of spondylitis in the case of a young male. In others pain may be referred directly from the affected spinal segment, and may simulate that of renal calculus, pleurisy or even of tabes. Sometimes neuritis or numbness and loss of power in the limbs, or severe spasm of the muscles of the back may be complained of. All these symptoms are usually intensified on forced movement of the spine,

and in addition pain is often elicited by firmly tapping the spinous processes involved.

There is also increasing stiffness in the back, and ultimately this may become completely rigid (" poker back "), generally in a position of kyphosis and slight forward flexion of the spine. In untreated cases this position becomes very exaggerated, the chin sinks into the chest wall, and the patient is unable to stand erect or move. Those who are not bedridden move with a characteristic slow, bent, shuffling gait. As the costo-vertebral joints ankylose, so does the respiratory expansion of the chest diminish.

For the results of radiological examination, see later.

Complications.—Those who die with this disease generally do so from an intercurrent respiratory infection, the result of the diminished or absent expansion permitted by the ankylosed costo-vertebral joints. The disease in itself usually does not shorten life.

Diagnosis.—All patients, especially young and athletic males, who complain of vague pains affecting the limbs and upper part of the body which do not respond to treatment should be suspect. The presence of detectable rigidity of the spine is unusual at this stage. If the blood sedimentation rate test is high this increases the suspicion and should lead to the patient being X-rayed. The radiological appearances at this stage are loss of definition (" wooliness ") of the sacro-iliac joints, osteoporosis of the neighbouring bones and lumbar vertebral bodies. Later, the sacro-iliac joints become obliterated, and the intervertebral joints, if they can be shown, are hazy and later on ankylosed. The edges of the intervertebral discs and the spinal ligaments are calcified. The vertebræ primarily affected are usually those of the lumbar and lower cervical portions of the spine. Osteophytes are not found. At this later stage the diagnosis becomes obvious as the whole spine is rigid, and the hip and shoulder joints are also sometimes ankylosed. Hysterical contracture of the spine should be differentiated.

Course and Prognosis.—In cases in which the onset is acute, ankylosis of the spine may occur within a few months. The younger the patient the more likely is this to happen. An associated swelling of other joints in the body develops at some period of the disease in about 25 per cent. of all cases, while the hips and shoulder joints are liable to be permanently involved in about the same proportion of cases. Iritis will occur in a small proportion, and is of bad prognosis.

Treatment.—It should be remembered that spondylitis of this type is a systemic disease and thus needs general treatment, as well as more specialised attention to the spine. Treatment will be more successful if it can be started at an early stage of the disease. The general treatment is similar to that advised for rheumatoid arthritis: rest, high caloric and vitamin diet. Vitamin C is particularly important in this malady. Any definite foci of infection should be cautiously removed and anæmia counteracted. Ultra-violet light is a useful general tonic, but gold salts do not appear to be useful in this condition.

The special treatment needed in the active stage includes rest in bed and mobilisation of the chest by means of breathing exercises. Fracture boards should be placed under the mattress, to prevent sagging, and the patient should be as flat as possible, the pillows being removed several times daily to hyperextend the spine. Breathing exercises done in this position

should aim at keeping the chest wall mobile by restricting abdominal breathing. Physical treatment is chiefly of value in the form of radiant heat or infra-red rays to the back, in order to relax spasm and relieve pain. Gentle massage is useful for the same purpose, and later on active movements should be performed under supervision. If there is already some deformity of the spine a plaster cast of the back should be made, and the patient should lie in this night and day to relax completely all spasm. The shell should be altered frequently so as to take advantage of the gradual postural improvement, and when the patient first assumes the upright position he should be fitted with a light spinal brace to relieve the back from all strain.

Recently X-ray treatment has been advocated in the treatment of this disease, but the results so far are variable.

Even with the greatest care it is sometimes impossible to avoid ankylosis of the spine supervening. But if it does occur it is almost always possible to ensure that it does so in the optimum position and thus the patient will ultimately be able to lead his life in an erect posture.

2. RHEUMATOID ARTHRITIC TYPE OF SPONDYLITIS (see p. 1352)

3. OSTEO-ARTHRITIC TYPE OF SPONDYLITIS

Synonyms.—Osteo-arthritis of Spine; Hypertrophic Spondylitis; Degenerative Spondylitis.

Some degree of osteo-arthritis of the spine is said to be demonstrable by means of the X-rays of nearly all those over 50 years of age; but it is rare for these changes to cause symptoms. Its incidence is rather higher in men than women, and it seldom occurs before middle life.

Pathology.—The changes are not inflammatory in nature and are identical with those of osteo-arthritis met with elsewhere in the body. Bony ankylosis does not occur in this type of arthritis, but osteophyte formation is always seen, together with narrowing of the intervertebral spaces. The areas most commonly involved are the cervical and lumbar regions.

Symptoms.—When symptoms are present they may include the following: root pains, of which sciatica is the most frequent example; increasing stiffness of the back, which, however, never becomes completely rigid. Headache and pains in the neck, shoulders, and arms are common, sometimes in conjunction with areas of paræsthesia or anæsthesia in the skin. Pains are generally aggravated by movement of the spine, as the nerve roots are liable to pressure in or around their exit from the spinal foramina.

Diagnosis.—This should be confirmed radiologically. The lesion typical of osteo-arthritis is the osteophyte; while marginal exostosis and shrinking of the vertebral margins, with narrowing of the intervertebral spaces, are also seen. The sacro-iliac joints are generally normal. In all cases the possibility of malignant growths in the spine should be borne in mind, as the symptomatology is the same.

Treatment.—This is similar to that of osteo-arthritis elsewhere in the body, and includes the correction of body posture and flat feet. The frequent application of heat in some form, followed by rest and muscular relaxation, is important. Massage and faradism at a later stage will tone up muscular support of the back. Sometimes it will be necessary to supplement the

support of the back mechanically by a plaster shell or a brace for a time. Deep X-ray treatment will sometimes relieve intractable pain which proves resistant to analgesic drugs, such as aspirin, pyramidon, or codein.

7. STILLS' DISEASE

This disease is believed by some observers to represent true rheumatoid arthritis in childhood. But although the articular changes are of an inflammatory nature their effects are more often confined to the periarticular tissues. Again, even in cases of long standing, it is not unusual to find little or no radiological evidence of destruction at the actual joint surfaces.

Ætiology.—The sexes are affected in about equal proportion, while the age incidence resembles that of rheumatic fever, namely, that the malady is seldom met with before the age of three years and the average age at onset is between six and seven years. The cause remains unknown, although the factors held to be responsible for rheumatoid arthritis are usually invoked to account also for Stills' disease. These are focal infection, such as in the teeth, tonsils, sinuses or bowel; metabolic or endocrine disorder; or, in some cases, unsuspected or attenuated infection with the bacillus tuberculosis. A hæmolytic streptococcus is commonly present in the nasopharynx and other foci. In the majority of cases it is very difficult to assign any one cause. It is certain, however, that once the disease has commenced a cold damp environment will exacerbate it considerably.

Symptoms.—If a careful inquiry of the history of the patient's illness be made it will very often be found that a considerable period of prodromal ill-health preceded the onset of the joint symptoms.

The onset of the joint condition is not infrequently rapid, with pyrexia, and pain and swelling of several joints. This often leads to an initial diagnosis of rheumatic fever, but it is soon found that sodium salicylate has no beneficial effect and that the joint swellings, far from being transient, increase in number and intensity. When the onset is gradual there is little pyrexia but a slowly progressive degree of swelling and limitation in movement of the knees, wrists, elbows, fingers and ankles—usually in that order. Later, the cervical spine and also the hips may become affected and the patient will be completely crippled. The foregoing joints are usually attacked symmetrically, and their appearance is characteristic in that the periarticular swelling renders the joint fusiform in shape. The skin over this swelling is rather stretched and often slightly bluish. The muscles adjacent to the affected joints waste, which further exaggerates the fusiform appearance. In the case of the wrists, ankles and elbows the bony contours are often completely obscured. The affected joints are generally tender on pressure, but usually are not painful except on movement. This leads to further voluntary limitation of movement and so intensifies the muscle wasting already present.

In many cases of this disease the joint swelling and muscular wasting is accompanied by a lymphatic reaction which will show as enlargement of the superficial lymphatic glands, especially those around the elbows and in the axillæ. In about half the cases seen there is also enlargement of the spleen. These changes were present in the cases originally described by Still

in 1897. The enlarged glands are not tender, and are generally discrete and "rubbery." Subcutaneous nodules are sometimes found in addition.

There is generally a secondary anæmia, and quite often patches of light-brown pigmentation on the skin. When the disease is established, the extremities are always cold and clammy, and there is an increase in the temperature before other joints are affected. In long-standing cases normal growth is considerably interfered with.

In the late stages the type of deformity seen in adult rheumatoid arthritis develops. There is flexion of the fingers and ulnar deviation of the hands, and also flexion of the knees and elbows.

The X-ray picture is chiefly remarkable for the advanced degree of the osteoporosis which always occurs. There is often little or no actual joint changes, and osteophytes are never found in this type of disease.

The end result, so far as the joints are concerned, is a fibrous ankylosis, or a fibrosis of the joint capsule, which is sufficiently complete to resist all attempts at movement of the joint.

It is stated that at post-mortem examination diffuse pericardial adhesions and adhesive mediastinitis are often discovered, although unsuspected during life, and evidence of valvular disease of the heart has occasionally been reported.

Prognosis.—Until recently there was considerable doubt as to what was the ultimate fate of these patients. Some authorities held that they recovered while others explained the rarity of the affection seen in Adult Out-patient Departments by assuming that the patients either died or became rapidly bedridden after leaving the children's departments. A recent "follow-up" at Great Ormond Street Hospital showed that the mortality in those under five years of age is about 25 per cent. (due to intercurrent infections); that complete recovery occurred in a small proportion; while in the majority, the disease remains apparently arrested, often for several years at a time, only to resume its ravages at increasing intervals until the patients are entirely crippled and bedridden.

Treatment.—When the presence of an infective focus is established, this should be dealt with at an early stage of the disease. In all cases the child's resistance should be built up by all available means. A nourishing diet, an open-air life and a dry sunny climate are indicated. In addition, cod-liver oil and malt, syrup of iodide of iron, and courses of an arsenic-containing tonic are important.

Vaccine treatment with autogenous very weak vaccine is worthy of trial if a hæmolytic streptococcus is found.

Salicylates have little or no beneficial effect. Non-specific protein therapy is often recommended, but is possibly too drastic and temporary a measure to employ except in the later stages. Recently good results have been reported from small doses of gold salts administered intramuscularly in short courses. But in some cases these salts provoke unfavourable reactions, and so should be used very cautiously. No case should receive a larger dose than 0.1 gm. and a total course of 0.75 gm. should rarely be exceeded. The injections should be given at weekly intervals. At least six weeks should elapse before any subsequent injections are administered, and the onset of toxic nephritis, stomatitis, diarrhoea and dermatitis should be especially looked for.

All swollen joints should be bandaged and lightly splinted, or put into thin plaster-of-paris casts, to avoid the contraction deformities which will otherwise inevitably occur. The child should always sleep in these, and will soon become accustomed to them. They should be removed daily, however, for a short period, during which the joint must be given passive movement, to prevent fixation. Dry heat from a radiant heat or infra-red ray lamp is comfortable and renders the performance of these daily active movements easier. These movements are also essential to remedy the muscular atrophy present. Massage is generally unnecessary in these cases.

In the very late stages, and the patient is bedridden owing to extensive contraction deformity, minor surgical procedures, such as tenotomy, are sometimes justifiable to remedy the deformity.

PSEUDO-ARTHRITIS (JOINT EFFUSIONS)

Effusion of fluid into the joints may be associated with various conditions, and is often of a temporary or intermittent nature. If not followed by damage to the joint surface, it should not be strictly referred to as an arthritis.

Apart from the various forms of arthritis already described, the following may give rise to joint effusion :

(i) *The specific fevers*, especially scarlet fever, meningococcal fever, puerperal fever, influenza, syphilis, subacute bacterial endocarditis, typhoid or paratyphoid fever, measles and malaria. In the United States a form of pseudo-arthritis associated with lympho-granuloma venereum is not uncommon.

(ii) *Abnormal blood conditions*, such as purpura simplex or purpura rheumatica (Schönlein's disease), scurvy and hæmophilia will give rise to swelling of the joints due to an effusion of blood. The knees are most frequently affected.

(iii) *Growths affecting the bones*, in near proximity to a joint. An X-ray examination will elucidate the ætiology in such cases.

(iv) *Injection of animal sera*.

(v) *Trauma* will cause synovitis of the affected joint. It should be noted that the strain imposed on certain joints due to faulty body posture will often result in a chronic form of hydrarthrosis.

(vi) *Intermittent Hydrarthrosis*. A periodic recurrence of joint effusion of unknown ætiology which persists for several days and usually affects the knees. Attacks tend to recur at regular intervals ; they show no local evidence of inflammation ; and they are refractory to most forms of treatment. Some authorities consider this condition is the result of an allergic sensitisation, and advocate treatment directed along these lines.

NON-ARTICULAR RHEUMATISM ; FIBROSITIS

Fibrositis may be defined as a condition in which acute or chronic inflammatory changes involve the fibrous tissues of the body, such as the subcutaneous tissues, the superficial and deep fascia, the muscle sheaths and tendons, the fibrous portions of the joint capsules and ligaments, the bursæ,

and the fibrous sheaths of the nerves. The affection gives rise to pain and impairment of movement. It may be subdivided broadly into three types according to the nature of the structures primarily attacked :

1. Panniculitis : Inflammation of the subcutaneous tissue and fat.
2. Inflammation of the muscle sheath and the fibrous tissue between the muscle fibres, the aponeuroses, the tendons and the superficial and deep fascia.
3. Peri-arthritis : Inflammation affecting principally the fibrous portions of the joint capsules, ligaments and bursæ (bursitis).
4. Peri-neuritis : Inflammation affecting primarily the nerve sheath (perineurium) and the fibrous tissue between the nerve fibres.

Ætiology.—A large group of cases can be traced to the presence of focal infection, and both the teeth and the tonsils are generally suspect. This is perhaps more particularly so in cases in which the upper part of the body, including the arms, are chiefly affected. In this connection it is to be remembered that negative X-ray evidence of apical infection of the teeth should not be accepted as conclusive evidence, since changes which can be demonstrated by this means are necessarily of a chronic and advanced nature. Further foci of infection should be sought in the sinuses. Occasionally the bowel, particularly the colon, may be thought of as a focus of infection, but when considering the question of vaccine treatment the old aphorism that it is “a poor bowel which does not grow something” may be remembered with advantage. Another focus of infection, which is sometimes overlooked, is the prostate, and in the absence of other infection it is worth while to perform prostatic massage and culture the “bead” so obtained.

A further group of cases would appear not to be of a primarily infective nature but to be allied to gout or to a special sensitivity to certain types of food.

Finally, a certain proportion of cases of fibrositis can be traced to chronic strain, often secondary to faulty posture. A common example of this will be found in those cases in which the fascia lata of the thighs is tender and painful in conjunction with a flattened plantar arch. Such patients are usually cured when the distribution of the body weight is readjusted by raising the inner edges of the shoes. Some cases of low backache seem also to be attributable to the same cause. Other causes of this chronic strain will arise out of the occupation, or sometimes the sports of the patient, while any unaccustomed muscular exercise should be inquired into, especially if in conjunction it has been associated with exposure to cold and wet, *e.g.* the frequent occurrence of sciatica in doctors who drive a car in wet clothes for a long period.

Morbid Anatomy.—The morbid anatomy of this affection was investigated originally by Stockman, who found that the results of injury to fibrous tissue, whether bacterial or traumatic, had the effect of producing in the acute stage an exudate and inflammatory œdema. This is followed by organisation of the exudate, and the growth of new fibroblasts and new blood vessels with thickened walls. There is no migration of polymorphonuclear leucocytes and no pus formation, a few lymphocytes alone being attracted to the inflamed area. As the condition becomes chronic there is a production of dense connective tissue in nodules or strands, which differ from normal fibrous tissue in having more fibroblasts, in having arteries

with thickened walls, and in leaving the sheaths of the nerves passing through it in a state of interstitial inflammation. The pain of fibrositis is apparently due to the swelling of the tissue through the inflamed exudate, and subsequently to the involvement of nerve fibres in the new fibrosis, apart from the interstitial inflammation of the nerve twigs themselves.

Symptoms.—It is to be noted that the pain complained of in fibrositis is not always at the real seat of the lesion but may be referred to other areas and therefore careful location of the actual seat of the inflammation is essential. Again, the symptoms will vary according to the area of the body affected. As examples, the predominant effect of involvement of the muscles of the neck will often be headache; of those of the limbs, numbness and tingling; and of the fibrous tissue surrounding joints, stiffness and pain on moving these, which is often wrongly attributed to true arthritis.

Panniculitis is met with most typically in the early stages of "menopausal arthritis" in which tender pads of thickened tissue are found over the internal aspects of the knees, the back of the neck, the extensor surfaces of the arms, the outer aspects of the thighs and elsewhere. It occurs most frequently in stout people, and Dercum's disease or adiposis dolorosa is a progression of this process. In some cases this syndrome appears to be associated with moderate hypothyroidism.

In the case of inflammation of the muscle sheaths and the intramuscular fibrous tissue, the local effect is to keep the muscles in a state of spasm during the acute period. Subsequently the spasm will relax, but localised patches of nodular induration may be palpable in the muscles. Occasionally, also, thin fibrous cords may be felt running through the subcutaneous tissues. These "nodules" will generally, but not invariably, prove tender on palpation. Lumbago is perhaps the most common manifestation. Its onset may often be very acute. It should be distinguished from arthritis or caries of the spine, sacro-iliac disease, perinephric abscess and renal disease, all of which may simulate it. Pleurodynia is a rheumatic inflammation of the intercostal muscles which gives rise to severe pain when the affected muscles are brought into action, as on coughing or deep breathing. Usually local tenderness can be elicited on palpation; but careful examination is needed to exclude such sources of pain as pleurisy or intercostal neuralgia. The muscles of the abdomen are sometimes the site of a local lesion, and this will occasionally simulate intra-abdominal disease. They are also affected in epidemic myalgia (Bornholm disease). The extensor muscles of the thighs, when they are the seat of rheumatic inflammation, give the clinical appearance of sciatica, and this possibility should always be considered before making a diagnosis.

One of the commonest causes of pain, generally diagnosed as brachial neuritis, is the presence of a degenerative lesion in the tendon of the supraspinatus muscle. This lesion will sometimes calcify and may then be seen in an X-ray, if the shoulder be externally rotated. This type of lesion is often the cause of inflammation of the subacromial bursa through which this tendon passes. "Tennis elbow" is the term applied to the painful fibrositis affecting the origin of the extensor tendons of the forearm from the external condyle of the humerus.

Bursitis may occur in any of the large bursæ. The most commonly affected is the subacromial bursa, referred to above. The chief clinical

manifestations of "deltoid bursitis" are great pain on actively abducting the affected arm to an angle of 90° with the body. Above this point abduction can generally be completed without pain; the pain returning, however, at the same point when the arm is again lowered. Passive movement through this range is not painful. Pain of this type will encourage the sufferer to immobilise the affected arm, and this will allow the inflammation to spread to the joint capsule, which will contract, and so ultimately limit, or even entirely prevent, movement taking place in the joint subsequently. The patient is often unaware of the full degree of limitation of the movement which has occurred in such cases, as a considerable degree of movement is possible by virtue of the mobility of the scapula. This condition is the severest type of peri-arthritis, and the apparent ankylosis of the joint must be differentiated from a true arthritis by means of radiology, as peri-arthritis of this type may be cured by the employment of diathermy, gentle manipulation, and remedial exercises. The next most commonly affected bursæ are those over the olecranon process, around the knee joint, over the ischium and over the great trochanter. Inflammation of any of these should be differentiated from a true arthritis of the neighbouring joint. Another form of peri-arthritis depends on inflammation of the tendon sheaths of muscles surrounding joints. This may occur as part of a chronic rheumatic process unassociated with trauma or gout. Its association with gonorrhœal and dysenteric infections has been mentioned. The flexor tendons of the wrists and knees are the most commonly attacked. Pain, swelling or crepitus results and sometimes synovial effusion. The palmar fascia is sometimes the site of a chronic fibrositic process, and the resulting thickening and contracture is known as Dupuytren's contracture. This is seldom painful, but can give rise to considerable disablement of a somewhat intractable nature. The condition, which is commoner in males, is often found to be familial. A somewhat similar condition, which, however, does not cause so much contracture, is known as "painful heel." In some cases a small spur of bone is found radiographically at the insertion of the plantar fascia into the os calcis. But in the majority of cases, no cause for the pain can be found.

In the case of peri-neuritis, the sciatic nerve is the most commonly affected, next in order are the nerves of the brachial plexus, and then the intercostal nerves. Some forms of Bell's "palsy" are thought to be of similar origin. The symptoms in a fully developed case do not differ from those of a true neuritis, but the distinction can generally be made from a history of an initial fibrositis of neighbouring structures which later spreads to the nerve sheath. Since the introduction of radium therapy for carcinoma of the lung, cases of axillary fibrosis, followed by severe and sometimes permanent brachial neuritis, have been seen.

The subjects of fibrositis are usually found to have some degree of defective skin circulation, as evidenced by abnormal sensitivity to cold weather or to local draughts, spontaneous bruising, or the fact that they perspire in the hottest weather only with difficulty. Attacks of fibrositis will occur in many people without apparent detriment to their general health, and in such persons the suspicion should arise that the cause may not be primarily an infective one.

The group of cases which appear to be allied to gout or to a special sensitivity to certain types of food may be suspected by the excellent general

health, even during attacks, the periodic or seasonal nature of the attacks, a history of familial gout or of being "unable to digest" certain foods or drinks, and finally by the fact that the fibrositis tends to affect the lower limbs and other lower parts of the body. Such patients, in addition, often exhibit the symptom-complex described by the French as "hépatisme." This is shown principally by morning headache, furred tongue, and a tendency to incomplete bowel emptying, with light-coloured and offensive stools; often, too, there is slight tenderness on palpation in the neighbourhood of the liver.

Prognosis.—Provided sufficient care be taken and the value of external as well as internal remedies is remembered, the outlook is good. An exception, however, is in the case of very old patients, for the senile form of fibrositis is sometimes intractable to all the usual remedies.

Treatment.—When symptoms do not call for urgent treatment, the first indication is to investigate the ætiology. In the great majority of cases in which the malady is believed to be allied to gout or to a special sensitivity to certain types of food, appropriate treatment should be adopted.

At the outset, a mercurial purgative, such as calomel (gr. $\frac{1}{2}$ –2), followed next morning by a saline, should be prescribed.

In the acute stage rest in bed is desirable. The internal administration of analgesic drugs is indicated. Aspirin and calcium acetylsalicylate (5–15 grs.) are in the majority of cases the most efficient for this purpose. If necessary, potassium iodide (2–5 grs.) may be added, as may phenacetin (5–10 grs.) or caffeine (5 grs.) at four-hourly intervals. A useful addition to aspirin and phenacetin is codeine phosphate ($\frac{1}{4}$ th gr.) or Dover's powder (5–15 grs.). Amidopyrine (3–10 grs.), a centrally acting drug, is sometimes effective when the salicylates fail. It should be borne in mind, however, that if this drug is used there is a danger of agranulocytosis in susceptible individuals and therefore frequent blood counts are required. An ointment designed to act either as a rubefacient or as a counter-irritant should also be prescribed. A hot linseed poultice containing opium will also often give considerable relief if applied every few hours; as may a hot cloth wrung out in a solution of ordinary mustard in water. Massage is undesirable in the acute stage.

In certain cases benefit will result from a short course of colonic lavage twice a week for 2 to 3 weeks.

When it is desired to immobilise the muscles of the back during the acute stage of lumbago, the most effective method is by means of a perforated belladonna plaster, which should be made to cross the mid-line behind and come round to the front. Ordinary wide strapping is a good substitute.

When the condition is less acute and the patient is able to get up, the application of both heat and massage to the affected regions is indicated. The former may be applied in a dry or a moist form, the one often succeeding when the other has failed. Dry heat may be given by means of a portable lamp, an electric heating pad, exposure to a gas-fire (which gives out infra-red rays), the application of a hot iron through brown paper applied to the skin, a hot-water bottle, or a bag of salt or sand which has been heated thoroughly in the oven. When the condition is deep-seated, diathermy will be the best form in which to apply it.

Moist heat may be applied in the form of kaolin, bread or linseed poultices, kaolin poultice (antiphlogistine), mud packs, or applications of hot paraffin wax of a special melting-point (which is sold for this purpose), or, if the patient is in a condition to have such, a Turkish bath. In the course of the last, the patient should be instructed to drink fluids copiously during and after it, as otherwise the temporary concentration of the blood is likely to provoke a further acute attack. Perhaps the simplest method of moist heat is an ordinary bath, to which 4 lb. of Epsom salts (or common salt) have been added. This should be taken as hot as possible, and, contrary to the general belief, the patient should not "soak in it" but should get out after only 5 to 10 minutes' immersion and be briskly rubbed down, after which some analgesic ointment should be rubbed rapidly into the affected areas, and he should be wrapped in a blanket and put to bed for several hours. After this, deep massage should be ordered for the affected areas, although painful at first.

Hydrotherapy and Counter-irritation.—When the patient is near a Spa or an Institution equipped for hydrotherapy, "Vichy" douche massage, followed by "contrast douching" (alternate hot and cold water directed on to the painful areas under pressure from a hose-pipe), is probably the best follow-up treatment; it stimulates the skin to resume its normal function.

In certain cases, particularly when the complaint is that of lumbago, counter-irritation by means of a small cautery may be of great value. A small blister should result from each application, and the whole area may then be covered over with a gauze dressing. Another method is to produce blisters by means of "blistering fluid," or cantharidin plasters, but these are not of such value as the actual cautery. Dry-cupping is a somewhat obsolete method of treatment, but is occasionally effective; as is full exposure to a mercury vapour lamp at a distance of 18 to 24 inches from the affected area.

In the chronic stage massage is essential if an attack is to be terminated in the minimum time and also if recurrence is to be avoided. To be effective, the indurated areas (nodules) should be carefully sought for in the muscles, and at these points the massage should be very deep. It will be found that following deep kneading with the finger-tips or thumbs, after an initial period during which they may be increasingly painful, they will gradually become insensible to palpation, and ultimately disappear. This process will be considerably facilitated if it be preceded on each occasion by 20 to 30 minutes' application of heat in one of the forms mentioned above. In certain cases a "nodule" will prove to be too painful for deep massage treatment, which will then induce protective spasm in the surrounding muscles, and so render further "kneading" impossible. In such cases a dose of aspirin or some other analgesic may usefully be administered before beginning the treatment. When one or more discrete nodules can be felt and when the pain is found to be chiefly localised in these sites, the effect of injecting a few cubic centimetres of a local anæsthetic, such as procaine hydrochloride ($\frac{1}{2}$ per cent. in saline) or "A.B.A." compound, is sometimes dramatic. When there is more diffuse pain and tenderness, this method of treatment is not of much use, and unless the injection is made with great accuracy into the nodule the trouble may even be exacerbated.

Diet and After-care.—If there is obesity, this should be treated (see

p. 438). In cases in which a gouty origin is suspected, this should be corrected (see pp. 435, 436).

After an attack of fibrositis, it is important that the patient should be taught to contract the affected muscles daily by means of appropriate exercises. He should also make a point of obtaining some regular exercise in the open air, even at the cost of rising somewhat earlier in order to walk part of the way to the office. The obese subject must not be allowed to regain his lost weight once the attack recedes. The question of the localisation of attacks, as the result of the occupation or hobbies of those predisposed to suffer, should not be omitted.

MYOSITIS OR INFLAMMATION OF THE VOLUNTARY MUSCLES

Three forms occur—(1) the suppurative type; (2) the non-suppurative type; and (3) myositis ossificans progressiva.

1. SUPPURATIVE MYOSITIS.—In this condition there is a primary inflammation of the affected muscles associated with the local signs of inflammation and the general symptoms of a septic infection. Abscesses form in the affected muscles, which require incision, and in the pus obtained pyogenic organisms, such as staphylococci, or less commonly streptococci, are usually found.

2. NON-SUPPURATIVE MYOSITIS.—It must be remembered that the voluntary muscles are affected in the course of other diseases. Thus, degeneration of the striped muscle, known as Zenker's degeneration, may occur in any acute infection of long duration, and it was first observed in typhoid fever. In scurvy, intra-muscular hæmorrhages are very common, and these are followed by a chronic inflammation, which usually clears up; but in a few of such cases we have seen suppuration occur. Trichinosis is accompanied by a myositis, set up by the encapsulated larvæ of the trichina spiralis deposited in the voluntary muscles.

Dermato-myositis is an acute or subacute inflammation of the muscles of unknown origin, which is associated with dermatitis and œdema. The onset is usually gradual, and ultimately all the muscles of the body may be involved. Pain is an early symptom, and fever of a mild intermittent type occurs. Œdema develops over the affected muscles, and is accompanied by a dermatitis of erythematous or urticarial type. Sweating is common, and enlargement of the spleen usually develops. Owing to involvement of the respiratory muscles broncho-pneumonia is a late complication. The disease is usually progressive, and generally fatal, though some recoveries have been recorded. The treatment adopted has been for the relief of symptoms, and no specific treatment is known at present.

A type of the disease in which hæmorrhages occur in and between the muscles is known as "polymyositis hæmorrhagica."

3. MYOSITIS OSSIFICANS PROGRESSIVA.—This is a progressive inflammatory affection of the locomotor system of unknown origin, characterised by the deposition of bony substance in the fasciæ, muscles, aponeuroses, tendons, ligaments and bones, with resulting ankylosis of most of the articulations. The disease is rare. It usually commences in early life, and is commoner in males. Three stages occur in the muscle changes. In the first stage, swelling

and infiltration of the affected muscle with embryonic connective tissue occurs. In the second stage, the embryonic connective tissue becomes organised and forms ordinary connective tissue, which retracts to a hard fibrous mass. In the third stage, calcification of the fibrous mass occurs, and this becomes replaced by bone.

The muscles of the back and neck are usually the first involved, and the vertebral ligaments become ossified, so that irregular bony swelling occurs and deformity and fixation of the spine result. The upper and lower limb are later involved, the muscles contracting and causing fixation of the joints. The muscles of mastication become finally involved and prevent movement of the lower jaw. Ultimately the patient becomes helpless and bedridden, and usually dies from some intercurrent affection, such as pneumonia, or pyæmia resulting from bedsores. The disease is always progressive, but is usually of long duration, and there may be a cessation in its progress for several years. No specific treatment of value is known.

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SECTION XVIII

DISEASES OF THE SKELETON

Diseases of the skeleton will be considered under three headings, according to whether bone, endochondral ossification, or bone marrow is primarily affected.

DISEASES OF BONE

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Synonyms.—Hippocratic Fingers ; Marie's Disease ; Acropachy.

Definition.—A symmetrical enlargement of the bones of the hands and feet, and of the distal ends of the long bones, accompanied by clubbing of the fingers and toes, occurring in association with certain chronic diseases, especially of the lungs.

Ætiology.—The primary diseases in the course of which hypertrophic osteo-arthritis may develop are :

1. Diseases of the lungs, such as (a) chronic cavitating tuberculosis and fibroid phthisis ; (b) empyema and bronchiectasis ; (c) malignant disease of the lung, pleura or mediastinum ; and (d) fibrosis of the lung, non-tuberculous in nature.

2. Congenital heart disease, and infective endocarditis.

3. Chronic diseases such as dysentery, pyelonephritis, alcoholism, Raynaud's disease, and jaundice, as in hypertrophic cirrhosis of the liver.

4. Rarely a neuritis may lead to clubbing of the fingers and osteo-arthritis. Pressure on the brachial plexus by a subclavian aneurysm has given rise to clubbing of the fingers on the affected side.

It appears that in the majority of cases a chronic infection leads to the development of osteo-arthritis. This is the case in the lung diseases above mentioned. In congenital heart disease, the circulatory defect itself will lead to marked clubbing of the fingers ; in this condition, however, the long bones are not appreciably affected, and the changes are limited to the soft parts of the terminal phalanges, the hypertrophy of which leads to clubbing.

The disease is eight times more common in males than females. All ages may be affected. The most striking examples are seen from 30 to 50 ; but in congenital heart disease and in chronic lung disease in children, such as bronchiectasis and fibroid phthisis, signs of the disease often appear in early life.

Pathology.—The bones most frequently affected are the metacarpal bones and the first two rows of phalanges. The radius and ulna may be

affected, and more rarely the lower end of the humerus and the scapula. In the lower extremities the corresponding bones are affected. X-ray examination shows a thin layer of newly formed bone spread over the shaft. The periosteum is raised unevenly, so that the outline appears serrated and the deposits beneath it are unevenly calcified giving a lace-work effect. The bony changes are the result of a chronic inflammation, and the thickening of the periosteum and new formation of bone beneath it may be accompanied by atrophy and rarefaction of the pre-existing bone. There are no bony changes in the terminal phalanges, the soft tissues and nails alone being affected.

Symptoms.—The onset is usually gradual, and little local pain is experienced, though stiffness and clumsiness of movements occur. Sometimes marked clubbing of the fingers develops in a few weeks; but usually several months or more elapse before the condition is characteristic. There is a remarkable symmetry in the pathological changes. The ends of the fingers and toes may be cyanosed. The nails are large, broad and curved, both longitudinally and transversely—the so-called parrot-beak. They show longitudinal striation and are brittle and easily split. The root of the nail is raised above its bed, and if pressure is applied at the root a distinct space between them can be made out. Sometimes the joints in the neighbourhood of the affected bones show swelling, from effusion and thickening of the synovial membrane. Osteo-arthritis changes in the joints are only present in the severe osteo-arthritis type.

Three types of cases are seen, but these may be only grades in the development of the extreme form of the disease.

1. *Cases showing only clubbing of the fingers, in addition to the signs of the general primary disease.*—This symptom may disappear if the primary disease is cured, as, for example, empyema.

2. *Cases showing clubbing of the fingers and painful thickening of the bones of the hands and feet, forearms and legs, in addition to symptoms of the primary disease.*

3. *The "osteoarthritis hypertrophica" type.*—The hands and feet become greatly enlarged, owing to the bony changes and thickening of the soft parts. The forearms and legs are thickened. The pelvis, sternum, ribs and clavicles may be thickened, and the vertebræ may show changes resulting in kyphosis. Osteo-arthritis occurs in the parts involved, so that movement of the joints is painful and difficult. In this type of case the very remarkable changes in the bones and joints overshadow the symptoms of the primary disease.

Diagnosis.—The disease is recognised by the presence of the characteristic changes in the extremities, and by the presence of signs of one of the primary diseases already mentioned.

Infective arthritis is distinguished by the absence of clubbing of the fingers, and by the characteristic changes shown by X-ray examination.

Acromegaly is to be distinguished by the spade-like hand, the spatulate fingers, enlarged knuckles, and the characteristic facial appearance. The kyphosis is more often cervico-dorsal, whereas in hypertrophic pulmonary osteo-arthropathy it is more often dorso-lumbar.

Osteitis deformans shows irregular enlargement of the bones but there is a good deal of bowing, the hands are normal, and the X-ray appearances are pathognomonic.

Prognosis.—The prognosis appears to depend on the primary disease. If that can be arrested or cured, there is hope of arrest or improvement in the hypertrophic osteo-arthritis.

Treatment.—This should be directed towards the cure or improvement of the primary disease. Other treatment is symptomatic and similar to that adopted in the treatment of infective arthritis.

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OSTEITIS DEFORMANS

Synonym.—Paget's Disease of Bone.

Definition.—A chronic and somewhat rare disorder causing enlargement and deformity of many bones. It is not a generalised disease of the skeleton. The bones are affected in the following order of frequency: pelvis, spine, femur, tibia, skull, fibula, clavicle, humerus, radius, and rib. In a few cases the disease is confined to one bone or to part of one bone: tibia, femur, clavicle, a vertebra, the ilium, or half the pelvis.

Ætiology.—This is unknown. The disease is sometimes familial. It rarely begins before the age of 40, and the commonest age of onset is 55. The sexes are affected in the proportion of three men to two women. Osteitis deformans is not inflammatory in origin. It seems likely that it is a disorder of mineral metabolism. Syphilis is not an ætiological factor. No alteration in the parathyroid glands nor in any other endocrine gland has been demonstrated. Both histological and chemical investigations have proved beyond doubt that generalised osteitis fibrosa (hyperparathyroidism) is unrelated to osteitis deformans.

Pathology.—There is a great alteration in the architecture of the bones affected. They become enlarged, irregularly thickened, and sometimes bowed. The skull is very thick, the sutures and foramina being narrowed in consequence. The cortex of the long bones ceases to be pure ivory bone but looks coarse and spongy with red streaks and dots. Histologically there is continuous excessive resorption of bone associated with an increased new bone formation that more than compensates for the bone lost. The excessive erosion disturbs the skeletal architecture, the compact bone being replaced by irregular angular trabeculae, which also form the cancellous bone. There is still an attempt at structural adaptation to stresses, but this is very imperfectly achieved because the material is not used to the best mechanical advantage.

Biochemistry.—The serum calcium and plasma phosphorus are normal. The plasma phosphatase is constantly high, as in many other diseases of bone. In more than 80 per cent. of cases the calcium output in the urine is increased and sometimes reaches four or five times the normal figure. There seems to be a complete absence of correlation between the length of history, the density of bone shadows in radiographs, and the calcium output. A case showing increased density of bone trabeculae throughout pelvis, lumbar spine, and femora is just as likely to reveal a high output of calcium in the urine as a low output.

Symptoms.—The disease may remain symptomless for ten years or more. It is very slow in progress and rarely influences the general health, giving rise in most cases to few symptoms other than those which are due

to changes in the shape of the bones. In 80 per cent. of cases there is pain, and the patient usually recognises its origin in the bones. It varies widely in severity from a dull ache to a severe shooting or stabbing like a knife. The back and lower limbs are the parts usually affected but headache is fairly common. When the skull is involved the patient may have to take a larger size in hats. The enlargement in the circumference of the head leads to the forehead being prominent and the face small in proportion. In the later stages the head is held forward and the back is so bent that the arms appear too long and an ape-like attitude results. There may be considerable reduction in total height. The lower limbs especially are bowed, the knees being widely separated and held slightly flexed. The bones are enlarged, and bowing usually takes place in such a manner as to accentuate the normal curve of the bone. The enlargement is particularly noticeable in the case of the tibia. The changes in the vertebrae may cause encroachment on the spinal canal, resulting in compression paraplegia. Bony compression of the optic nerve may lead to optic atrophy, and of the oculomotor nerves to diplopia. Otosclerotic deafness is common in advanced cases. Spontaneous fracture is rare but when it takes place there is no delay in union. Osteogenic sarcoma may occur, but is much less common than Paget thought and is not seen until the changes in the bones have been present for ten years or more. Osteo-arthritis of the hip, knee, ankle, or spine is an occasional complication. Arterial degeneration, sometimes with hypertension, is found in most cases over the age of 50. It is possible that the excess of phosphoric esterase in the blood accelerates and intensifies the deposition of calcium salts in degenerate vessels. Retinal arteriosclerosis is a frequent finding, and it may be associated both with retinal hæmorrhages and extensive choroidal changes.

Radiological appearances.—The altered bone appears in radiographs in two forms, which may be called the spongy and the amorphous, the former being the more common. The two types are often found in the same patient. The spongy form consists of coarse irregular striæ arranged either as parallel trabeculae or running in the direction of normal lamellæ of cancellous bone. The amorphous form is a generalised deposit producing an opaque finely granular appearance. The diameter of the bone is increased, sometimes to a marked degree, and in the medullary cavity the trabeculae are accentuated and too widely separated, giving a streaky appearance. The corticalis is partly or entirely replaced by bone similar to that seen in the medullary cavity, and in an extreme case the impression is that the whole bone consists of cancellous tissue highly magnified. Irregular cyst-like areas are sometimes observed. Widening and bowing of bones are important points in the radiological diagnosis. The vault of the skull is thickened, and the differentiation between the inner and outer tables is lost. Small islands of dense bone are evident alongside pale, cyst-like areas. A large clean cut area called *osteoporosis circumscripta* may sometimes be noted. In those cases in which part of one bone is affected there is a definite line of demarcation where the abnormal ends and the normal begins. Thus there may be definite changes in the upper two thirds of the tibia, while the lower third is normal. The average rate of progress of such a lesion is about 1 cm. in two years. Radiographs reveal the shadows of arterial calcification in more than 40 per cent. of cases. Such calcified arteries are best seen in the lower limbs. There

is no evidence of a higher incidence of renal or vesical calculus in osteitis deformans than in the normal.

Diagnosis.—When advanced the condition is unmistakable. In the early stages muscular rheumatism or osteo-arthritis may be wrongly diagnosed. Pulmonary osteo-arthropathy is distinguished by the clubbed fingers. In radiographs the amorphous type of osteitis deformans is sometimes mistaken for secondary carcinomatosis of the osteoplastic type. The difference is distinct and important, namely, that in carcinomatosis the bones are neither enlarged nor bowed. Syphilis of bones is now very rare, but when only one or two bones are involved in a supposed case of Paget's disease the Wassermann reaction should be performed.

Prognosis.—Because the disease is uncommon there is a tendency to regard its effects as dreadful. To announce the diagnosis as though it were a profound mystery may alarm both patient and relatives unnecessarily. Paget's disease is slowly progressive but does not usually shorten life. Thus, one patient though much deformed, continued to drive a crane in a dock-yard 15 years after the onset of the disease. Another was quite happy to have somebody hold him on a rock while he fished a stream, long after he was unable to walk unaided. Death usually results from the effects of arteriosclerosis or intercurrent infection, and only rarely from compression paraplegia or sarcoma of bone.

Treatment.—No known treatment alters the course of osteitis deformans in the slightest degree. Since the bones at one stage are sufficiently decalcified to bend, methods have been used which aim at increasing the calcium intake. The patient is given a high calcium diet, that is a diet containing three pints of milk or milk products daily, together with butter, cheese, and eggs. If milk is not tolerated in these quantities calcium caseinate or calcium lactate (10 grammes a day) may be prescribed. Vitamin D may be conveniently given in the form of tab. calciferol. (3000 units) one or two daily. The claim that prolonged exposure to general ultra-violet irradiation has resulted in increased density of the shadows of bones in radiographs has not been confirmed. Such treatment can be carried out, starting with short exposures to the mercury vapour or carbon arc lamp. Paget treated his patients with potassium iodide, but was not enthusiastic over the results. When there is pain in the bones Lugol's solution (of iodine in potassium iodide) may be given in milk, beginning with a dose of three minims three times a day, and increasing to ten times this amount. If iodine fails to relieve the pain, aspirin, amidopyrine, or allonal should be tried. Exploration of the neck for a parathyroid tumour is never justified. Osteotomy is rarely necessary, but it is interesting that when portions of bone have been removed for histological section relief of pain has sometimes occurred. Occasionally, and especially in those cases with secondary osteo-arthritis of the hip joint or knee joint, an ambulatory splint supporting the weight of the body on the tuber ischii is of value. A cork sole is often necessary, and when kyphosis causes pain a spinal jacket is useful.

LEONTIASIS OSSEA

Synonyms.—General Hyperostosis of the Skull; Cranio-sclerosis; Megaloccephaly.

Definition.—The term *leontiasis ossea* is now used in two senses, specifically for a progressive sclerosing hyperostosis of the skull, and symptomatically when osteitis deformans and the various types of osteitis fibrosa happen to involve the bones of the calvaria and face.

Ætiology.—This is unknown. The fact that the disease commonly arises in the region of the nasal sinuses has led to an erroneous view that it is infective in origin.

Pathology.—When Virchow suggested the use of the term "*leontiasis ossea*" in cases of hyperostosis of the skull he had in mind fibroma molluscum in which masses of new connective tissue develop in the skin. He believed that the overgrowth of bone in hyperostosis corresponded exactly to elephantiasis of the soft parts, and he decided to call these cases *leontiasis ossea*, not because the bone disease produced a leonine appearance, but because he considered it to be analogous to the disease of the soft parts which did. The disease is very rare. It occurs in either sex, arising usually in early adult life. In most instances it begins in the nasal fossæ and sinuses, though in some cases the origin is near the orbit or in the base of the skull. Dense ivory bone appears and spreads slowly under the periosteum, being held up sometimes in the region of the suture lines but ultimately breaking through and spreading in many directions across the skull. The serum calcium and plasma phosphorus are normal.

Symptoms.—The early clinical features include nasal obstruction, blocking of the lachrymal ducts, and alteration in the shape of the face and jaws. Ultimately large masses of bone, increasing in various directions, give rise to terrible disfigurement. The cavities of the mouth, nose, and orbit may be greatly lessened. The eyeballs may protrude even beyond the lids, and blindness may occur from optic atrophy. There may be loss of the sense of smell, and interference with the mobility of the lower jaw. Except in the later stages pain is unusual.

Diagnosis.—Paget's disease usually begins at 55, and the pelvis, spine, and lower limbs are nearly always affected. Generalised osteitis fibrosa leads to decalcification of the whole skeleton, with a high blood calcium and low blood phosphorus. Focal osteitis fibrosa often shows multiple lesions scattered throughout the skeleton.

Treatment.—No treatment has any permanent effect though it may be possible to remove some of the more disfiguring masses of bone.

HYPERPARATHYROIDISM (GENERALISED OSTEITIS FIBROSA CYSTICA (see p. 498)

FOCAL OSTEITIS FIBROSA

Synonyms.—Osteitis Fibrosa Circumscripta (Schmidt); Local Fibrocystic Disease; Benign Giant-celled Tumour; Osteoclastoma; Osteogenetic Myeloma; Myeloid Sarcoma.

Definition.—A focal or multifocal disease of bone unassociated with constitutional symptoms or with any known endocrine disturbance.

Ætiology.—This is unknown. The disease occurs chiefly in adolescence,

and is much more common than is generalised osteitis fibrosa (hyperparathyroidism).

Pathology.—The lesions are benign, firm, grey or brown tumours. Histologically they show osteogenetic fibrous tissue and giant cells which, of course, are osteoclasts. This explains the numerous synonyms which are used. The tumours sometimes expand the corticalis and may give rise to cysts lined by osteoclasts. Even when the lesions are multiple the rest of the skeleton consists of normal bone. The figures for serum calcium and plasma phosphorus are invariably normal, a finding in striking contrast to that of the generalised disease. The calcium balance is usually normal, and, taken in conjunction with the normal blood chemistry, this finding is strong evidence against hyperparathyroidism.

Symptoms.—The malady affects one or more bones, is usually not disabling, is of slow progress, and shows a tendency to become arrested. Pain is unusual and the disease is often symptomless until spontaneous fracture occurs. Severe cases of the multifocal type may show considerable deformity, especially of the pelvis, femora and skull.

Radiological appearances.—In radiographs the principal changes are found in the ends of the long bones. Usually more than one-third of the shaft is affected by a fusiform enlargement composed of a pale cyst-like area divided by a few coarse trabecular strands. The cortex is thin and may be expanded. The periosteum and adjacent bone are normal. Radiographs taken with controls show that the whole skeleton apart from the lesions is normally calcified. The floor of the skull and the lower jaw may be affected.

Diagnosis.—The normal blood chemistry serves to distinguish the focal from the generalised disease. In adult cases it is sometimes difficult to differentiate between focal osteitis fibrosa and osteitis deformans, and it may then be necessary to follow the progress of the condition over a period of time before a definite conclusion is reached.

Treatment.—Fractures are treated in the usual way. If spontaneous fracture occurs in a long bone through one of the lesions, union is usually strong, and radiographs subsequently show that the pale cyst-like area of osteitis fibrosa becomes filled with bone. Exploration of the neck for a parathyroid tumour is quite unjustified.

THYROTOXIC OSTEOPOROSIS (see Hyperthyroidism, p. 488)

OSTEOMALACIA

Synonym.—Mollities Ossium ; Adult Rickets.

Definition.—A generalised disease of the skeleton due to vitamin D deficiency. Two types are found. The first is due to a diet deficient in vitamin D and calcium salts and may be referred to as dietetic osteomalacia. The second is a conditioned dietary deficiency disease, arising from deficient absorption of vitamin D and calcium salts ; it is seen in idiopathic steatorrhœa.

Ætiology.—Osteomalacia is rare in England. It is endemic over wide areas in Northern India, Japan and Northern China, and occurs sporadically in the Rhine Valley, Danube Valley, Vienna, and certain parts of Italy, Switzerland, Flanders and the Balkans. Heredity plays no part. The

disease pre-eminently affects women, and is likely to recur earlier and with greater severity with each successive pregnancy. But it is a mistake to suppose that pregnancy is essential in the aetiology. The malady is sometimes seen at puberty and is quite well known to occur, though rarely, in boys and men. In the majority of cases the symptoms begin between the twentieth and thirtieth year.

Pathology.—Rickets and osteomalacia are essentially identical. What difference exists is merely that of age incidence. Osteomalacia is adult rickets. Morbid anatomists agree that in rickets and osteomalacia the essential abnormality is a deficient calcification of osteoid tissue. This deficiency is generalised throughout the skeleton. The broad osteoid seams in both diseases are due to deficiency of the calcifying mechanism, which should convert osteoid tissue into true bone. In osteomalacia the bones throughout the skeleton are so soft that they readily bend and cut with a knife like rotten wood. Spontaneous fractures are common. The blood chemistry is comparable in experimental rickets of rats, in children with rickets, and in women with osteomalacia. The plasma phosphorus and sometimes also the serum calcium are diminished. The occurrence of foetal rickets has been proved in babies born of osteomalacic mothers.

Symptoms.—Pain is a prominent symptom. It occurs especially in the back and thighs, is aching in character and is worse in the winter months. The pelvis, thorax, or long bones exhibit deformity in a haphazard way; one woman suffers in the pelvis, another in the ribs, and a third in both. Besides the changes in the pelvis, marked deformities occur in the chest and spine. Severe kypho-scoliosis may reduce the height by several inches and cause the head and neck to sink downwards and forwards on to the chest. Deformities of the sternum and ribs give rise to marked prominences and depressions in the chest wall. Coxa vara and irregularly curved long bones are less common. The bones are soft and flexible, rather than fragile, so that bending is much more common than is spontaneous fracture, though both are well recognised. The patient develops a characteristic waddling gait, and muscular weakness may add to her incapacity. In many cases the pelvic deformities interfere with marital relations or with labour, Cæsarean section frequently being necessary. Tetany is common. The teeth are normal. The course of the disease may be fairly rapid, lasting several months, but untreated cases extend over many years. The patient then becomes bedridden, spontaneous fractures, anæmia, cachexia, and bedsores adding to her discomfort and to the difficulties of nursing.

Radiological appearances.—The degree of lack of calcification in radiographs will vary with the severity of the disease, and it is therefore important to take radiographs with controls. In the slight cases the bones of the patient will be slightly more translucent than those of the control. The cortex will be less dense than normal but the bone pattern, especially the trabeculation, will be accentuated by contrast. In the severe examples there will be little or no difference between the density of the bone and surrounding soft tissues, and the cortex will appear as a mere pencilled outline. The bone pattern will have disappeared, the long bones will bend, and occasionally show fracture. All deformities apart from fracture are the result of weight stress or muscular action. The pelvis is tri-foolate, owing to the thrusts of the heads of the femora and sacrum. Lordosis is marked and kyphosis may

be present. In severe cases the chest and ribs are usually deformed. The vertebræ are biconcave, having the appearance of fish vertebræ. In severe cases the vault of the skull may show numerous areas of uneven translucence, varying in size and shape but all fairly clean cut. The spontaneous fractures are usually subperiosteal, and radiographs sometimes show pseudo-fractures. These appear as areas of complete translucence, running across the bone, the edges being quite clean cut, and separated from each other by one or two millimetres.

Diagnosis.—The occurrence of pregnancy and the examination consequent upon this lead commonly to the recognition of the pelvic deformity and of the disease which has given rise to it. Differential diagnosis from other generalised diseases of the skeleton usually produces no difficulty. In hyperparathyroidism there is a high serum calcium, a low plasma phosphorus, and an increased calcium excretion in the urine. In senile osteoporosis the patient suffers from kyphosis and a tendency to fractures particularly of the neck of the femur, and the blood chemistry is normal. In thyrotoxic osteoporosis, the usual signs of hyperthyroidism are present, and the blood chemistry is normal. In myelomatosis the Bence Jones protein is found in the urine in 75 per cent. of cases, the serum globulin is usually increased, and the albumin:globulin ratio diminished. The serum calcium is usually normal, but sometimes raised. The plasma phosphorus is normal, but it rises in cases showing renal insufficiency. In radiographs the condition may closely resemble osteomalacia.

Treatment.—Pure vitamin D is called calciferol because of its power to induce calcification in tissues, especially in osteoid tissue. It is 300,000 times as potent as cod-liver oil, weight for weight. The good effects not only of calciferol but also of cod-liver oil and ultra-violet irradiation have been noted both clinically and chemically, since they are capable of raising the serum calcium to normal. In cases where tetany is present calcium salts should be administered in addition. The diet of a woman suffering from osteomalacia should contain 3 pints of milk a day, with plenty of milk puddings, eggs, butter, cheese, green vegetables, and even nuts and raisins. The dose of cod-liver oil should be large, up to 2 or 4 oz. daily. This treatment relieves the pain in 3 to 4 weeks. Some cases are refractory, and it is then necessary to add 0.5 mg. of calciferol to the cod-liver oil daily. Tetany is rapidly removed by treatment with cod-liver oil and calcium lactate. A powder containing at least 10 grammes of the latter should be used daily, and is best administered fasting with a glass of milk. The patient should be exposed to sunlight when this is possible; otherwise treatment by ultra-violet irradiation may be used, starting with a short exposure to a carbon arc lamp and increasing gradually up to 30 minutes. There is no evidence that phosphorus is of any value in the treatment of osteomalacia. Where the disease exists in great endemic areas, questions of diet, and social and religious customs are proving very difficult. In large areas of China and India the diet is often deficient in quantity, and inadequate in calcium and vitamin D. In the high mountain valleys of these countries and in areas of India where purdah is practised, darkness adds to the danger by causing further deprivation of vitamin D. With regard to China, Maxwell states: "We want flocks and herds, milk and meat, with security of life and property." The suggestion has been made that it might be practicable

in India and China to dispense calciferol freely at a low price just as quinine is dispensed in malarial districts. The relation of ovarian function to calcium metabolism has not yet been settled. Osteomalacia gets worse during lactation, no doubt because of the great drain of calcium from the body. Improvement has been observed after ovariectomy. This operation may act merely by preventing pregnancy, and it is presumably just as reasonable to ligate the Fallopian tubes. When pelvic deformity demands it Cæsarean section is necessary.

OSTEOMALACIA IN IDIOPATHIC STEATORRHOEA

When osteomalacia occurs in the course of idiopathic steatorrhœa (Gee's disease), the following features may be present: fatty stools, dilatation of the colon, tetany, anæmia, skin lesions, and infantilism (see p. 476). The disease occurs in both sexes and the history nearly always goes back to early childhood. The symptoms develop in spite of an adequate diet. We must, therefore, suppose that there is some disturbance of gastro-intestinal function resulting in deficient production, absorption or utilisation of one or more essential factors. The serum calcium is low and the plasma phosphorus is low or normal. The total fat in the stools may reach 40 per cent, or more, and the bulk of this is unsplit fat. The clinical and radiological features are exactly the same as in dietetic osteomalacia. An opaque enema will reveal dilatation of the colon. In treatment the fat in the diet must be cut down to a minimum, and the calcium salts and vitamins kept high. Vitamin D must be given in a solid and not in an oily medium. The prognosis of this type of osteomalacia is good especially in young people. Splinting or even osteotomy may be necessary to correct deformities such as genu valgum. The pelvic deformity may necessitate Cæsarean section.

OSTEOGENESIS IMPERFECTA

Synonyms.—Fragilitas Ossium Congenita; Osteoporosis Congenita; Congenital Osteopsathyrosis; Osteopsathyrosis Idiopathica.

Definition.—A generalised disease of the skeleton, congenital, and in some 25 per cent. of cases hereditary, in which the bones are so fragile that repeated fractures occur. Multiple fractures may occur in utero (pre-natal type of Vrolik, 1849), or fractures may not occur until after birth (post-natal type of Lobstein, 1833). Both sexes are affected equally.

Ætiology.—This is unknown.

Pathology.—In both types the basic defect appears to be defective osteoblastic activity. The cortex of the bones may be scarcely thicker than paper, and the trabeculæ of spongy bone are extremely thin. In the pre-natal type many fractures are seen; in some cases practically every bone in the body has been fractured. The older fractures exhibit good callus formation. In extreme cases, especially in the pre-natal type, the cranial ossification is so disorganised that the vault of the skull consists of a mosaic of small Wormian bones. Congenital hypoplasia occurs in other mesenchymal tissues, notably the ligaments and the sclerotics. There is no evidence whatever of vitamin deficiency. No abnormality in the serum calcium,

plasma phosphorus, or calcium output has been demonstrated. The plasma phosphatase tends to show a raised value but this is not constant.

Symptoms.—The general health of the patient is good but fractures occur from the most trivial violence or even normal muscle action. In the course of time, 20, 30, or even 100 spontaneous fractures may occur. They are often subperiosteal and cause little pain. The patient tends to be short in stature and slender in build. As a result of anomalous cranial ossification, the shape of the head is often striking. A bitemporal protuberance so marked as to turn the ears outwards is frequently observed, but protuberances in the occipital and frontal regions are also seen. Every bone in the body may be deformed. The limbs are often bowed and of unequal length. Kypho-scoliosis, distortion of the ribs and sternum, and asymmetry of the pelvis all occur. Three other defects are commonly found in association with the fragile bones, namely, leaden blue sclerotics, a tendency to dislocation of joints, and after the age of 20 years otosclerotic deafness. Amongst the adult population affected with blue sclerotics approximately 60 per cent. have an associated liability to fracture, approximately 60 per cent. an associated otosclerosis, and 44 per cent. suffer from all three defects. Osteogenesis imperfecta sometimes occurs in an hereditary form without blue sclerotics.

Diagnosis.—Severe cases and all those with blue sclerotics are unmistakable. In the new born great shortening of the limbs may suggest achondroplasia, but the skull is quite different. Cases of spontaneous fracture in the adult occurring in hyperparathyroidism, hyperthyroidism, myelomatosis, osteoclastic carcinomatosis, and neuropathic atrophy of bones really cause no difficulty.

Prognosis.—Severe cases of the pre-natal type are either stillborn or live only for a short time. In post-natal cases the condition proves more severe the earlier the first fracture appears. Multiple fractures in the first few years of life may lead to such deformities that the patient can never walk and may die before puberty. In those who survive, the liability to fractures tends to become less before puberty. In general the longer the patient lives the greater will be the improvement, and in many of the adult cases the disability is slight only.

Treatment.—The utmost care must be taken to avoid the occurrence of fractures. Treatment consists in gentle handling and careful splinting. Union usually occurs without delay and is firm. Dislocations are reduced without difficulty. Vitamin D, calcium salts, and a high calcium diet have no effect on the course of the illness.

OXYCEPHALY

Synonyms.—Tower Skull; Steeple Head; Sugar Loaf Head; Acrocephaly; Craniostenosis.

Definition.—A congenital deformity of the skull due to premature synostosis of the cranial sutures. The skull is short from front to back and its vertical diameter is increased. Allied forms of craniostenosis are scaphocephaly, the boat-shaped head, and plagiocephaly, the obliquely flattened head.

Ætiology.—This is unknown. The disease is more common in males than females. It is sometimes hereditary and familial. It is usually present at birth but it may develop subsequently up to the age of six.

Symptoms.—In its slightest form it attracts attention, while in its grosser forms there is no passer-by but is shocked by the disfigurement and repelled by its hideousness. The forehead is much increased in height, sloping gradually upwards to the vertex with feebly marked superciliary arches. The vertex of the skull appears pointed instead of flattened or rounded, and a thin bony prominence is sometimes felt in the region of the bregma. The hairy scalp may be raised above the normal level and present the appearance of being perched on the top of a cone. Viewed laterally, the ears appear placed on a lower level than normal. Proptosis is present in most cases, and it may be so considerable that the eyeballs become dislocated in front of the lids. Failure of closure of the eyes, especially during sleep, may lead to lachrymation and conjunctivitis. Divergent squint is common and nystagmus is present in some cases. Symptoms arise from insufficient room within the skull for the developing brain. There is increased intracranial pressure with headache and sometimes vertigo. The condition is compatible with normal intelligence, but not infrequently optic atrophy supervenes. This is secondary to papilloedema in some 85 per cent. of cases. In the remainder it is brought about by narrowing of the optic foramen and is of the primary type. The sense of smell is often completely lost, but taste is affected very rarely. Hearing is unaffected. The following associated congenital malformations have been described in a few cases: webbing of the fingers and toes; malformation of ears, elbow and shoulder joints, and fingers.

Radiological appearances.—Radiographs show an increased vertical diameter of the skull with its highest point either at the bregma or somewhere between it and the lambda. The anterior fontanelle closes late, and its site is marked by a slight protuberance over which the bone is thinned. The sutures of the vault are partly or entirely absent, but the basal suture between the sphenoid and the occipital bone may be widely open. The air sinuses are rudimentary, and the middle fossa bulges forward. The most characteristic feature is the presence of numerous deep convolutional markings.

Prognosis.—The optic atrophy, whether primary or secondary, may advance to complete blindness. There is nothing to show that oxycephaly shortens life.

Treatment.—Anodynes should be used in the relief of headache. If the symptoms of increased intracranial pressure become marked, and the changes in the optic discs progress, decompression may be necessary.

DISEASES OF ENDOCHONDRAL OSSIFICATION

ACHONDROPLASIA

Synonyms.—Chondrodystrophia foetalis (Kaufmann); Micromelia foetalis.

Definition.—A disease of foetal life in which defective endochondral

ossification makes the bones preformed in cartilage short, but stout and strong.

Ætiology.—This is unknown. Both sexes are affected equally. It is hereditary and has been recorded in six generations. Several members of the same family may be affected. The condition is unrelated to rickets, cretinism, syphilis, or tuberculosis.

Pathology.—The essential abnormality is found in the cartilaginous epiphyses. The cartilage does not prepare itself for ossification, which is in consequence so slow that the long bones are too short. Since, however, the periosteum goes on laying down bone normally, the bones are stout and strong. The membrane bones of the skull are unaffected, so that the calvaria is of normal size. Premature synostosis of the cartilaginous bones at the base of the skull leads to shortening, and consequent depression of the bridge of the nose. The clavicles are not affected. The pelvis is distorted and contracted, the sacrum being tilted forwards. Extreme lordosis may be present. The costo-chondral junctions are enlarged to form a rosary. The scapula is so small that the glenoid fossa scarcely holds the head of the humerus.

Symptoms.—The patient is dwarfed but of normal intelligence. The usual height of the adult is about four feet. The vault of the head is large and the frontal and parietal eminences prominent. The face is small and the nose has a depressed and flattened bridge. The nostrils are large, the lips thick, and the lower jaw and chin well developed. The teeth are normal. The trunk is of normal size but the extremities are much shortened, and with the arms at the sides the fingers reach no farther than the great trochanter of the femur. The humerus and femur are relatively more shortened than the other bones of the extremities, so that the proximal segments of the limbs show the most marked shortening. The arms are muscular and are held a little abducted from the trunk. The hands are short, thick, and trident-shaped, the fingers being almost equal in length. The lower limbs are thick and often show deep transverse furrows as if there were redundancy of the soft parts. This appearance is due to the packing of well-developed muscles into the restricted long axis of the limb. This muscular development enables the achondroplastic to perform feats which are surprising in one so small. He rises from the lying-down position by a characteristic springing movement from the legs without any assistance from the arms. The curving and enlargement of the ends of certain bones gives rise to bow legs and beading of the ribs. The lumbar curve is increased owing to tilting forward of the sacrum and excessive development of the buttocks. In consequence the gait has a peculiar duck-like waddling character. The genital organs are normal. The fact that the female may become pregnant makes the pelvic deformity of great importance. The conjugate diameter is greatly narrowed, and it is almost impossible for an achondroplastic woman to give birth to a living child except by Cæsarean section. That the disease has existed for something like five thousand years is known from models found in mummies of two achondroplastic gods of ancient Egypt, namely Ptah-Sokar and Bes. In the Middle Ages the attractive antics of achondroplastics made them much sought after as court jesters or dwarfs. To-day not infrequently they play the parts of clowns at fairs, circuses and music-halls, and sometimes break chains on the stage.

Diagnosis.—In the new born the great shortening of the limbs may suggest osteogenesis imperfecta, but the skull is quite different. In childhood the malady is readily distinguished from rickets and congenital syphilis by careful attention to the physical signs. Achondroplasia differs from cretinism in that the patient is of average intelligence, and has normal skin, hair and voice. The pituitary dwarf presents no difficulty because the limbs and trunk are in perfect proportion.

Prognosis.—The majority of infants suffering from achondroplasia are either still-born or die shortly after birth. If the child does survive, the expectation of life is normal. The female achondroplastic faces greater risks in parturition than a normal woman.

Treatment.—No treatment is of any avail. Orthopædic treatment for bow legs is unnecessary. The pelvic deformity may necessitate Cæsarean section.

DYSCHONDROPLASIA

Three clinical conditions are included under this heading. In all of them islands of ectopic cartilage are found giving rise to multiple ecchondromata or enchondromata. The three conditions are grouped together because of one feature they have in common, namely arrest or perversion of the normal process of endochondral ossification in certain bones. This change differs from that seen in achondroplasia only because it is neither symmetrical nor universal. Different manifestations of dyschondroplasia may occur in various members of the same family.

(i) *Hereditary multiple ossifying ecchondromata (hereditary deforming chondrodysplasia, diaphysal aclasis, or multiple cartilaginous exostoses).* This is a fairly common disease in which multiple bony tumours are found in association with certain other skeletal deformities. It is hereditary and may affect several individuals of the same family. It is more common in males than in females in the proportion of 3 to 1. It is usually discovered in childhood. Palpable bony tumours up to 2 cm. or more across are found more or less symmetrically placed near the knee, shoulder, hip, ankle and wrist. The scapula, ribs and pelvic bones may sometimes be affected. The stature is shortened and the limbs may be unequal in length. In the majority of cases the ulna and fibula are disproportionately short in relation to the radius and tibia. Bowing of the radius, ulnar deviation of the hand, irregular length of the fingers, and valgus deformity of the foot all may occur. Sarcoma supervenes in 5 per cent. of cases. Local exacerbation of symptoms in a patient over 30 years of age may be the first indication of its onset. Rarely pressure of an exostosis upon the spinal cord may cause paraplegia, or upon a nerve trunk pain or local paralysis. Aneurysm has been recorded from pressure upon an artery. The radiological appearances are characteristic. The metaphysis of the bone affected is broadened and distorted, and ossifying ecchondromata with broad bases and pointed tips project from it. The cartilaginous cap of the tumour is not seen unless it is calcified. The earlier the ecchondroma occurs the nearer to the centre of the shaft will it be. Where ecchondromata protrude between adjacent bones such as the tibia and fibula, local fusion may occur. The ulna is

likely to be short and to end in a point, articulating with the radius on its mesial aspect but not partaking in the carpal articulation. Usually no treatment is required but should it be necessary to remove any particular swelling this is easily carried out.

(ii) *Multiple chondromata (Enchondromatosis)*. This is a rare disease affecting the bones of the hands and feet. Cartilaginous swellings in the fingers and toes begin in childhood and increase in size up to the age of 30 years. The swellings are firm, elastic, rounded and slightly translucent. The skin over the larger ones may be tightly stretched and shiny and show prominent veins. The hands and feet may become hideously deformed. Sometimes a rib near the costal cartilage, the sternum, the pelvis, and the scapula are affected. In certain cases the ulna and fibula are disproportionately short as in diaphysial aclasis. Spontaneous fractures may occur, and sarcoma may supervene after years. Radiologically chondromata are seen as rounded, eccentric translucent areas expanding the corticalis, interrupting its outline, and projecting into the soft tissues. Sometimes the swellings are trabeculated and they may contain dense, punctate, calcified areas. Where operation is undertaken to excise some of the chondromata care must be exercised to avoid spontaneous fracture of the phalanges or metacarpals.

(iii) *Unilateral chondrodysplasia (Ollier's disease)*. This is a very rare type of chondrodysplasia occurring in children and sometimes familial. It usually has a completely unilateral distribution, but some cases have only one bone or one limb affected, and others are bilateral. Some abnormality is often first noticed between the first and second years of life, when as a rule one limb is found to be shorter than its fellow. The difference in length becomes progressively greater as growth proceeds. Deformity may occur either because weight bearing causes bending of the bone, or because of the different rate of growth where only one of the paired bones is affected. Most patients seem to reach adult life, when their symptoms are mainly those of their deformities and sometimes of a secondary arthritis. In a small proportion of cases sarcoma supervenes. The diagnosis largely depends upon examination of radiographs. The ends of the long bones show translucent longitudinal striæ interrupted by small pale mottled areas and dark punctate spots. In the areas affected there is extensive alteration in the pattern of the corticalis and spongiosa, but the centre of the shaft remains normal. As the child grows older the typical striped appearance disappears and is replaced by dense punctate speckling due to areas of calcification. The disease has occasionally been mistaken for osteitis fibrosa, but the radiological appearances are pathognomonic. Treatment is concerned with the prevention and relief of deformities, and proceeds along the usual orthopædic lines. Osteotomy is sometimes necessary. Fractures are of fairly common occurrence, and like the osteotomies appear to unite well.

DISEASES OF THE BONE MARROW

MULTIPLE MYELOMA

Synonyms. — Myelomatosis ; Kahler's Disease ; Plasmacytoma ; Hæmatogenous Myeloma.

Definition.—A fatal disease characterised by the development of multiple tumours in the skeleton, which arise from cells of the bone marrow. It is very rare. The bones are affected in the following order of frequency : spine, ribs, sternum, skull, scapula, pelvis, clavicle, humerus and femur.

Ætiology.—Multiple myeloma is of unknown origin. It is a malignant neoplasm of the hæmatogenous marrow occurring in multiple foci. The disease is related to leukemia, but differs from it in the sharper localisation of the neoplasia, the absence of enlargement of the spleen or lymph-glands, the much smaller tendency for the abnormal cells to enter the blood stream and the frequent appearance of Bence Jones protein in the urine. Intermediate forms occur with features of both diseases. It is associated with interesting alterations of protein metabolism. The disease is sometimes familial. It begins most commonly at the age of 55, and only 10 per cent. of cases occur before 40. The sexes are affected in the proportion of three men to two women.

Pathology.—Multiple deep red or reddish-grey sharply defined tumours are found distributed throughout the red bone marrow. They are usually a few millimetres in diameter and very numerous. Occasionally a tumour may reach a diameter as great as 5 cm. They are composed of blood-forming cells, either myelocytes, myeloblasts, erythroblasts, or cells resembling plasma cells. They erode bone, sometimes expand the cortex, and cause deformities and spontaneous fractures. Rarely a diffuse hyperplasia of the marrow is associated with foci of tumour formation. Tumours may also be found outside the skeleton in the tonsils, liver, spleen, kidneys, or sex-glands, and these lesions may even precede those in the bones. The marrow tumours give rise in the urine to the Bence Jones protein which appears as a cloud when the urine is heated to 55° C., redissolves at 85° but reappears on cooling. It is found in 75 per cent. of cases, from a trace to a large amount. In some cases it appears early in the disease, in others late. Its occurrence may be continuous or periodic. Sometimes a substance allied to amyloid material is deposited in the muscles and in nodules connected with the periosteum, bursæ, tendon sheaths and joints. It is possible that both the Bence Jones protein and the amyloid substance are produced from the breakdown of myelomata. The serum globulin is usually increased even as much as 8 per cent. (normal 2 per cent.). The albumin-globulin ratio may drop from the normal 2·2 to a figure as low as 0·5. The formol-gel reaction is positive (see p. 253), and there is a very rapid rate of sedimentation of the blood. Metastatic calcification while by no means constant has been frequently observed in the kidney, lung, stomach, myocardium and uterine mucosa. The serum calcium is usually normal, but, taking into account the bone destruction which occurs as the result of erosion by the marrow tumours and also the metastatic calcification, it is not surprising

that high serum calcium values have sometimes been recorded. Figures from 13 to 16 mg. per 100 c.c. have been found. Those cases with a normal serum calcium have a normal calcium output, while those with a high serum calcium have an output up to double the normal. Where renal insufficiency complicates multiple myeloma the plasma phosphorus is found to be high and may rise as the kidney condition becomes worse. The parathyroids are not enlarged in multiple myeloma.

Symptoms.—The initial symptom is pain, often bilateral, in the thoracic, abdominal and lumbar regions, and sometimes in the neighbourhood of the joints. Progressive kyphosis or angular curvature of the spine with loss of total height follows. The spine, sternum and ribs may be tender on percussion. It is unusual for any of the myelomata to be palpable. In 60 per cent. of all cases spontaneous fracture occurs in the ribs, sternum, or later in the long bones. In no other type of bone tumour does pathological fracture occur so frequently. In some cases amyloid masses may be palpable as firm, rounded, slightly tender, subcutaneous nodules more than a centimetre in diameter. They are felt especially in the scalp, along the spine, near the joints, and in the musculature, particularly that of the pelvic and shoulder girdles. There is usually a hypochromic anæmia, which becomes aggravated in the terminal stages. In a few instances cells of the type which constitutes the tumour enter the blood stream in larger or smaller numbers, and it is probable that they can be found in the majority of cases if a sufficiently careful search is made. In rare instances the anæmia is of the leucocrythroblastic type (p. 783). Nephritis without hypertension is fairly common. The temperature is usually normal but recurring fever has been observed. The patient ultimately becomes bedridden and cachectic. It seems justifiable on clinical grounds to consider separately what may be called the vertebral form of the disease. Here the growth is confined for some time to the vertebral and extradural tissues. Moreover, death may occur before the growths become widespread, and sometimes without the Bence Jones protein having appeared in the urine. In this variety the patient rapidly develops signs of a transverse spinal lesion with blockage of the spinal canal. The thoracic cord is usually the site of compression and there is focal spinal tenderness. Radiographs show destruction of the corresponding vertebral body.

Radiological Appearances.—In radiographs the marrow tumours are found mainly in the spine, ribs, sternum and skull. They are seen as clean-cut elliptical or circular areas of complete translucence, set closely together and varying from 1 mm. to 5 cm. in diameter. The larger tumours may expand the cortex of the bone affected. There is a good deal of generalised osteoporosis throughout the affected bones. The spine shows collapse of the bodies of one or more vertebræ. The skull is not thickened. Pathological fractures, especially in the ribs, are very common.

Diagnosis.—Once the lesions have appeared in many bones the diagnosis is easily made. The age of the patient, multiple involvement of the bones of the thoracic cage, spontaneous fracture of a rib, Bence Jones protein in the urine, progressive anæmia, cachexia and characteristic radiographs make an unmistakable clinical picture. Biopsy of a portion of bone or examination of a bone-marrow smear from a sternal puncture may reveal the characteristic myeloma cells. Secondary carcinomatosis of bones may

cause difficulty, especially in cases in which the primary growth is symptomless. It is essential to differentiate the disease from generalised osteitis fibrosa (hyperparathyroidism). There is some resemblance in the clinical picture as it affects the skeleton, but the presence of the Bence Jones protein and the blood chemistry are characteristic. In multiple myeloma the serum calcium is usually normal. If it is high it is associated with a high plasma phosphorus, whereas the characteristic effect produced by parathyroid hyperfunction is a high serum calcium with a low plasma phosphorus. In osteomalacia the patient is usually a woman in the child-bearing period of life, and a good deal of bending occurs in the bones. The blood chemistry is characteristic. Sometimes in the early stages of multiple myeloma widespread pain in the thoracic, abdominal and lumbar regions may lead to a mistaken diagnosis of fibro-myositis. In tuberculous caries of the spine neither the ribs nor the sternum are involved. The fact that the Bence Jones protein is found in the urine in an occasional case of leukemia need cause no mistake. In those cases in which nephritis complicates multiple myeloma the albuminuria may cause difficulty. The Bence Jones protein may be detected in the presence of albumin by making the urine slightly acid with acetic acid, boiling it and filtering while hot, using a funnel with a hot-water jacket. If Bence Jones protein is present the filtrate will become cloudy as it cools.

Prognosis.—The prognosis is hopeless. Death often occurs within six months of the onset of symptoms, but occasionally a patient survives for two years or more. Broncho-pneumonia, cachexia, or compression paraplegia with ascending pyelo-nephritis are the usual terminal events.

Treatment.—The patient should be treated by rest in bed, anodynes and suitable splinting when necessary. Occasionally deep X-irradiation can be used with good effect. It alleviates pain and reduces the size of the tumours, but it does not retard the progress of the disease. It is clearly unjustifiable to explore the neck in search of a parathyroid tumour. When the symptoms and signs point to compression of the spinal cord surgical intervention may be worth while. Laminectomy reveals a grey or reddish-grey extradural mass either pushing the cord backward or encircling it. Removal of the mass decompresses the cord and is followed by improvement. Deep X-irradiation and the wearing of a spinal brace are advised after laminectomy.

DONALD HUNTER.

LESLIE J. WITTS.

GAUCHIER'S DISEASE

In 1922 Pick discovered a gross osseous form of Gaucher's disease. It is exceedingly rare. The symptoms are pain in the bones, pathological fractures and sometimes angular curvature of the spine. In radiographs the bones show patchy osteoporosis. A characteristic feature is that the ends of the femora are widened evenly. Both skull and pelvis may be involved. Sometimes scattered through the bones there are focal pale rounded areas which expand the cortex. These areas are deposits of kersin, a galactolipin. The usual characteristics of Gaucher's disease are, of course, present (see pp. 834, 835).

HAND-SCHÜLLER-CHRISTIAN'S DISEASE

The lesions of Hand-Schüller-Christian's syndrome (lipoid *granulomatosis* or *xanthomatosis* of bones) are not confined to the calvaria, the orbit or the sella turcica (see p. 836). Erosions of the maxilla and mandible have been described, resulting in loosening or falling out of the teeth. Erosion of the petrous bones may lead to a syndrome simulating otitis media, and bilateral deafness has been observed. Large areas of rarefaction have been described in the long bones of the extremities, and in the spine, pelvis, ribs and clavicles. Pain may occur in the bones affected, especially the head, pelvis and thigh. Spontaneous fracture is not uncommon. When the pelvis is involved there may be deformity, including shortening of one lower limb. In some cases the skull escapes entirely, diabetes insipidus and exophthalmos being absent. Radiologically the deposits of cholesterol-ester are seen as irregular clean-cut translucent areas sometimes with a few coarse trabeculæ. In order to distinguish the condition from multifocal osteitis fibrosa it may be necessary to excise a portion of bone for histological section. The lesions tend to yield temporarily to treatment by X-irradiation.

DONALD HUNTER.

SECTION XIX

DISEASES OF THE SKIN

I. ANATOMY AND PHYSIOLOGY

IN order that the diseases which affect the skin may be understood it is necessary to give a brief account of the anatomy, physiology and general pathology of the skin.

ANATOMY.—The skin is a fibrous structure varying considerably in thickness in different parts of the body and covered externally by several layers of epithelial cells. On section its main bulk is seen to be made up of white fibrous tissue bundles running chiefly parallel to the surface and bound together by thin fibres of elastic tissue. The surface of this fibrous mass, which is called the *dermis*, is not level but is surmounted by a number of finger-like projections, called *papillæ*, which fit into corresponding depressions or pits on the under surface of the epithelial covering which is called the *epidermis*.

In the fibrous stroma of the dermis blood vessels, lymphatics and nerves ramify. The *arteries* form a plexus of large vessels at the junction of the dermis with the subcutaneous fatty layer and from this deep plexus arteries pass upwards, frequently near hair follicles or sweat ducts to which numerous twigs are sent, to another superficial or sub-papillary plexus situated just below the bases of the *papillæ*. From this smaller vessels pass upwards to end in the *papillæ*. The *veins* follow a similar course in the opposite direction.

Lymph circulates freely in the spaces between the cells of the epidermis and the fibres of the dermis, but definite lymphatic vessels are also found in the *papillæ* and in the dermis, accompanying the blood vessels.

The *nerves* of the skin are both medullated and non-medullated. They also follow the course of the blood vessels and are distributed to the hair-follicles, sweat and sebaceous glands, blood vessels, *arrectores pili* muscles and to the connective tissue bundles in their passage through the dermis. Losing their medullary sheath in the sub-papillary layer some fibres pass up and are distributed to the *papillæ* and to the basal and mucous layers of the epidermis, while other medullated fibres end in curious whorls in the *papillæ*, which are called the touch corpuscles of Meissner; a few end in small ovoid bodies, known as Pacinian bodies, in the subcutaneous tissue.

The *epidermis* consists of several layers of cells, varying considerably in thickness in various parts of the body. The layer nearest the dermis consists of regular cubical cells to which it is intimately attached, and it is from this layer that the rest of the epidermis is developed; it is spoken of as the *stratum germinativum* or *basal layer*. The layers above this consist

of cells in various phases of transformation into horn cells, which are seen in their final form in the outermost layers. Above the basal layer there are several layers of large polyhedral cells with large nuclei and a spongy cell substance; they are bound to one another by fine fibrils from which they have obtained the name "prickle" cells; this is the *mucous* or *Malpighian layer*. Above this are one or two layers of lozenge-shaped cells, lying parallel to the surface of the skin, whose protoplasm contains large deeply staining granules, giving to it the name *granular layer*. Then comes a thin transparent layer, the *stratum lucidum*, and above this the *horny layer*. Here the cells have lost their nuclei and protoplasm, and consist only of a cell capsule which has been converted into a highly resisting substance called keratin; the cells are intimately bound together and can only be separated with great difficulty. Thus a strong protective layer is produced which can only be destroyed by strong acids or alkalis or by violence.

In the cells of the basal layer are produced granules of *pigment* which act as a protection against light rays. The pigment is an iron-free substance named *melanin*, and its method of production is still a matter of controversy. In the dark races the deeper cells of the mucous layer also contain melanin granules, and these can also be found in wandering cells in the dermis but are not formed in these cells.

Dipping down from the epidermis into the dermis are certain epithelial structures, the hair follicles with their sebaceous glands, and the sweat glands.

The *hair follicles* are pockets of epithelium which contain in their walls all the layers of the epidermis in a modified form. They penetrate the whole thickness of the dermis and often pass into the subcutaneous tissue for some distance. The *hairs* grow from enlarged papillæ at the bottom of the pits and also consist of modified epidermis, so modified that the cellular structure is only visible on the outer layers formed of superimposed scale-like cells, the cuticle of the hair; the remainder of the hair structure consists of an outer fibrous part, the cortex, and a more succulent centre, the medulla. Hairs are present all over the skin except on the palms and soles, and vary very much in size. Their ordinary characteristics need no description. The hair follicle is inserted obliquely in the skin, and on the aspect where it forms an obtuse angle with the surface, a small band of unstriated muscle, the *arrector pili*, is found, attached below to the hair follicle near the papilla and above to the fibrous tissue underlying the surface epidermis. This muscle on contraction erects the hair.

From the same side of the hair-follicle, and lying between it and the muscle, so that it is compressed when the muscle contracts, is found a sacculated gland growing out of the follicle; this is the *sebaceous gland*. It secretes an oily substance which lubricates the hair and the skin surrounding the follicle. The secretion is produced by fatty degeneration of the cells of the gland itself, and is expressed by contraction of the *arrector pili* muscle. These glands vary much in size and in some cases far exceed that of the hair follicle; in this case they often open directly on the surface of the skin in common with the hair follicle. They are most developed on the face, back, chest and scrotum.

The other epithelial appendages are the *sweat glands*, which are found everywhere in the skin. They are tubular structures which pass down to the lowest part of the dermis or into the subcutaneous tissue and end in a coil,

the *sweat* or *coil gland*, the straight portion passing to the surface being the *sweat duct*. Both the duct and gland consist of a single layer of cubical cells which becomes continuous with the basal layer of the epidermis. The duct has no special epithelial lining through the epidermis. Involuntary muscular fibres, which expel the secretion of the gland and are under the control of special pilo-motor centres, are present among the coils of the glands. The secretion of the gland is a true secretion and is not produced by degeneration of its cells, as in the case of the sebaceous glands. Certain large sweat glands, called *apocrine glands*, occur in the axilla, nipples and pubic region, which do, however, show breaking up of the cell protoplasm during activity.

The only other skin structures that require mention are the *nails*. These are simply modifications of the horny layer of the skin. The nail grows from that portion of the nail bed which is partly hidden by the nail fold and partly seen as the lunula of the nail, which forms a pale half-moon shaped area above that structure; this area is called the *matrix*.

PHYSIOLOGY AND PATHOLOGY.—The functions of the skin are four in number—(1) It forms a protective covering over the whole body; (2) it is an organ of secretion; (3) it is the seat of tactile sensation; and (4) it plays an important part in regulating the temperature of the body. The skin also allows of absorption, though this can scarcely be considered one of its primary functions. From the point of view of dermatology the two first functions are the most important.

The *protective function* is a double one: firstly the skin as a whole lying on a loose connective tissue pad, protects the deeper structures from damage by acting as a buffer; secondly, the resistant characters of the horny layer protect from irritants, not only the deeper structures, but also the layers of the skin lying beneath it, for the moist cells of the body unprotected by these dry keratinised cells would perish if exposed even to the ordinary atmosphere. Damage to the horny layer is responsible for a very large group of inflammations of the skin.

The horny layer, however, does not act quite alone: it is made more impermeable to simple external irritants by the presence of a thin layer of oil on its surface which is provided by the secretion of the sebaceous and sweat glands. As will be shown later, absence or deficiency of this oily secretion renders the skin much more susceptible to external irritants. On the other hand, excessive sweat secretion from the large amount of water it contains may make the horny layer sodden, and therefore more liable to damage. Similarly an excessive sebaceous secretion tends to make the horny layer thicker and is an excellent medium for the growth of organisms.

It must further be remembered that the horny layer does not form a complete sheet, but that innumerable invaginations which form the hair follicles and sweat ducts are present. These considerably weaken the protective power of the horny layer, and it will be found that at these spots inflammatory reactions, due to damage of this layer, are most likely to occur. It is also practically certain that absorption takes place at these follicular openings.

The *secretions of the skin* are the sweat and the sebum, the latter of which is the secretion of the sebaceous glands. The former is a watery fluid which contains traces of sodium chloride and other mineral salts, extractives, and a very small quantity of urea and fats. It varies very much in quantity, but

normally about equals the quantity of urine voided and, therefore, is responsible for the removal of nearly 50 per cent. of the total water excreted by the body. The main function of this excretion of water is to maintain the temperature balance of the body and, therefore, in hot weather the amount of sweat is increased in order to cope with more rapid evaporation and so keep the body surface cool, the quantity of urine being correspondingly diminished unless larger quantities of water are imbibed. In cold weather the reverse is the case.

A small amount of carbon-dioxide is also excreted by the skin, and the latter may therefore be said to be an accessory *organ of respiration*.

The sweat has special interest to the dermatologist not only from its lubricating effect on the horny layer, but also from the fact that certain drugs are sometimes excreted by it, and it is possible that some of the eruptions caused by the ingestion of these drugs may be produced by their irritating action during the process of excretion by the sweat.

The sebum is an oily secretion whose function appears to be entirely that of lubricating the hairs and surface of the skin: it not only helps to protect the skin from chemical irritants, but also from the actinic rays of the sun.

The other two functions of the skin, namely, the *tactile sense* and the *regulation of temperature*, will have been dealt with elsewhere, and as they affect the dermatologist but little, they will not be further considered here.

II. GENERAL DESCRIPTION OF SKIN DISEASES

The bulk of skin diseases fall into two great classes, inflammations and new-growths. There are in addition certain conditions which cannot be included under either of these headings, and will require special mention, namely, the disorders of secretion, of sensation, of the circulation, and of pigmentation; atrophies of the skin and certain congenital abnormalities. It will also be necessary to describe the diseases of the hair separately. As so many of the inflammations depend upon disorders of secretion, sensation and circulation, it is proposed to deal with these first. Before proceeding, however, to deal with pathological conditions of the skin, it will be useful to define the terms used in describing clinical manifestations. It must be realised, however, that these terms are used very loosely, and are only a convenient form of nomenclature.

A *macule* is a spot which is not raised above the skin; it may be vascular or pigmentary. The term is usually applied to small lesions up to the size of a pea, a larger lesion being called a "plaque" or "tâche." Large sheets of redness are generally called an "erythema."

A *papule* is a solid elevation usually not exceeding the size of a pea. If the surface is flat and smooth it is called a "plane" papule; if pointed an "acuminate" papule.

A *tubercle* or *nodule* is an elevation usually between a pea and a hazel nut in size. The term nodule is also used for small solid swellings in the substance of the skin and subcutaneous tissue which do not necessarily project above the surface.

A *tumour* is a swelling exceeding a hazel nut in size. It need not

necessarily be solid, but this term is not usually applied to thin-walled superficial fluid swellings.

A *wheel* is a circumscribed elevation of the skin of a transitory character in which œdema is so marked as to force the blood out of the superficial capillaries and so produce a dead white elevation.

A *vesicle* is an elevation not larger than a pea containing clear fluid.

A *bullæ* is a similar lesion of larger size ; in other words, a *blister* or a *bleb*.

A *pustule* is a similar lesion to a vesicle, but contains pus instead of clear fluid.

A *scale* is a lamella of the horny layer of the skin.

A *crust* is a mass produced by the drying of exudates on the skin.

An *excoriation* is an abrasion of the superficial layers of the epidermis.

A *fissure* is a crack in the skin.

An *ulcer* is a circumscribed loss of tissue involving the whole thickness of the epidermis.

A. M. H. GRAY.

III. CONDITIONS PREDISPOSING TO SKIN DISEASES

A.—DISORDERS OF SECRETION

Under this heading are included deficiency or absence of sweat and sebaceous secretion, and also excessive secretion.

ANIDROSIS OR DIMINUTION OF SWEAT SECRETION

This occurs in many diseases, but is seen in its most marked form in xeroderma and ichthyosis. It also is seen in hypo-thyroidism and in its more marked form myxedema, in the degenerating skin of old people, and in poisoning by certain drugs, of which arsenic is one of the most frequent examples.

The milder cases of hypo-thyroidism show dryness of the skin, dryness, brittleness and thinning of the hair. They improve rapidly under the judicious administration of thyroid extract.

XERODERMA AND ICHTHYOSIS

These two names are applied to the mild and severe types of the same disease. The condition is one of abnormal dryness of the skin owing to the almost complete absence of sweat secretion accompanied by an overgrowth of the horny layer of the epidermis (hyperkeratosis).

Ætiology and Pathology.—The disease is inherited and often occurs in several members of the same family. It attacks both sexes equally. The disease is usually noticed about the second year of life, but some children are born with a condition closely resembling it (*ichthyosis congenita*): these children are frequently premature and generally stillborn. There is no very

definite evidence as to whether the changes in the epidermis follow the absence of secretions, or vice versa, or whether both are dependent on a common cause; possibly the overgrowth of the horny layer is an attempt on the part of Nature to compensate for the protection usually supplied by the only secretions. Histological examination shows very marked increase in the thickness of the horny layer, which is irregular and grows directly from the mucous layer, the granular layer being absent. The sweat glands are apparently normal histologically, although they do not function normally.

Symptoms.—In the milder cases (*xeroderma*) the skin is dry and rough, and there may be a certain amount of branny scaling on the surface. On the extensor aspect of the limbs the hair follicles are prominent and contain small horny spines. The palms and soles are more lined than normal, while the flexures of the body show little change. The hair is dry and lustreless, and occasionally stunted and brittle, while in a few cases only down grows on the scalp.

In the more marked cases (*ichthyosis*) the body is covered with large fish-like scales which are firmly adherent. They may be thin, transparent and colourless, or thick and dark in colour (the so-called alligator skin). In these cases the trunk and extensor aspect of the limbs are most involved, the face and scalp often showing little change, though the changes mentioned above may be present. This dry skin is particularly liable to become inflamed on account of alteration in its protective mechanism.

There is another form in which the disease develops in localised sheets, lines or bands (*ichthyosis hystrix*), but this condition is closely related to the linear nævi and will be dealt with under that heading.

Prognosis.—The disease persists throughout life, and although it can be relieved by appropriate treatment it never really gets well.

Diagnosis.—The dryness of the skin, the origin of the disease in early life, and its persistence, and the presence of fine or coarse scaling with the absence of inflammation render the diagnosis easy.

Treatment.—This consists in an attempt to replace the natural oil of the skin. Frequent warm baths, followed by the application of some oily preparation, are usually sufficient. One of the most useful preparations is glycerin. amyli, adip. lanæ hydros. āā ptes. æq., to which may be added 2 per cent. or 3 per cent. of salicylic acid if desired. Too vigorous use of soap is to be discouraged.

Some authorities recommend thyroid internally, on the grounds that the condition is due to deficient thyroid activity, but the results obtained have been scarcely sufficient to confirm this view.

HYPERIDROSIS

Definition.—This is a condition of over-activity of the sweat glands. It may be general or local.

Ætiology and Pathology.—Sweating in febrile illnesses is not included under this heading. The generalised forms are usually seen in adults, while the localised varieties are not infrequently seen in younger people. They are both probably due to disturbances of the nervous system, though it is difficult to say that they always occur in neurotic individuals. There is

no doubt, however, that hyperidrosis is very liable to produce a neurotic condition.

Symptoms.—*Generalised hyperidrosis.*—In this condition the patient sweats excessively, often on the least exertion or excitement. The sweating may be so severe that the patient has to change his clothes several times a day—even in cool weather.

Localised hyperidrosis.—There are certain regions of the body particularly liable to excessive sweating, namely, the palms and soles, the axillæ, and the genital region and perineum. The sweating is often very excessive, and may last for a very long time; there is, however, a tendency for the condition to diminish with age, it being most marked in the latter half of the second and the third decades of life.

The sweat allows certain saprophytic organisms to grow freely, with the result that decomposition takes place, and an extremely offensive odour develops. This is chiefly noticed in the feet, and is spoken of as *bromidrosis*. Occasionally the sweat is coloured (*chromidrosis*), due to bacterial activity.

The skin constantly soaked in sweat is subject to attacks by irritants, bacterial and otherwise, and various forms of dermatitis are frequent complications of hyperidrosis, especially the forms spoken of as *miliaria rubra* and *dysidrosis* (see p. 1414).

Prognosis.—Localised cases will generally respond to treatment; but the more severe generalised cases are apt to be very persistent.

Treatment.—For the *generalised cases* frequent warm baths are required, to keep the skin clean. Dabbing on a solution of tannic acid (1 per cent.) in 50 per cent. alcohol, or a dusting powder of talc containing 3 per cent. salicylic acid, is often useful. The general health should be looked to, and all dietetic errors and habits liable to cause sweating rectified. Some cases benefit by the internal administration of bromides and belladonna.

The *localised cases*, when extreme, are best dealt with by X-rays. Ten or twelve doses, each of 120 r ($\frac{1}{3}$ skin unit), given in groups of four doses at weekly intervals, with intervals of one or two months between the groups, usually give a satisfactory result. Bromidrosis of the feet is best dealt with by frequent washing and change of socks, by bathing in 1 in 4000 potassium permanganate solution, or by washing with lysoform, or other formalin soap. The feet should then be freely dusted with the powder mentioned above.

SEBORRHOEA

Definition.—By the term seborrhœa is meant an over-activity of the sebaceous glands, resulting in an abnormally greasy skin.

Ætiology.—This condition occurs from the time of puberty onwards, gradually diminishing as age increases. It tends to affect certain races and families, but is also influenced by the habits of individuals. Gastric disturbance, constipation, anæmia, uterine trouble and the like all tend to exaggerate the condition.

Pathology.—The condition appears to be due to some disturbance of metabolism not yet fully determined. Some authorities consider that infection by certain organisms play a part, but the evidence is inconclusive.

Symptoms.—The regions affected are the face—especially the nose, naso-labial folds—the scalp, chest and back. In the milder cases the skin is greasy; in the more severe cases it is thickened—giving rise to a muddy appearance—and the follicles are patulous. This condition Darier has labelled “la kérose.” In other cases the sebaceous follicles are plugged with semi-solid sebaceous material.

Complications.—Seborrhœa is the underlying cause of many skin affections. Acne vulgaris is merely a more marked stage of the follicular plugging noted above. Infection by certain organisms producing seborrhœic dermatitis is very common, while the skin is especially liable to ordinary eczema and impetigo contagiosa. Acne rosacea is particularly liable to occur in seborrhœic individuals.

Treatment.—The general health must receive attention. Diet should be regulated to get rid of dyspepsia and constipation; especially should excess of sugars and starches be avoided. Iron and arsenic are indicated in anæmia, and uterine troubles should be appropriately treated.

Frequent washing with soap and water is necessary. Sulphur has a marked effect in diminishing the secretion and especially in preventing organisms from growing in it; it may be used as a powder—sulphur. precip. 5 parts, pulv. talc to 100 parts; or as a lotion, potass. sulphurat. min. 60; sp. vini. rect. fl. oz. 2, aquam ad fl. oz. 8.

B.—DISORDERS OF SENSATION

The disorders of sensation comprise hyperæsthesia, anæsthesia and paræsthesia.

Hyperæsthesia is generally symptomatic of some organic or functional disease of the nervous system, and has little or no importance in dermatology.

Anæsthesia also is usually symptomatic; but one form occasionally comes under the notice of the dermatologist first, namely, that associated with syringomyelia. The individuals affected show anæsthesia with trophic changes in the skin of the fingers, often with whitlows and other signs of skin sepsis. Further investigation shows the lesions to be only part of a more general disease of the nervous system. This type is spoken of as Morvan's disease, and is dealt with elsewhere (p. 1731). Localised areas of anæsthesia, with redness, are frequently an early sign of leprosy (see p. 122).

Paræsthesia forms the most important group from the dermatological point of view, as it includes itching or pruritus.

PRURITUS

Under this heading are included those cases of itching of the skin in which there is no other obvious dermatosis. It may be general or local.

GENERALISED PRURITUS

Ætiology and Pathology.—The causes of general pruritus, apart from that due to animal parasites, may roughly be classified as follows :

1. The presence of toxic substances circulating in the blood. For instance, in diabetes and jaundice from the presence of sugar and bile-salts in the blood. Similarly certain ingested drugs, such as opium, will cause it. It also occurs in gout and nephritis.

2. As a symptom of some blood diseases, such as leukæmia and lymphadenoma.

3. In atrophy of the skin in old people, the so-called senile atrophy.

4. In a large group of cases in which no cause can be found, and which are considered of functional origin.

Symptoms.—In the majority of cases itching is the only or main symptom, but others, such as tingling and burning, may be present. It may vary very much in intensity, and may be intermittent. Some people suffer most in hot weather ; but it is more frequent on exposure to cold. Hot baths sometimes being on attacks. In the more severe types the condition is most distressing, the patient rarely has any peace, but is constantly scratching, it keeps him awake at night, and as a consequence his general health suffers and he becomes a nervous wreck. In spite of this, it is extraordinary how little sign there is of scratching on the skin ; some cases show a certain number of linear excoriations, but the scratch lesions are rarely as marked as they are in the localised forms and in parasitic affections. Sepsis of the skin is rarely seen.

Diagnosis.—It is essential to try and find the cause. Parasitic infection should be excluded in the first place, and then internal ailments, including the blood diseases. The presence of marked scratch lesions, especially with sepsis, suggests a parasitic origin ; the special distribution of parasitic lesions will be considered under their appropriate headings (p. 1441 *et seq.*).

Treatment.—In the cases in which no definite cause can be assigned it is necessary to attend to the general hygiene of the patient. Diet requires careful regulation ; alcohol, strong tea and coffee should be forbidden, as should all substances likely to produce urticaria, *e.g.* shell-fish, strawberries, etc. ; hot and highly seasoned dishes and excess of nitrogenous food are better avoided. Any disturbance of digestion, especially constipation, should be treated. In bad cases sedative drugs may be required, of which bromides, belladonna, cannabis indica and valerian are the most useful. Injections of pilocarpine are recommended in dry skins. In cases of sleeplessness, hypnotics may be required. Opium should be avoided, as, apart from other reasons, it may increase the itching.

Local treatment depends on the conditions which excite the pruritus : for instance, in some cases baths are advantageous, while in others they increase the itching. If the skin is dry, as in senile pruritus or when associated with xeroderma, oily preparations are beneficial : glycerin of starch and lanolin in equal parts, or cocoa-nut oil with a little soft paraffin, are useful preparations, especially if 1 or 2 per cent. menthol or phenol is added. Liq. picis carbonis and liq. plumb. subacet. fort. aa min. 120, and milk to fl. oz. 8 is often satisfactory. Most cases, however, get most relief from alkaline

baths and lotions: for the latter *lotio alkalina* (sodium bicarbonate and borax, 1 per cent. of each in distilled water) answers well.

It is very important to see that the patient changes his undervest at night. Many patients do not do so, and this undoubtedly predisposes to pediculosis. Even, however, in cases where no pediculosis appears to be present, cases are often cured by attention to this detail.

LOCALISED PRURITUS

Certain parts of the body are liable to pruritus; these are the anus, vulva, and scrotum. Other local areas, however, may be attacked, such as the front of the ankle, lower part of the leg, thighs, back of neck and scalp.

Ætiology and Pathology.—Most of these localised cases probably start from some transitory cause, which gets better; but a vicious circle has been started, the scratching bringing on itching, and this causes scratching again. In the case of the anus, piles are a frequent cause. Some cases are, as Castellani has shown, due to fungus infection. Vaginal discharge frequently starts a vulval pruritus, as do sugar and other irritating substances in the urine. Sweating and friction of clothes, and possibly some parasitic condition, such as *dhobie itch*, may start a scrotal pruritus.

For the other cases it is generally difficult to find a cause, and it is usually necessary to treat symptoms.

Symptoms.—The localised itching is often followed by marked changes in the skin from rubbing and scratching. The usual change noted is that called *lichenification*, in which the skin becomes thick and rigid, the lines of the skin deeper, and the area assumes a dull purplish colour, and on clearing up often leaves deep brown pigmentation. This is well seen in the patches in the flexures and on the limbs, but is modified in the moist parts, where it usually takes on a white sodden and swollen appearance, surrounded by a bright red inflammatory zone. The surface is often covered by numerous excoriations or blood-stained crusts. Occasionally these excoriations may become septic, and ulceration may occur. The symptoms are often so severe as to affect the patient's health by sleeplessness and worry.

Treatment.—The treatment recommended for generalised pruritus is often ineffective in the localised cases, such as that directed towards obtaining sleep.

The first thing to do is to remove any local cause; *e.g.* vaginal discharges may require treatment. The bowels should be made to act freely by paraffin, saline aperients or enemata; aloes is better avoided. Irritating food, especially coffee, alcohol, curries, etc., should be interdicted. Piles may require surgical treatment, and any rectal discharge, fistula or worms should be dealt with. All the parts should be carefully washed, mild alkaline lotions or weak antiseptics being useful, and afterwards dried thoroughly and a talc or zinc oxide powder applied. Further relief may be obtained by the application of 1 per cent. phenol and camphor cream, or 5 per cent. oleinum cocainæ (B.P.C. 1923). If these milder measures fail, the parts may be painted with silver nitrate, grs. 10, ap. æther. nit. fl. oz. 1, twice or three times a week, and a bland cream or mild alkaline lotion applied. In cases due to fungus infection, Castellani's fuchsin paint (see p. 1438) usually proves efficacious.

The most radical results, however, are obtained by X-rays. Three or four doses of 120 r ($\frac{1}{2}$ skin unit) given at weekly intervals to the affected area will nearly always remove the itching completely, and the secondary changes in the skin will disappear. For localised body pruritus, excellent results are obtained by painting the affected parts with crude coal tar, which is allowed to dry, and a talc powder applied. This method should not be used if sepsis is present. Further, much relief is often given by exposures to the ultra-violet rays of the mercury-vapour lamp.

C.—DISORDERS OF CIRCULATION

Only certain circulatory disorders have any bearing on skin diseases, if we do not include those disturbances associated with inflammation. Those which are referred to below usually come to the dermatologist on account of secondary changes produced in the skin.

ACROCYANOSIS

Synonym.—Chilblain Circulation.

This is a condition most frequently met with in young women, though by no means confined to them, characterised by persistent blueness of the extremities, including the hands and feet, the nose and the ears. It includes the condition known as erythrocyanosis crurum puellarium frigida, which is fully described on p. 1079.

CHRONIC VASCULAR STASIS OF THE LOWER LIMBS

This condition occurs in almost all individuals approaching middle life, and progresses with age. In individuals suffering from varicose veins it commences earlier. As a rule no special symptoms are produced, but if the skin of the lower part of the leg is damaged—and it is particularly prone to injury—it does not heal well, and there is a great tendency for a dermatitis to be set up. In some cases, however, the venous congestion causes itching, and if the skin in this region is scratched a moist dermatitis is liable to arise, which becomes septic, and healing does not readily occur. In these ways we have the well-known “eczema of the leg” produced. These cases frequently go on to ulceration, and the familiar chronic *varicose ulcer* of the leg is the result.

ROSACEA (ACNE ROSACEA)

Definition.—This is a chronic vascular congestion of the nose and central part of the face, resulting from dyspepsia and other internal conditions, and followed by secondary inflammatory changes in the skin.

Ætiology and Pathology.—The disease is common in both sexes, but rather more so in women. It begins usually after 30, but is occasionally

seen before that age. It is generally associated with dyspepsia, usually of the flatulent type, though in many cases there is no very obvious gastric disorder. Cases have been recorded in which complete achylia gastrica was present, or in which there was considerable diminution in the hydrochloric acid in the gastric juice. Uterine disturbance and the menopause are responsible for some cases. Alcohol and strong tea drinking are potent causes.

The mechanism of this vascular dilatation is not quite clear. It is generally assumed that some toxic substance is absorbed, and acts on the vasomotor system; but it is more probably a neurosis. The follicular lesions are the result of the congestion and of the increased sebaceous secretion which the hyperemia causes, as well as of increased activity of the skin cocci.

Symptoms.—The early symptoms are either those of transitory flushing of the face, or the nose gradually becomes red. Examination shows the presence of dilated vessels on the alæ of the nose. Later the congestion becomes more marked and not only affects the nose but the adjoining parts of the cheeks, the chin and the centre of the forehead. The redness may be persistent or remittent; it is worse after meals. There is usually an increase in sebaceous secretion so that the skin becomes abnormally greasy. Scattered red papules now appear at the follicular openings, and often a bead of pus is seen in them, but no sebaceous plug or comedo. This is the typical "acne" rosacea. If the skin is very dry, this papular rash may be absent; but the whole affected area may become dry and scaly, especially if exposed to the weather, showing that the congestion renders the skin more susceptible to mild external irritants. In other cases, these inflammatory conditions are absent; but the vessels become very dilated, and much disfigurement results. In the most severe cases there occurs an overgrowth of skin and subcutaneous tissue which converts the nose into a lobulated tumour—*rhinophyma*. A number of cases show a persistent type of conjunctivitis, sometimes associated with a keratitis and corneal ulceration. The severity of the eye symptoms does not, however, appear to correspond with that of the skin lesions.

The patients complain of few symptoms except dyspepsia and flushing of the face; but the unsightliness of the condition brings them for relief.

Diagnosis.—The "acne" variety must be distinguished from acne vulgaris, by the limitation of the lesions on the centre of the face, by the underlying congestion and vascular dilatation, and by the absence of the comedo. The age is also a help, as acne vulgaris is commonest between 15 to 30. It must be remembered that sometimes the two conditions occur together. In dry "eczemas" of the face, the possibility of an underlying rosacea should not be overlooked.

Treatment.—The cause must first be dealt with. A fractional test meal will give useful information as to digestive function. Easily digested food, with a minimum of carbohydrates and green vegetables, should be ordered, little or no fluid should be taken with meals, and alcohol and strong infusions forbidden. Sod. bicarb. grs. 15 to 20 with a bitter three times a day after food is of great help. Dilute hydrochloric acid, min. 20 thrice daily, may be given in achlorhydric cases. The non-dyspeptic cases often do

well on bromides and belladonna. Ichthammol grs. 3 to 5 (in capsules), or menthol, gr. 1, t.d.s., is often useful. The bowels should be regulated.

Local treatment should be sedative in the main. In the acneiform cases, calamine lotion with liquor calcis sulphuratæ (1 in 8) applied two or three times a day should suffice. In the dryer forms, ung. aquosum (B.P.) should be applied night and morning. When the veins are much dilated and unpleasantly prominent they may be destroyed by electrolysis or a fine pointed cautery. In the cases with much hypertrophy it may be advisable to remove some of the overgrown tissue with a knife. This can be done without leaving much scarring.

Of the other disorders of circulation which occasionally come under the notice of the dermatologist may be mentioned *Raynaud's disease* and *erythromelalgia*; but these are dealt with in other sections of this work (see pp. 1075, 1078).

A. M. H. GRAY.

IV. INFLAMMATIONS OF THE SKIN

Having dealt with some of those disorders which predispose to inflammatory changes, it is now possible to consider the Inflammatory Diseases of the Skin. These may be divided roughly into two great classes: (1) The superficial inflammatory dermatoses, due mainly to irritants applied externally; and (2) the deep inflammatory dermatoses, due mainly to toxic substances circulating in the blood. This division is not quite so definite as one might suppose, but it is a good basis on which to work. There are, however, a certain number of inflammatory dermatoses which cannot easily be placed in either group; these will have to be considered separately.

A.—THE SUPERFICIAL INFLAMMATORY DERMATOSES

These are produced as a rule by the application of external irritants to the skin, but there are a certain number of cases in which the external irritant cannot be traced, and in which the general symptoms suggest an internal toxin. External irritants may also cause deep-seated inflammatory dermatoses, but only when they are introduced through the epidermis; thus, the puncture of the hairs of the nettle may produce an urticaria, the infection of a crack an erysipelas, a syphilitic chancre or a patch of lupus vulgaris. Nevertheless, the general rule is that a superficially applied irritant produces a superficial dermatosis.

External irritants may be classified into the following groups: (a) chemical; (b) heat and cold; (c) actinic; (d) bacterial; and (e) mechanical. This order is chosen because the clinical types can best be explained in this way. The reaction of the skin to these different irritants is generally of the catarrhal type, which is known as "eczema." This term has, therefore, been

used freely to label lesions, but, as will be explained later, is used more rigidly in describing cases.

Ætiology and Pathology.—Chemical irritants applied to the skin may cause immediate, or primary, local necrosis, but only those reactions of the skin to irritants which do not cause local death of the tissues are dealt with in this section. Secondary local necrosis does, however, sometimes result from such reactions, as in the case of chronic leg ulcer following a varicose "eczema."

Chemical irritants applied to the skin produce different forms of reaction according to the intensity of the irritant. The reactions also vary considerably in degree according to the sensitiveness or susceptibility of the subject to varying irritants.

Recently a great deal of attention has been directed to the subject of "sensitiveness," or "allergy," of the skin. It is recognised that sensitivity may be congenital or acquired, and also that it may be specific for certain substances, or more or less general. For instance, certain individuals are congenitally sensitive to the "*primula obconica*," or Chinese primrose, and whenever they come in contact with this plant a dermatitis will result. On the other hand, persons who are not sensitive to this plant can be made so by rubbing the leaf into scarifications made in the skin, or by injecting into the skin, in appropriate doses, the active principle which has been isolated by Bloch. These are examples of congenital and acquired specific sensitiveness respectively.

General sensitiveness is less well defined, but again may be congenital or acquired. In this connection, congenital peculiarities of the skin, such as xeroderma, have to be considered.

If an intense irritant, such as a mustard plaster, is applied to the skin, the whole area to which it is applied becomes intensely red, owing to congestion of the papillary vessels; the epidermis becomes œdematous, and if the action is prolonged, small vesicles or even large bullæ develop under the horny layer. Lesions of this type are spoken of as *erythematous eczema*.

If a milder irritant is applied it may only attack the follicles, which are the most vulnerable part of the protective mechanism of the skin. In this case small follicular papules are produced, which in the more acute cases are surmounted by a small vesicle. These papules tend to group together on an erythematous base forming circumscribed patches. These are the *papular* and *vesiculo-papular eczemas*. With some irritants—croton oil, for example—follicular pustulation may also occur. The pus in this case is sterile, and the lesions dry up when the irritant is removed. This constitutes *pustular eczema*.

Lastly, there is a type in which the primary lesion appears to be produced by cracking of the horny layer. It is seen, for instance, on the face in children who dribble, and on the hands of those who use soap and water to excess. Under these conditions the horny layer becomes sodden with water, and this takes place more readily if alkalis, such as soap, are present. Then the skin dries quickly, especially when exposed to the wind, hot sun or a fire, and the horny cells tend to separate from one another, exposing the delicate mucous layer. In this type scaling first appears, followed rapidly by erythema. This is one form of *squamous* or *erythematous-squamous eczema*.

All these primary reactions are liable to undergo secondary changes.

Thus, fluid may exude from the surface from rupture of the vesicles and bullæ, producing a weeping eruption (*eczema rubrum*). Or, in the drier types, scaling may occur from irregular formation of the horny layer (this is the secondary type of *squamous eczema*). The moist cases may become infected with pus organisms, and the exudate may dry in the form of crusts (*eczema crustosum*). Thickening of the horny layer may occur, especially when the palms and soles are attacked, and this leads to cracking in the deeper folds of the skin (*eczema rimosum*). Occasionally in the lower extremities lymphatic obstruction and an overgrowth of the epidermis is produced (*eczema verrucosum*), or even elephantiasis may occur.

In order to classify these eczematous lesions a little more usefully it is advisable not to speak of a superficial inflammation produced by a known external irritant as an "eczema," but to call it a "dermatitis," qualified by the name of the irritant which causes it, as, for example, "formalin dermatitis." To use the word dermatitis without qualification is not so informing as to use the word "eczema."

For a large group of cases in which the external irritant cannot be traced, or in which it is one of those mild irritants to which every one is exposed, such as the air, or the friction of clothes, and also for certain cases in which the lesions are disseminated over the body, the term "eczema" is still used for want of further ætiological knowledge.

DERMATITIS FROM CHEMICAL IRRITANTS

These irritants are so numerous that it is impossible in a work such as this to attempt to give a complete description of them. They may, however, roughly be divided into the following classes: (1) Due to animal poisons; (2) due to plants; (3) due to chemical agents used in medicine; (4) due to chemical agents used in trades; and (5) due to decomposition of body secretions.

1. Superficial dermatitis due to ANIMAL POISONS is rare, most of the reactions being of the deep type, as they are injected by the stings and bites of insects. Some forms of caterpillar, *e.g.* the woolly-bear, however, occasionally produce an eczematous reaction.

2. PLANTS are probably responsible for more cases than are diagnosed. The *Rhus toxicodendron* or poison ivy produces the most marked symptoms, but this plant is rarely found in this country. The symptoms are an acute erythematous dermatitis, usually with much bullous formation, attacking chiefly the exposed parts, face and hands, but also affecting the moist parts of the body, the genitals, axillæ and flexures. Japanese lacquer, which is made from one of the *Rhus* family, may also give rise to a dermatitis in susceptible individuals. The commonest plant in this country to produce a dermatitis is the *Primula obconica*, but other species of the primula may attack susceptible individuals. The lesions are similar to those mentioned above, but much less severe. Among other plants which may produce a dermatitis are *Daphne mezereum*, oleander, rue, parsnip, daffodil, and chrysanthemum, while handling certain woods, such as teak, satin-wood and ebony, may produce similar effects. In all doubtful cases of "eczema" it is well to look for the presence of one of these irritating plants.

3. Of the CHEMICAL AGENTS USED IN MEDICINE some, such as cantharides, mustard, croton oil, chrysarobin and iodine, are used to produce varying degrees of dermatitis. Others may produce it unintentionally, among these being boric acid, iodoform, sulphur, carbolic acid and perchloride of mercury. Formalin, much used in pathological laboratories, produces a damaging effect on the horny layer, followed by a squamous and fissuring dermatitis. Sulphur, used in the treatment of scabies, is a common cause of an erythematous-squamous dermatitis on the flexor aspects of the limbs, on the abdomen and back, and is associated with intense irritation. Further, surgeons, students and nurses are apt to develop a dermatitis of the hands from the use of various antiseptics.

4. TRADE DERMATITIS.—This is very common, and the lesions produced are often specific. They are very numerous, for a description of the majority of which special works should be consulted. Among the more common are the soap and water dermatitis seen in washerwomen and in those engaged in household duties. This may take the form of a dry fissuring dermatitis on the back of the hands and forearms, or a papulo-vesicular dermatitis in the same situation. Its ætiology has already been discussed. Grocers and bakers are subject to a vesicular dermatitis of the hands, from handling sugar and dough: the so-called grocer's and baker's itch. French polishers, photographers, leather workers, etc., are frequently subject to dermatitis from articles used in their trades. These affections are usually of the papulo-vesicular type.

An acute erythematous dermatitis affecting the exposed parts of the body has been seen in those engaged in making explosives, also a more acute form caused by "mustard gas." The lesions in this case closely resemble those produced by poison ivy, the same distribution being observed.

Tar and oil acne.—Tar and various oils commonly give rise to a folliculitis, with a central keratotic plug like a comedo, associated with perifollicular inflammatory papules and pustules, and found on those parts of the body which come into contact with the irritants.

Grouped comedones.—In infants who have been rubbed with camphorated oil an eruption often occurs about the chest, neck and chin. The lesions are small black follicular plugs, closely placed, and are often associated with troublesome inflammatory complications.

Hair dyes.—An acute dermatitis of the face, particularly affecting the eyelids, is seen in persons who use certain hair dyes, especially those containing *paraphenylenediamine*.

Fur dyes.—Certain cheap furs, chiefly rabbit skin dyed with some of the phenylenediamine group of dyes, are responsible for a dermatitis involving the neck and chin. There is often a considerable latent period between the first wearing of the fur and the appearance of the eruption.

5. INTERTRIGO.—Decomposition of sweat and other body discharges may set up a dermatitis, usually of the erythematous type. This is best seen in fat women who are not too cleanly in their habits, the lesions being found under the breasts, in the folds of the abdomen and groins, and on the genitalia. A similar condition is often seen about the napkin region of infants. An erythema first appears, the horny layer of the skin, becomes sodden and is removed by friction of the parts, and a raw oozing surface results.

Jacquet's erythema infantum.—In some children an eruption consisting of pea-sized papules occurs about the prominences of the buttocks, thighs and abdomen under the napkin; the depths of the folds escape. Occasionally these lesions spread beyond the napkin area and frequently they are capped by a vesicle or crust. These cases appear to be due to ammoniacal urine and must be carefully distinguished from congenital syphilis. The condition is spoken of as "infantile erythema of Jacquet."

DERMATITIS FROM HEAT AND COLD

The erythema, followed often by blistering, as a result of a burn or scald, is well known. Similar but usually milder and more transient erythema may follow exposure to cold. The application of carbon-dioxide snow to the skin for purposes of treatment is a good example of the effects of extreme cold.

ERYTHEMA AB IGNE.—Frequent exposure to the fire produces a curious reticular erythema, followed by pigmentation.

ERYTHEMA PERNIO (CHILBLAINS).—Chilblains are frequently seen after exposure to cold. They occur chiefly in children and old people, and particularly in those whose peripheral circulation is sluggish. The lesions are salmon-pink to purplish, varying in colour, which occur chiefly on the fingers, toes and the lower part of the legs, and which itch intensely. The lesions may ulcerate and produce extremely indolent sores. When the helices of the ears are attacked, as they sometimes are in old people, considerable loss of tissue may result. The treatment for this condition is the same as for "Acrocyanosis" (see p. 1079).

TRENCH FOOT.—A somewhat analogous condition was met with in the trenches in France during the War of 1914–1918, in men who had to stand for long periods in the wet and cold. The feet became swollen and painful, the skin was reddened and blistering, and even necrosis occurred. The condition took a long time to subside.

DERMATITIS FROM ACTINIC RAYS

ERYTHEMA SOLARE.—Light, whether from the direct rays of the sun or from artificial sources, produces marked inflammatory changes in the skin. The sun's rays produce first a transient erythema which subsides, leaving pigmentation; but in other cases a more persistent erythematous dermatitis occurs, associated with oedema and thickening of the skin and scaling or blistering: this is the so-called "erythema solare."

TROPICAL SKIN.—Exposure to tropical sun for many years may lead to atrophy of the skin with pigmentation and warty formation.

PRURIGO ESTIVALE.—A rather rare condition occurs in some children in which it appears that the sun's rays are an exciting cause. It consists of the appearance on the face and hands of small very itchy papules which appear during the summer and disappear in the winter. They are not always limited to exposed areas, but these regions are always the most severely attacked. The condition is a very persistent one, and any treatment, except protection from the sun's rays, has little or no effect.

HYDROA ÆSTIVALE.—This is a much rarer condition than the preceding one and occurs in persons suffering from hæmatoporphyria congenita, though not in these cases only. The lesions are blisters, which appear on the parts exposed to the sun's rays, and scars are left when the blisters disappear.

X-RAY AND RADIUM DERMATITIS.—X-rays produce much more persistent forms of dermatitis than the sun's rays. If a slight overdose of X-rays is given, an erythema develops in the course of 1 to 3 weeks, which then gradually subsides. If a larger dose is given, the erythema may come on earlier and blistering may occur, which takes weeks to get well. Finally, in the still more intense burns, necrosis of the skin, with the formation of an extremely indolent ulcer, develops. In other cases, atrophy of the skin, with telangiectases and pigmentation, occurs, which may, after many years, break down into an indolent and painful ulcer, and this may in turn become epitheliomatous.

Radium may produce a similar series of changes.

ECZEMA

A description of the more specialised superficial dermatosis produced by mechanical and bacterial irritants will be left until those eruptions for which we reserve the term "eczema" have been discussed. The diagnosis and treatment of the dermatoses dealt with in the preceding paragraphs, together with that of eczema, will be considered at the end of this section. The reason for this is because it is probable that the lesions of eczema are produced, in part at least, by chemical, thermal and actinic irritants, and that pathogenic bacteria do not play an active part. Mechanical irritants do play a secondary rôle, and irritating chemical substances produced by saprophytic organisms are also concerned, but the latter are really chemical and not bacterial irritants.

Under the term "eczema" are included—(1) Certain cases of dermatitis, probably due to chemical irritants, the identity or nature of which has not been discovered. (2) Cases in which individuals are so susceptible to irritants that they react to mild stimuli that would not ordinarily be classed as irritants, such as a slight exposure to the sun, a cold wind, the warmth of a fire, or even to the friction of the clothes. (3) Cases which, having commenced with a simple dermatitis caused by an irritant, fail to get well on its removal and the patient becomes hypersensitive, so that fresh patches are produced, either in the neighbourhood of the original lesion or in other parts of the body. In fact, another factor is present which is spoken of as "sensitiveness."

Ætiology.—Many views are held as to the causes of this "sensitiveness." First there is the possibility of an inherited susceptibility or diathesis. This may be nothing more than some congenital peculiarity of the skin of which a recognisable form is xeroderma, already discussed. Abnormalities of secretion and of circulation alter the resisting power of the skin. So, probably, do certain toxic states, such as gout and rheumatism; deficient elimination, as occurring in nephritis; chronic infections, as in pyorrhœa and tonsillar sepsis; digestive disturbances and alcoholism. Disturbances of the nervous system, such as teething in infants, uterine troubles and the neuroses, may play a part.

More important, however, is the possible absorption of toxins from a local focus of dermatitis producing a hypersensitiveness or "allergic" condition, so that a violent local reaction occurs in the skin if the secretions from the original focus are brought in contact with other areas.

Pathology.—In eczema and superficial dermatitis the anatomical changes are most marked in the epidermis and papillary layers. There is congestion of the papillary vessels, with overgrowth (acanthosis) and œdema (spongiosis) of the mucous layer, and wandering cells may be present throughout the epidermis. Ballooning of the cell of the mucous layer occurs and minute vesicles appear. The horny layer is improperly formed, the cells retaining their protoplasm and nuclei, with the result that they shrink up on reaching the surface, from evaporation of water in the protoplasm, and so scaling is produced. This pathological condition is called *parakeratosis*.

Symptoms.—The general type of lesion found in eczema has already been described (p. 1397). It now remains to describe some of the common types of case met with, and this is conveniently done by referring to the regions of the body affected.

FACE AND SCALP.—Eczema of the face of a very definite type is frequently met with in infants. It usually occurs in the first year of life, and is more frequently met with in the winter months. It begins with a red irritable spot on one or both cheeks, and spreads fairly rapidly, so as to involve the whole face and often the scalp. In the more severe cases lesions are found on other parts of the body. The initial lesion is a red swollen patch or a group of follicular papules which later develop into an eczema of a scaly or weeping type. Itching is always intense but paroxysmal, the child rubbing the parts violently with the hands or against the pillow. In the moist variety septic infection may occur and thick yellow-green crusts form on the surface. Infantile eczema is often very resistant to treatment and may last from six months to a year, even under careful treatment; it usually dies out, however, during the second year of life.

This type of eczema occurs about three times as frequently in males as in females, and is often followed later in childhood by a condition frequently known as "flexural eczema," which is dealt with below.

Another type, also seen in infants, commences on the scalp, usually as a scaly or crusted patch, which becomes eczematized, usually as a result of scratching, and tends to spread down to the forehead and face.

In slightly older children a generalised eczematous condition of similar distribution, but of a septic type, is seen in association with nasal and aural discharge. Small follicular pustules are often present, and blepharitis is common. This is really an eczematized impetigo—that is, a direct bacterial infection, and not of the same nature as the first named, which is non-bacterial in origin.

Another common form of eczema met with on the face in children consists of sharply circumscribed scaly patches, always dry and with a surface like crêpe. These patches occur in the region of the mouth and nose, and can generally be traced to dribbling, running at the nose, the habit of licking the lip or smearing the face with a wet finger, or to the use of strong soaps. Some cases, however, are of streptococcal origin (p. 1423). The condition is produced by rapid drying of a sodden horny layer as described above, and goes by the name of "pityriasis simplex."

In adult life, especially in oldish people, an acute erythematous eczema of the face is apt to develop. It usually appears quite suddenly. The whole face becomes acutely red and swollen; the œdema of the eyelids often being so great as completely to close the eyes. In severe cases blistering may occur; but usually the acute œdema subsides and scaling ensues. This stage may either clear up completely or a chronic eczema characterised by redness, thickening of the skin, and scaling may follow. These acute cases are often associated with a similar condition of the hands and forearms, in fact the parts exposed to the air are most likely to be affected, and this condition is particularly prone to occur in cold weather. In some cases also a history can be obtained of a coexisting septic dermatitis elsewhere, frequently on the leg, and in these cases it is possible that some absorption from this has rendered the patient susceptible. Once a patient has had an attack he is always liable to recurrences, and great care must be taken to avoid exposure to extremes of temperature. Cases of this type may be limited to the eyelids, and a troublesome and chronic condition develops.

Occasionally the papulo-vesicular type of eczema is met with on the face; it is not infrequent on the forehead, generally in men under the hat-band, and often occurs in those who perspire freely.

Eczema of the scalp is usually of bacterial origin. This also applies to the ears (see *Seborrhœic Dermatitis* p. 1427).

"**FLEXURAL ECZEMA.**"—This is a well-recognised condition, which occurs usually in children, but may continue into adult life, and occasionally commences after puberty. It is a very specialised condition, and has been variously named "*Besnier's prurigo*," or "*flexural prurigo*." It frequently follows facial eczema in infants, and is also frequently associated with asthma. These three conditions form a syndrome and are manifestations of an underlying congenital condition named by Czerny the "*exudative diathesis*." The lesions are those produced by friction, and vary from moist eczematous patches to patches of chronic lichenification. The areas affected are chiefly the flexures of the elbows and knees, less frequently the backs and fronts of the wrists, the back of the hands, the sides of the neck and the face. Other parts of the body may be affected, and an extensive eruption is sometimes present. The condition is essentially a curious form of pruritus, the cause of which is not yet established. Experiments made with a view to demonstrating protein sensitiveness are still inconclusive. In the majority of cases the condition tends to die out before puberty.

UPPER LIMBS.—The hands and forearms, also being exposed to the weather, are subject to eczema. The erythematous type frequently complicates that of the face, and runs a similar course.

Papulo-vesicular eczema of the backs of the hands and the forearms is very frequent. It is generally produced by external irritants, whose nature can often not be determined. The lesions usually consist of rather sharply circumscribed red patches covered with numerous vesicles which rupture and leave oozing, pitted raw areas of the size of a pin's head. Sometimes the whole patch is considerably swollen with œdema. There is a great tendency in this type for fresh patches to appear in the neighbourhood, and even on distant parts.

A chronic form is sometimes met with in the palm, chiefly along the deeper folds. It begins as an ill-defined red patch, and subsequently marked

thickening of the horny layer takes place. Owing to its inelastic nature skin cracks and deep fissures are produced, which are very painful and very chronic. This type is spoken of as *eczema rimosum*.

An acute vesicular form is also seen on the hands, chiefly on the palms and sides of the fingers, especially in people whose hands perspire freely. Owing to the thickness of the horny layer on the palm, these vesicles are very deep-seated, and appear like sago-grains in the skin. At first they do not rupture, but run together and form large blebs which can often be seen to be purulent. The attacks usually come on quite suddenly, and the feet are often attacked simultaneously; they occur chiefly in the hot weather when sweating is profuse. This condition is called *dysidrosis* or *cheiropompholyx*, and was originally thought to be due to obstruction of the sweat ducts, with the formation of retention cysts. This is now known not to be the case, and that the vesicles are produced by an inflammatory exudate. It is probable that the skin is made sodden by excessive sweating, and this renders it susceptible to the attack of some external irritant. A similar condition has been observed from handling aurantia, a substance used in explosives, which shows that the condition may be produced by an external irritant. A number of these cases are due to fungus infection, the so-called "eczematoid ring-worms" of the hands and feet (see p. 1437).

TRUNK.—Eczema on the trunk is nearly always secondary to patches starting elsewhere, if seborrhœic dermatitis and dermatitis due to irritants such as sulphur are excluded. There is, however, one type to which reference should be made. In people who sweat much, and especially in infants, an eruption of small vesicles, each surrounded by an inflammatory zone, sometimes appears on the trunk. The lesions appear to be formed around the sweat duct openings. This condition is called *miliaria rubra*, or *prickly heat*, and is probably of the same nature as the vesicular eczema of the hands, the mouths of the sweat follicles being softened by the sweat and some irritant, possibly a bacterial irritant, causing an inflammation.

The nipples are sometimes the seat of an eczema; but this is nearly always of external origin, either from careless treatment during suckling, or from injury from stays.

GENITALS AND ANUS.—The moist areas of the genital and anal regions are liable to be attacked. These are not infrequently secondary to a pruritus, a traumatic dermatitis being produced. On the anus and vulva, thickening of the skin, called *lichenification*, is most common, and has been already mentioned. The scrotum is occasionally the seat of an erythematous dermatitis very distressing to the patient, and very intractable. A considerable number of cases of eczema in the genito-crural region are due to infection with fungi, or yeast-like organisms, and in order to exclude these a careful microscopic examination of the scales should be made (see p. 1436).

LOWER LIMBS.—A special form of eczema is very common on the lower part of the legs. It is associated with chronic vascular stasis. It is generally met with in middle-aged or old people, but is frequently seen in younger persons who suffer from varicose veins, hence its designation *varicose eczema*. It begins either from an infected abrasion which does not heal, or from scratching an itchy leg. Once started the inflammation spreads, as the condition of the skin does not favour resolution. The extension is often due to ill-devised dressings which further lower the resisting power of the skin and

favour the retention of discharges. An extensive weeping or crusted dermatitis, therefore, follows, and this is rarely confined to the leg on which it starts, the other soon becoming infected, probably from contact in bed. Owing to the poorly nourished condition of the skin, ulceration is very prone to occur, and thus the chronic varicose ulcer, so familiar to the out-patient department of any hospital, develops. These cases are particularly prone to spread to other parts of the body by the mechanism referred to above.

The feet are subject to the same type of acute vesicular eczema as the hands.

THE NAILS.—The matrix of the nails may be involved in an eczematous process affecting the hands and feet, and may either show a marked irregularity of growth, with roughening of the surface of the nail, or the nail-plate may be pushed up from the nail-bed by parakeratosis beneath.

Diagnosis.—Keeping in view the types of eczema already described, the diagnosis of the lesions should present little difficulty; to determine the cause, however, is not so easy. Efforts must, however, first be directed to try and discover an irritant, and if it cannot be found, or if it appears to be one of those mild irritants which do not normally produce a skin reaction, the cause of the patient's susceptibility must be investigated. These causes have already been discussed and require no repetition. As to the nature of the irritant, some help is obtained by the type of reaction and by its distribution; for instance, in an acute eczema affecting the face and hands, exposure to the wind or sun or to some irritant, as the primula, is suggested. With trades certain parts of the body tend to be especially exposed. Lesions affecting the exposed parts and the moist parts of the body suggest some strong volatile irritant, such as rhus poisoning.

Acute erythematous eczema has occasionally been mistaken for erysipelas; but the absence of a sharp line of demarcation, a slowly spreading edge and high fever, should render the diagnosis simple. Acute giant urticaria of the face is unassociated with redness or vesication. The lesions of erythema multiforme are smaller, more sharply defined, and deeper-seated.

The squamous forms have to be distinguished from seborrhœic dermatitis; this is often difficult, but the characteristic features of this latter disease will be considered later.

Ringworm of the glabrous skin tends to occur in circumscribed circular patches or rings, and the fungus can easily be found under the microscope. A special form occurring in the groins is characterised by its bilateral symmetry, its sharp spreading edge, and the presence of fungus in the scales.

Pityriasis rosea in extreme forms may lead to confusion, but the acute generalised onset, and the presence of some of the typical oval lesions, with a collarette of scales attached about a millimetre from the free edge, will usually settle the diagnosis.

Psoriasis is rarely confused, owing to its characteristic distribution on the extensor aspects of the limbs, its usual sharply defined patches, and the dry silvery scaling, seen even in the smallest papules. A few cases, however, of isolated patches made up of small aggregated psoriasis papules may be very difficult to distinguish from localised patches of squamous eczema.

The moist forms have to be distinguished from impetigo contagiosa. In

this disease, however, the vesicles are larger but rarely seen, while the presence of isolated crusted lesions of varying size, with little or no inflammatory zone surrounding them, is characteristic.

The vesicular eczemas of the hands and feet may be caused by a ringworm fungus. This should always be suspected in the chronic spreading cases, and must also be looked for in the acute cases. The diagnosis is made by finding the mycelium of the fungus in the walls of the vesicles—a task not always easy. A curious type of fissuring eczema between the toes is almost invariably caused by a ringworm fungus.

The eczemas found in the course of animal parasitic affections, such as pediculosis and scabies, will be dealt with later.

Prognosis.—This is always uncertain. Most cases of dermatitis due to an external irritant applied on a single occasion get well readily when the irritant is removed. Those caused by the repeated application of the same irritant, as in trade dermatitis, are apt to be more persistent, while recurrent attacks may be extremely troublesome. Once the skin has been damaged subsequent attacks are more common and more resistant to treatment.

Those cases in which some underlying susceptibility exists are always apt to be resistant to treatment.

Treatment.—**PROPHYLACTIC.**—This depends on the search for the irritant, and its removal. The latter is not always possible in case of trades; but much can be done to insist on scrupulous cleanliness. It must not, however, be forgotten that the use of strong soaps, soda and turpentine to remove traces of a man's occupation are often the cause of the dermatitis. In cases where these substances have to be used, by washerwomen, etc., the use of a cold cream or some glycerine preparation to replace the grease of the skin will prevent a good deal of trouble.

Eczematous subjects should protect themselves from the sun, cold wind, and heat of the fire.

LOCAL TREATMENT.—This applies equally to the cases of dermatitis due to known irritants, and to those we have labelled "eczema." The main treatment in the early stages, after removing the cause, is to protect the skin and to provide soothing application to allay the inflammation. The use of soap and water will generally have to be forbidden.

In the early and acute stages lotions are most suitable, grease in any form being badly tolerated. Calamine lotion can be applied frequently and allowed to dry on the skin, the powder it contains forming a protective dressing over the surface; it is best used in the acute erythematous and papulo-vesicular form in which there is not much oozing. In the weeping cases, lead lotion applied on linen and kept moist is more suitable; it forms an insoluble albuminate of lead which acts as a protective layer. If, however, much sepsis is present, it is well first to use a mild antiseptic, baths of 1 in 4000 potassium permanganate, or lotions of 1 in 4000 perchloride of mercury, or 1 in 1000 acriflavine being very suitable. If these lotions dry the skin too much 3 per cent. glycerin may be added.

As soon as the acute stage has subsided oily preparations are better. It is well to begin with one containing a considerable percentage of water, the lin. calaminæ (B.P.C.) or linimentum calcis being the type. Ichthammol, 2 per cent., may with advantage be added in most cases, and if the itching is severe 1 per cent. to 2 per cent. phenol. Later the water can be given

up and either pure oily preparations as *lin. calaminæ co.* (B.P.C.), or ointments used. These latter are not satisfactory if there is much discharge ; but this can be checked by painting the surface once every second or third day with 2 per cent. to 3 per cent. silver nitrate in *sp. æther. nit.*

Once the chronic stage has been reached pastes are the best means of applying medicaments. Zinc paste consists of zinc oxide, 25 ; *pulv. amyl.*, 25 ; *paraff. moll.*, 50 parts, and makes a firm dressing when spread on linen or lint. It not only affords good protection, but allows a certain amount of absorption to take place.

If the chronic cases do not respond to treatment stimulating preparations are required, and can be incorporated in the zinc paste. *Ac. pyrogall.*, 1 per cent. to 2 per cent. ; coal tar, 1 per cent. to 5 per cent. ; or oil of cade, 5 per cent. to 10 per cent., are useful, and where there is thickening of the horny layer 1 per cent. to 3 per cent. *ac. salicylic.* should be added. Chronic dry cases, and even moist ones, if not septic, often do well if painted with crude coal tar which is allowed to dry on. X-rays, 120 r ($\frac{1}{3}$ of a skin unit), repeated 3 or 4 times at weekly intervals, are extremely valuable in resistant cases, and cause rapid disappearance of the lesions.

In septic cases the crusts should be removed by warm oil or starch-poultices, and a weak mercurial or flavine lotion first applied, and afterwards a zinc paste containing 3 per cent. ammoniated or yellow oxide of mercury.

The gelatine paste of Unna is very useful in the chronic eczemas of the leg, after any sepsis has been removed by antiseptic dressings. Certain chronic eczemas of the legs do well when strapped with varicosan or elastoplast bandages recommended by Dickson Wright, and this method is particularly valuable when ulcers are present.

For facial eczema of infants, 3 per cent. crude coal tar in zinc paste, spread on a mask and continuously applied, is of great value ; or the special tar paste devised by White of Boston, U.S.A., may be employed. The same paste is the most satisfactory application in cases of flexural eczema of the non-infective type.

GENERAL TREATMENT.—The patient must be examined for any conditions liable to lower his general resistance. Septic foci, such as pyorrhœa, or tonsillar sepsis, should be removed. In the more acute cases it is advisable to put the patient on milk diet, and to keep him in bed. In the less severe cases a light diet, the avoidance of alcohol, strong coffee and tea, hot and highly seasoned dishes, shell-fish, salted meats and cheese, should be prescribed. Constipation should be dealt with, while intestinal fermentation may be met by the exhibition of salol or bismuth salicylate, grs. 10 to 15 ; *ichthammol*, min. 2 to 5 ; or menthol, gr. 1 in capsules three times a day. In gouty subjects alkaline waters and colchicum are indicated.

In the infantile facial cases, the children are usually overfed and some reduction in diet is often required.

In debilitated cases cod-liver oil is of value, while arsenic and iron are helpful when anæmia is present. In acute cases *vin. antimoniale*, min. 5, t.d.s., has been much recommended.

Sleep is often disturbed, and will require sedative drugs to allay itching, and in the worst cases hypnotics : bromides are useful for the former, while for the latter sulphonal, methylsulphonal and chloral hydrate are among

the best. Morphine should be avoided, owing to the prolonged nature of the cases and to its tendency to increase itching.

Desensitisation.—As has been noted above, of recent years it has been realised more and more that many cases of eczema are dependent on the sensitiveness to specific irritant. The offending substance can sometimes be determined by the reaction produced when it is applied to the skin, or in doubtful cases a series of substances can be applied under pieces of strapping and can be introduced by puncture or scarification, all tests being carefully controlled. In the so-called “patch test,” when the offending substance is applied under strapping, a local eczematous reaction appears; when puncture or scarification is employed, a wheal is produced.

If the cause of sensitiveness is thus discovered it is possible, in some cases, to desensitise the patient by injecting intradermically an extract of the offending substance in minute and gradually increasing doses. Further, in cases where no specific substance can be determined, it has been found possible to desensitise patients by the injection of non-specific protein substances.

A method much in vogue at the present time is to withdraw 5 to 20 c.c. of blood from a vein of the patient and to inject either the whole blood or the serum from it into the gluteal muscles. Another method is to inject 5 to 10 c.c. of sterile milk on several occasions, at 2 to 3 days' interval, intramuscularly. Peptone is also used by some, and may be given either intravenously or intramuscularly.

DERMATITIS FROM MECHANICAL IRRITANTS

Acute dermatitis due to mechanical irritation is best seen in the redness and blisters found on the hands after rowing or on the seat after riding, in those unused to these exercises. The chronic form shows itself as a thickening of the horny layer as seen in the callosities on the hands and feet. The form of dermatitis of mechanical origin, however, which requires special attention here is that produced by the fingers and finger-nails.

1. SCRATCH ERUPTION

Constant friction applied to a localised area produces changes in the skin of a characteristic type. The skin becomes thickened and loses its elasticity; the folds and lines are much exaggerated, and the angular areas of skin intervening become prominent and shiny, resembling the papules of lichen planus; the colour may be the same as the normal skin, or red, but generally purplish, and sometimes the surface is finely scaly or warty. In old-standing cases much brown pigmentation may be present. This condition is spoken of as “*lichenification*,” and is seen at its best in localised pruritus, already described on p. 1403.

When general irritation is present the scratch lesions are more diffuse. The finger passing over the skin causes contraction of the *arrectores pili* muscles and the follicles are erected; the next sweep of the finger-nail scrapes the top off the erected follicle and a spot of blood appears, which dries as a blood-stained crust. In bad cases, linear excoriations are produced,

consisting of a line of blood-stained crusts. If sepsis supervenes, typical impetigo contagiosa lesions are produced, and these are particularly common in children; in other cases "eczematisation" occurs—that is, the inflamed papules group together to form a patch or patches, which may be dry and scaly or may weep. Patches of lichenification may also be found mixed with other scratch lesions, while in the most severe cases, ecthymatous lesions, boils and linear ulcers may occur.

2. CALLOSITIES AND CORNS

These are localised overgrowths of the horny layer, the result of local mechanical irritation. A corn differs from a callosity in that the central portion shows a much greater degree of overgrowth than the periphery, and forms an inverted horny cone which presses on the sensitive dermis, producing much pain. A corn may develop from a callosity, but frequently arises independently.

Symptoms.—*Callosities* are seen as a painless thickening of the horny layer over the ball of the foot and on the palms of the hands, in the latter situation especially in manual workers. They may also occur in other situations.

Corns may be of two kinds—(1) the hard and (2) the soft. The former are painful, horny elevations, chiefly seen on the feet, and especially in people who wear badly fitting boots. The common sites are on the dorsal surface of the little toes and on the plantar surface of the great toe and over the head of the first metatarsal bone. If the surface layers are removed with a razor a central "core," often stained black or dark brown from hæmorrhage, will be seen.

Soft corns are found on the lateral aspects of the toes in the interdigital spaces. They are usually lentil-sized raised swellings, covered with sodden epidermis, and intensely painful. Soft corns are invariably found associated with interdigital ringworm.

Treatment.—*Callosities* require no treatment. The principal point in the treatment of corns is to remove injurious pressure; this can be done firstly by fitting suitable boots, and secondly by taking pressure off the corn by wearing a ring of spongiopiline around it. The surface horny layer should be pared down with a sharp knife or razor and 10 per cent. salicylic acid plaster applied or salicylic acid collodion painted on, the softened horny layer being removed daily. Soft corns are treated as for interdigital ringworm (see p. 1438).

DERMATITIS ARTEFACTA

This is the name given to self-inflicted lesions of the skin. These are usually found in hysterical individuals, who produce them in order to induce sympathy, or in persons who are endeavouring to exact compensation or to avoid some unpleasant duty.

Symptoms.—The lesions are produced by various means, such as friction, the application of strong acids, or alkalis, or of blistering fluid, by heat or by the aid of some sharp instrument. All stages from simple erythema to actual destruction of the skin may occur. They may be single or multiple,

but are found on parts of the body easily accessible to the hands, and especially to the right hand (in left-handed people to the left hand). The lesions are very characteristic, especially those in which a liquid agent has been used. They have very sharp edges and the outline is angular, unlike that seen in any ordinary skin eruption; and not infrequently irregular patches near the main lesions have the appearance of having been produced by a spilt liquid. In addition, it may be noted that in the case of the malingerer the artefact may simply consist of keeping open an already existing lesion.

In hysterical cases anæsthesia of the palate has been frequently noted.

Treatment.—For effective cure the patient must be kept under observation, and caught in the act of producing the lesions. This may put a stop to further activities. Otherwise, occlusive dressing and mental treatment are required.

DERMATITIS FROM BACTERIAL IRRITANTS

Many different organisms are capable of producing dermatitis of external origin, and the eruptions produced are usually characteristic of the organism causing them. These will be described under the organisms concerned.

1. PYOGENIC INFECTIONS

It is not always possible on clinical examination to determine whether a given lesion is produced by the streptococcus or the staphylococcus. It used to be held that the superficial infective vesicular lesions were due to the streptococcus, while the follicular pustular lesions were of staphylococcal origin. While this appears to be true for the latter, it is now recognised that certain vesicular lesions may be of staphylococcal origin.

(a) IMPETIGO CONTAGIOSA

Symptoms.—This is an affection chiefly seen in children. It affects mainly the exposed parts, such as the face and hands. The initial lesion is a small pea-sized clear vesicle, which, owing to its superficial position between the horny and mucous layers, has an extremely thin wall and ruptures very easily. Before rupture, however, the fluid often becomes turbid, and if cultured in this condition contains both streptococci and staphylococci. If cultured, however, in the very early stages, pure growths of streptococci may usually be obtained. Once ruptured, fluid exudes freely from the base of the blister and dries as a crust. The crusts vary in thickness and character according to the amount of secondary infection, being thin and amber-coloured if little secondary infection is present, but thick and greenish if it is considerable. Usually the lesions are numerous; they are asymmetrical and obviously spread by local inoculation.

When the lesions occur in folds, such as at the angles of the mouth or nose and behind the ears, a troublesome fissure is likely to form, and generally crusting is absent, the fissure being surrounded by a moist, sodden, red area.

The disease is very contagious and children inoculate one another freely, any slight abrasion being sufficient to allow the entrance of the infecting

organism. One particularly common cause is pediculosis capitis, and in this case the scalp is usually first affected. In all cases of impetigo of the scalp or back of the neck, search should be made for pediculi.

Occasionally the blisters do not rupture early, but spread centrifugally, flattening down in the centre as they progress, and leaving a ring-like bullous margin (*impetigo circinata*). In other cases a large number of bullous lesions appear very rapidly, with little or no crust formation (*impetigo bullosa*). *Staphylococcus aureus* can usually be grown in pure culture from cases of these types.

Any of these varieties may occur in adults, but the crusted form has generally smaller crusts than in children. One of the most frequent areas to be affected in adults is the beard region, and impetigo contagiosa is one of the forms of so-called "barber's rash."

Diagnosis.—This is usually easy. The presence of scattered crusts, with little or no surrounding erythema, and the occasional small, very thin-walled blister, and an asymmetrical distribution chiefly on the exposed parts, is unlike any other condition.

Treatment.—In most cases the treatment is easy. The crusts should be removed by bathing in warm water, by fomentations or by warm olive-oil compresses, or in bad cases by starch poultices, and the raw surface covered with 2.5 per cent. ammoniated mercury ointment. In most cases it is advisable to incorporate the ammoniated mercury in Lassar's paste and spread this on lint and tie it on. The acutely spreading bullous form is treated in the first instance by pricking the blisters and dressing with 1 in 1000 acriflavine lotion. In very resistant cases, injections of mixed strepto- and staphylococcal vaccine have proved useful, but are rarely required.

(b) PEMPHIGUS NEONATORUM

Synonym.—*Impetigo Neonatorum*.

This is a form of bullous impetigo seen in newly-born infants and is characterised by the presence of varying-sized blisters on the skin.

Ætiology and Pathology.—*Pemphigus neonatorum* has precisely the same cause as *impetigo contagiosa*, but produces its characteristic features on account of the ease with which the horny layer separates from the underlying mucous layer in small infants. Infection is usually conveyed on the fingers of the mother or nurse. Pure cultures of *Staphylococcus aureus* can usually be obtained from the bullæ in their early stages.

Symptoms.—The eruption usually appears in the first few days of life. A clear blister appears, which rapidly increases in size, and others soon occur in the neighbourhood. There is little or no tendency to crust formation, though the blisters frequently rupture, the raw surface being protected by the loose blister wall which lies over it. Blisters vary in size from a pea up to a florin or larger, and in severe cases may be very numerous, covering practically the whole surface of the body. The lesions may commence on any part of the body, but are frequent about the napkin area. In the most severe forms the horny layer is so rapidly separated over large areas of the body that blister formation is not an obvious feature. This variety is known as *dermatitis exfoliativa infantum* or "Ritter's disease," and ends fatally in a large proportion of cases.

Diagnosis.—The pemphigoid syphilide must be distinguished from pemphigus neonatorum. In the former condition the eruption is symmetrical, is chiefly found on the prominences of the buttocks, on the palms and soles; other symptoms of syphilis are present, such as wasting, snuffles, fissures at the angles of the mouth, and other syphilitic skin eruptions. A Wassermann reaction will in doubtful cases settle the diagnosis.

Prognosis.—Mild cases respond rapidly to treatment, but in the more rapidly spreading cases the prognosis is always grave.

Treatment.—The bullæ should be opened and their contents absorbed with cotton-wool. Strips of lint soaked in 1 in 1000 acriflavine lotion should be applied, and changed three times a day or more often if necessary. It is important that the blister edge should be removed by forceps, so that the lotion may act on the spreading edge of the lesions. The child should be well wrapped up to prevent loss of heat.

(c) ECTHYMA

In this condition local gangrene of the skin occurs and an ulcer, surrounded by a deep inflammatory zone and covered by a crust, is produced. The lesions are not always of pyogenic origin, but may be brought about in various ways; but as they have some resemblance to impetigo contagiosa, it will be well to describe them here.

Ætiology and Pathology.—The type seen in children is often of streptococcal origin and begins as an impetigo. Scratching or a debilitated condition of the patient allows of a more violent reaction, and necrosis occurs. The frequency with which ecthyma is associated with urticaria papulosa, scabies and pediculosis points to trauma as an ætiological factor. The large round adult type, referred to below, is nearly always preceded by a boil, which is a staphylococcal infection, while the linear type can be shown to be produced by violent scratching, to which is added secondary pus infection.

Symptoms.—All varieties are most often seen on the legs and buttocks. The lesions are usually discrete and few in number, but there are exceptions. They have the appearance of impetigo contagiosa lesions, but there is a wide congested area around the crusts, and these latter are not "stuck on" but firmly fixed. On removal an ulcer the size of the crust is found. This type is usually found in children. Another variety is seen in adults especially, in association with pediculosis vestimentorum, and was seen very frequently during the War of 1914–1918. Two types are seen: the large circular type, which has the characters of those mentioned above, but the individual lesions are larger, and the linear or gutter-shaped type, in which long ulcers, often 2 or 3 inches in length and covered with a thick crust, are present.

Diagnosis.—This has to be made from the ecthymatous syphilide, usually a late tertiary manifestation. In this condition there is a tendency to grouping of the lesions, and they are of a more chronic type. Other syphilitic manifestations, a positive Wassermann reaction, and rapid response to anti-syphilitic remedies will settle the diagnosis.

Treatment.—Local treatment is similar to that of impetigo contagiosa. The crusts should be removed by baths or starch poultices, and Lassar's paste with 3 per cent. ammoniated mercury tied on. In the adult cases, after

the sepsis has been removed by 1 in 4000 perchloride of mercury or 1 in 1000 acriflavine dressings, Unna's paste should be applied and changed every 2 or 3 days until healing takes place. Where debility and malnutrition are present, suitable internal treatment must be resorted to, cod-liver oil and malt, and the preparations of iron, arsenic and the phosphates being most useful.

(d) PITYRIASIFORM DERMATITIS

Certain forms of circumscribed, dry, superficial dermatitis, with fine branny scales, are sometimes seen in association with impetigo contagiosa and appear to have the same origin. In fact, all stages between the two conditions can be traced. The name *impetigo pityroides* is sometimes applied to this type of case. In other cases dry scaly patches are found without any impetigo contagiosa lesions, and streptococci have been isolated from them. These cases are sometimes indistinguishable from the scaly patches which occur on the faces of children, and which are described in the section on Eczema (p. 1412), where it is suggested that moisture and soap play the chief part in their production. It would thus appear that the streptococcus may produce lesions clinically identical with those produced by these physical and chemical causes. It has been thought also that some forms of circumscribed scaly dermatitis found about the neck and trunk, and also in the flexures of the limbs, and which have in the past been loosely grouped as seborrhœic dermatitis, are probably of streptococcal origin, but further investigation is necessary in order to group them clearly.

These lesions are often resistant to treatment. They are frequently associated with a good deal of itching, and are often followed by secondary changes due to friction, namely, "lichenification" and "eczematization."

Treatment.—The early cases sometimes respond well to applications of dilute ammoniated mercury ointment; others, however, do best on ac. salicyl., grs. 15; liq. picis carbonis, min. 15; past. zinci ad 1 ounce. Once a condition of lichenification is established, the treatment should be on the lines laid down for local pruritus (p. 1403).

(e) FOLLICULAR IMPETIGO OF BOCKHART

This is the name given to a superficial pustular eruption of staphylococcal origin seen in connection with the hair follicles.

Symptoms.—The lesions consist of small beads of pus situated quite superficially at the mouths of the hair follicles, each being surrounded by a narrow red zone. Generally the hair can be seen penetrating the centre of the pustule. There is no tendency for the lesions to run into one another, each remaining quite distinct. Usually groups of them occur in localised areas, but sometimes their distribution is very extensive, cases occurring in which almost every stout hair is surrounded by a pustule. The most frequent sites are the fronts of the thighs, the legs, the genitals and the backs of the forearms. A very troublesome variety is seen on the scalp of children between the ages of 2 to 5, the infection being usually derived from a discharging ear or nose. The whole scalp is affected, and the condition is combined with a superficial septic dermatitis which affects also the face, and often spreads to other parts of the body. Ciliary blepharitis is a frequent complication.

This is the condition which was formerly described as *pustular eczema*. The majority of localised cases occur in conjunction with other forms of pyoderma; they are seen in scabies and pediculosis, and also in people suffering from boils.

Treatment.—The general health of the patient must be attended to, and all local foci of sepsis dealt with on surgical lines. A search for parasitic infestation must be made and appropriate measures adopted. In the localised cases, the pustules should be punctured, and 1 in 1000 acriflavine lotion applied; if this proves too irritating, lead or calamine lotion should be used, the accumulation of powder from the latter being bathed away daily with a weak alkaline lotion. Mercury lotions are better avoided, as they tend themselves to produce follicular pustulation.

The more extensive cases are very resistant to treatment. Shaving the affected areas, followed by the application of mild antiseptic lotions or sedative lotions and alkaline baths, is sometimes effective. Staphylococcal vaccines should be tried in addition, and in some cases stannoxyl (an oxide of tin) and injections of collosol manganese have given good results. In the pustular eczema of the scalp, the crusts should be removed with warm oil or starch poultices, and the lotions indicated above or acriflavine 1 in 1000 in liniment. calcis applied. At the same time nasal and ear discharges must be appropriately treated. These cases take a considerable time to cure, but the results repay the attention necessary.

(f) FURUNCLE

Boils or furuncles are deep-seated infections of the hair follicles with the *Staphylococcus aureus*.

Ætiology and Pathology.—The exciting cause of a boil appears to be the presence of virulent staphylococci in the hair follicles which occasion an intense reaction sufficient to cause local necrosis. This is the more liable to occur where the skin is thick, owing to the pressure exerted on the dense fibrous-tissue bundles and the consequent obstruction to the circulation. Scratching, which conveys the causative organism to the follicles and damages their orifices, predisposes to boils, as is seen by their frequent occurrence in parasitic affections. Lowering of tissue resistance, such as occurs in diabetes and in other conditions of lowered vitality, is also a predisposing cause. In other cases it is probable that a condition of allergy and hypersensitiveness to the staphylococcus is present, and this probably accounts for the constant recurrences which occur.

Symptoms.—Boils may attack any part of the body where hair follicles are present, but are most commonly seen on the neck, back and buttocks, regions where the skin is thick and exposed to pressure and friction. The lesions are usually single or few in number, but they tend to recur with great persistence, and recurrences may continue for a long period. The patients attacked are often in a low state of health. Boils are particularly liable to occur in diabetics, and the urine of patients should always be examined for sugar. A boil commences as a deep, tender infiltration, which rapidly increases in size so as to form a painful red swelling, up to an inch in diameter, which projects above the surface of the skin. Later, a small pustule appears in the centre of the swelling and this eventually bursts, exuding a small quantity of pus, which relieves the pain. Later still, a small

slough separates from the centre of the swelling, and when this has come away the boil heals, leaving a pitted scar. Some boils, however, subside without bursting. Sometimes the earliest lesion is a superficial pustule, which is followed by the rapid formation of an inflammatory zone. Later, infiltration of the deeper tissues follows, and a slough forms and is discharged, as in the first-mentioned type.

Treatment.—The general condition of the patient must first be dealt with on general lines. Certain internal remedies, such as Vitamin A, yeast and calcium sulphide, have occasionally proved of value. Vaccines and toxoid of *Staphylococcus aureus* produce results in some cases, but cannot be looked on as a specific. Injection of collosol manganese, or injection of manganese butyrate, suggested by McDonagh, beginning with 0.5 c.c. and repeated twice a week, in slightly increasing doses, for 3 to 4 injections, produces dramatic results in certain cases; others, however, are completely resistant. Stannoxyl, given by the mouth 2 to 3 tablets t.d.s., has also been claimed to produce excellent results.

Local measures are of great importance. Before the boils have ruptured, applications of kaolin poultice (antiphlogistine) are helpful in relieving pain and causing absorption of the exudate. Short-wave diathermy and small doses of X-rays may also produce a similar result. After rupture, dressings of 1 in 4000 perchloride of mercury, or pasta magnesiæ sulphatis (B.P.C.) are indicated. Boric acid fomentations should be avoided, as they spread the infection. Early or deep incision into boils is better avoided as it tends to spread the infection.

(g) CARBUNCLE

A carbuncle is a boil or group of boils in which the subcutaneous tissue has become involved in the infective and necrotic process.

Symptoms.—Usually only a single lesion is present. It may at the commencement appear like an ordinary boil, but the spread is rapid, and soon a large, red, indurated, painful area is produced. The lesion may attain a diameter of many inches. After a few days numerous points of pus appear on the surface of the swelling, and these burst and exude pus. The bridges of tissue between these openings may subsequently break down and reveal a large slough, which may take several weeks to separate if not removed by surgical means. Fever and other constitutional symptoms are generally present.

Treatment.—The general treatment is the same as for boils. Local surgical treatment may be required, and consists either of complete excision of the carbuncle, with the surrounding inflammatory tissue, or of making a crucial incision and removing the slough, the open wound being packed with bismuth-iodoform-paraffin paste or an appropriate antiseptic dressing.

(h) SYCOSIS BARBÆ

This is a staphylococcal infection of the hair follicles of the beard region, and is one of the three forms of "barber's rash," the other two being impetigo contagiosa of the beard region and ringworm of the beard.

Ætiology and Pathology.—The disease is produced by inoculation of staphylococci into the hair follicles in the beard area, either primarily or as a secondary infection to an impetigo contagiosa. Many cases doubtless start from infection conveyed in the barber's shop, but a considerable number also occur in patients who shave themselves. Scratches from the razor subsequently inoculated by the patient's finger are probably as common as direct infection from a barber's brush or razor.

Symptoms.—The disease usually begins at one spot in the beard or moustache area by the formation of pustules around the hairs. These pustules tend to occur in groups, and become surrounded by an inflammatory zone. As the deeper parts of the follicles become infected, nodules form and the whole affected area becomes swollen and œdematous. Pus discharging from the ruptured pustules dries and forms crusts. Subsequently some of the hairs loosen and can be pulled out without pain. Each hair on removal is seen to be surrounded by a swollen and transparent root-sheath, and often a bead of pus escapes from the follicle. The disease is progressive, and eventually the whole beard and moustache area, and not infrequently the eyebrows and eyelashes, may be attacked. It tends to become chronic, and in old-standing cases a large number of the hairs are lost, leaving a smooth, red, atrophied patch, not unlike lupus vulgaris, to which the name *lupoid sycosis* has been given. The presence of some follicular pustules and the absence of lupus nodules are, however, sufficient to separate the two conditions.

Diagnosis.—In addition to the diagnosis from lupus vulgaris just mentioned, sycosis has to be differentiated from impetigo contagiosa, and from ringworm. From the former the diagnosis is made by the involvement of the deeper structures of the skin and the presence of pus in the hair follicles; from ringworm by the absence of fungus in the scales and hairs (see *Tinea barbæ*, p. 1436).

Treatment.—The acute cases should be treated much in the same way as other acute inflammations of the skin, without any attempt being made directly to attack the organism responsible for the disease. Frequent bathing in warm water or oil should be used to remove crusts, or boracic-starch poultices may be used. Lead lotion, or 1 in 1000 acriflavine lotion, should be applied on lint and changed frequently. When the acute stage has subsided the parts should be kept clipped short with scissors. Hairs from infected follicles should be epilated and the skin dabbed with 5 per cent. ichthammol in calamine lotion or 1 in 4000 perchloride of mercury. As an adjunct staphylococcus vaccines or toxoid may be given. The local application of a vaccine has sometimes proved useful. In some cases excellent results have been obtained by the use of "quinolor" ointment (Squibb). Epilation by X-rays has been recommended, but the results are not very satisfactory, recurrence being frequent. Small doses of X-rays are, however, helpful.

2. ANTHRAX INFECTIONS

These lesions, which resemble in some respects those produced by the staphylococcus, are dealt with elsewhere (p. 57).

3. DIPHTHERIA INFECTIONS

The lesions produced by the diphtheria bacillus are rare, but are seen sufficiently often to require notice. Diphtheritic infection of wounds is a well-known condition, but does not need to be considered here. In children suffering from diphtheria, gangrenous patches occasionally develop, chiefly on the trunk, from which cultures of the organism may be obtained, but the organism may also attack the skin of otherwise healthy persons.

Symptoms.—A single lesion usually occurs, but there may be more than one. It begins as a clear blister, like an impetigo vesicle, and ruptures very easily; on about the second day a considerable red zone is present around the original lesion, and a central slough has formed, comparable to a small burn. This condition persists for some time, if not treated, and eventually the slough separates and the spot heals. Constitutional symptoms may be present. The writer has recently seen a case of paronychia from which a pure culture of the Klebs-Loeffler bacillus was obtained.

Treatment.—Prompt injection of diphtheria antitoxin, with the application of a local antiseptic dressing, is all that is required.

4. INFECTIONS BY THE SEBORRHŒIC ORGANISMS

Three organisms are commonly found in cases of seborrhœic dermatitis, but the part each plays is not yet conclusively proved, so that it will be convenient to group the seborrhœic conditions under one heading. The organisms found are the *acne bacillus*, the *bottle bacillus* (pityrosporon of Malassez), and the *Staphylococcus epidermidis albus*. The first named is a small bacillus which is found chiefly after puberty and is present in very large numbers in the comedo of acne vulgaris. The bottle bacillus is a yeast-like organism which buds and often shows itself as a flask-shaped body, and is found most plentifully in seborrhœic dermatitis of the scalp. The white skin staphylococcus is found pretty universally over the skin.

(a) SEBORRHŒIC DERMATITIS

Under this term we include a chronic scaly condition of the scalp, formerly called seborrhœa sicca, and also certain "eczematous" lesions of the face, chest and back, and occasionally on other parts of the body, which are characterised by the presence of more or less circumscribed reddish patches covered by greasy scales.

Ætiology and Pathology.—The histological changes in the skin are those of a chronic superficial dermatitis. The three organisms mentioned above are met with in the scales in all adult cases, but the origin and spread of both seborrhœic dermatitis of the scalp and the figurate type on the body suggest that the views of Sabouraud and Whitfield, that the bottle bacillus is the chief ætiological factor, are correct. Further, the ease with which most lesions clear up under treatment by sulphur supports these views. There is no doubt that an underlying seborrhœa is the main factor in causing the activity of these organisms.

Symptoms.—Seborrhœic dermatitis of the *scalp* is the well-known

"scurfy head," and is an extremely common affection, most individuals having it to a greater or less degree. It probably begins in early infancy, and is sometimes seen as a ringed lesion on the scalp of young infants. These lesions have been shown by Whitfield to contain the bottle bacillus in large numbers. These rings may disappear spontaneously, but the infection, which has presumably been conveyed from the mother or nurse, persists and lights up again later in life in certain individuals, especially in those prone to seborrhœa (see p. 1400). In the adult the affection consists of a diffuse branny scaling on the scalp, usually unassociated with any obvious inflammation of the skin. On close examination the scales are seen to be formed around the hairs, indicating that the inflammation is perifollicular. Varying degrees of scaliness are met with; in some cases it is scarcely perceptible, in others it consists of thick, greasy masses. Symptoms are generally absent, but occasionally a good deal of irritation is present, which leads to scratching, and small crusted lesions are then found among the scales. Sometimes a more acute inflammation supervenes, and the scalp becomes red and hot, and an exudate of fluid may occur, producing crusting. In these cases the inflammation usually extends for a centimetre or so beyond the hairy margin. The persistence of scaly seborrhœic dermatitis is considered by some to be an ætiological factor in producing that form of baldness known as alopecia prematura, which is characterised by the recession of the hair from the forehead and baldness on the crown of the head. It is probable, however, that other factors, such as heredity, also play a part in this condition.

The *face* may also be affected, especially the eyebrows, forehead, nasolabial folds, beard and mastoid regions. Here the lesions are dry, reddish or pale patches, surmounted by greasy scales or crusts. On close examination it can usually be seen that the lesions are follicular in origin and that the patches are formed by the aggregation of these follicular papules. The ears may be affected, especially the retroauricular sulcus and the concha, and some forms of blepharitis appear to have a seborrhœic origin. The lesions on the face are very liable to become infected with pus organisms and become thickly crusted. An intractable scaly inflammation of the lips, *cheilitis exfoliativa*, is also considered to be of seborrhœic origin.

On the *chest and back* ringed or figurate lesions are frequently seen, but here follicular papules may occur. The centre of the sternum and the interscapular area are the common sites. Occasionally patches occur among the pubic hairs.

Some authorities include under this heading cases in which circumscribed pinkish or red circular or oval patches, covered by fine branny scales, occur on the trunk and limbs. They are resistant to treatment, especially to the remedies useful in seborrhœic dermatitis, and there is some evidence that they are forms of streptococcal dermatitis. This type is prone to attack the flexures of the limbs, chiefly the axillæ and groins, as are other streptococcal infections.

Diagnosis.—All cases of scurfy head in children should be considered to be ringworm until careful examination has excluded this cause. The presence of stumps and the demonstration of the fungus will settle the diagnosis. In body ringworm the distribution is irregular, the lesions are sharply circular, and the scaling is not greasy. Fungus can be found in the scales.

Impetigo contagiosa of the small crusted type has a close resemblance to seborrhoeic dermatitis, especially on the face. The presence of some definite impetigo vesicles and crusts, and the history of its unilateral spread, may help to clear up the diagnosis.

Pityriasis versicolor occurs in the same regions as the body form of seborrhoeic dermatitis, but it has a fawn colour and no inflammatory reaction, and the fungus can be found in the scales. Pityriasis rosea can be distinguished by the presence of oval lesions with a collarette of scales within the edge of the lesion, by its acute onset and by its symmetrical distribution.

Prognosis.—The figurate variety on the trunk can always be kept under by appropriate treatment, but frequently recurs. The face is more resistant to treatment, and when much septic infection has taken place may take a long time to cure. On the scalp constant treatment is necessary, and a permanent cure can scarcely be hoped for, as the organisms invade the follicles. With proper hygiene and appropriate treatment, however, the condition can be kept quiescent.

Treatment.—*Scalp.*—Frequent washing is necessary to remove the scales and accumulated dirt. Unless there is any acute inflammation present, men should wash the scalp two or three times a week or even daily with *sp. sapon. kalin.* (B.P.C.), or *ext. quillaie liq.* (B.P.C.); sulphur or tar soap may be used. In women the washing should be done once a week. After drying, in severe non-inflammatory cases, an ointment containing 3 per cent. each of *ac. salicylic.* and precipitated sulphur in a basis of gr. 120 soft paraffin and gr. 360 cocoa-nut oil should be rubbed into the scalp. Resorcinol, thymol, anthrasol, thiol or ammoniated mercury 3 per cent. may be used as alternatives or in various combinations. In the milder cases lotions are preferable. Resorcinol or chloral hydrate min. 60, *sp. vin. rect.* fl. oz. 1, *aquam ad* fl. oz. 8, is a useful lotion. Resorcinol should not be used in fair or white-haired patients owing to its staining properties. In the acutely inflamed cases, washing with soap should be avoided, though crusts may be bathed away with warm water. After removal of the crusts, *ichthammol* gr. 30 in 1 fl. oz. of *lin. calcis* should be applied, the hair being cut short if necessary.

On the *face*, sulphur and salicylic acid ointment may be used in the chronic cases; if, however, sepsis is present the crusts must be removed and calamine or *ichthammol* liniment applied.

On the *body*, sulphur and salicylic acid ointment is usually all that is required.

(b) ACNE VULGARIS

This condition is characterised by the presence of greasy plugs, known as comedones, in the pilo-sebaceous follicles—particularly those on the face, shoulders, chest and back—often associated with perifollicular inflammation. It is an extremely common affection in its milder forms and by no means rare in its severest types.

Ætiology and Pathology.—The disease occurs chiefly in individuals between 15 and 25 years of age, and is seen in both sexes. There can be no doubt that it is dependent on the development of the sebaceous glands which occurs at puberty, and that there is, in addition, an individual pre-

disposition, probably inborn, to develop the affection. The affected individuals suffer from seborrhœa.

If a comedo is examined it is found to consist of epithelial cells, sebaceous material, and the three organisms which are associated with seborrhœic inflammations and, in addition, a small acarus, the *demodex folliculorum*, is sometimes found. In the greater mass of the comedo, the *acne bacillus* occurs almost pure, the other organisms being found chiefly near the mouth of the follicle. Accumulated evidence seems to show that the *acne bacillus* is the chief exciting cause of the comedo, but that secondary suppuration may be due to the activity of staphylococci, though this has been denied by Sabouraud. The bottle bacillus and the *demodex* appear to play no active part in the production of the disease. The excessive oily secretion of the skin, with the patulousness of the follicles which accompanies this condition (the so-called "kerose" of Darier), offers a field for the activities of the *acne bacillus*, which flourishes in the sebaceous secretion. The actual comedo is formed by exfoliated epithelial cells—produced by an inflammatory hyperkeratosis of the follicle—mixed with sebum.

Symptoms.—The earliest lesions are the comedones or "blackheads." These are small, black spots which are seen filling the dilated orifices of the pilo-sebaceous follicles, most frequently on the face, but also in the other sites mentioned above. If pressure is exerted on a follicle, a cocoon-like plug can be squeezed out, which is of a cream colour, except for the portion which fills the mouth of the follicle, where it is black. Isolated comedones are extremely common, but when large numbers of them occur the term *acne punctata* is applied to the condition. Frequently, however, the presence of these follicular plugs predisposes to an acute perifollicular inflammation, and the comedo becomes surrounded by a red zone; later, a small pustule may occur in the centre. This lesion is generally painful. When these inflammatory lesions predominate, we speak of the case as one of *acne papulosa*, or *pustulosa*; but it must be noted that all varieties tend to be present together. In some cases the inflammation does not start superficially around the follicular orifice, but deeper, in the region of the sebaceous gland. Here we find first a deep-seated lentil- or pea-sized nodule, often painful, which gradually increases in size, reddening the skin as it pushes upwards, and then sometimes bursts at once and discharges a small quantity of turbid yellow fluid with the remains of the comedo; or it may attain the size of a filbert, and present signs very similar to those of a sebaceous cyst. Sometimes the nodules disappear without rupturing. This type is usually spoken of as *acne nodularis*, and is particularly liable to appear on the back. It is the most persistent type, and often leads to much keloidal scarring.

Diagnosis.—Rosacea, especially the acneiform type, when it occurs in young people, may sometimes be mistaken for acne vulgaris, and indeed the two conditions may occur together. In rosacea, vascular congestion is the prominent symptom; the lesions are generally localised to the centre of the forehead, the nose and central portion of the cheeks and the chin—they are painless and the comedo is absent.

Acneiform lesions produced by the internal administration of bromides and iodides may simply be an exaggeration of a pre-existing acne vulgaris; but if not, the lesions tend to be more grouped and to produce tumour-like swellings. Other eruptions characteristic of these drugs may also be present.

The acne produced by the irritation of tar and paraffin is usually localised to the forearms and lower limbs.

Prognosis.—The condition tends to die out between the ages of 20 to 30; but the nodular type may often continue till a considerably greater age. Though improvement is sometimes slow, treatment materially hastens a cure. Bad scarring is sometimes left in severe cases.

Treatment.—As the causative organism is situated deeply in the follicle, it cannot be reached by the ordinary anti-parasitic remedies. Treatment must, therefore, be directed to emptying the follicles. In the mildest cases this is best done by frequent washing with soap and hot water. This is followed by gentle squeezing massage over the affected areas in order to empty the grease from the follicles. Comedones should also be squeezed out, preferably with a comedo extractor. After this a mild sulphur preparation, such as calamine lotion containing 2 per cent. or 3 per cent. of potass. sulphurata or sublimed sulphur should be dabbed on, or if this dries the skin too much 3 per cent. precipitated sulphur in ung. aquosum may be used. Ointments, however, should be avoided as far as possible, as they tend to block up the follicles. This treatment must be persisted in for a considerable period. More drastic treatment consists in exfoliating the skin with a resorcinol paste or with the mercury-vapour lamp.

Vaccines have not given very satisfactory results, though staphylococcal or mixed acne and staphylococcal vaccines have been of some value in the pustular cases.

Very satisfactory results have been effected by means of X-rays. It must be remembered, however, that atrophy and telangiectases sometimes occur many years after treatment. It should therefore be reserved for the more resistant cases and especially those which are tending to develop scars, and should only be given by those who have special experience.

In addition to local treatment the patient's general health must be attended to, constipation rectified, and such conditions as dyspepsia, anæmia and menstrual disturbances treated. A low carbohydrate diet should be advised.

(c) ACNE VARIOLIFORMIS

Synonym.—Acne Necrotica.

An inflammatory condition of the hair follicles, accompanied by local necrosis, and leaving pitted scars resembling those seen in variola.

Ætiology and Pathology.—The disease is seen chiefly in middle-aged persons of both sexes. It is believed to be of bacterial origin, and is attributed by Sabouraud to the acne bacillus. It occurs in seborrhœic individuals.

Symptoms.—The affection occurs chiefly on the scalp and forehead, but is occasionally seen on the face, neck, chest and back. The lesions usually come out a few at a time, and the attacks may persist for long periods; but there are generally intervals of complete freedom. Often change of residence has the effect of stopping or determining an attack.

The lesions at the commencement are pinhead-sized vesicles situated at the mouth of the hair follicles. These increase in size to that of a lentil or pea. The vesicles rapidly dry up without bursting, and scabs are formed. These are seen to be depressed below the surrounding skin. When the

scabs fall off after a week or so a small punched-out scar remains. The onset is usually accompanied by a good deal of itching or burning.

Diagnosis.—The condition has to be distinguished from the scattered crusts which occur in seborrhoeic dermatitis of the scalp as a result of scratching. The diagnosis can be made by the pre-existing vesicular lesions in the case of acne varioliformis, and by the scarring left.

Treatment.—These cases are often resistant to treatment. The general health should be attended to, and often change of air is very beneficial. Local anti-seborrhoeic remedies should be applied, such as salicylic acid and sulphur ointment (3 per cent.), ammoniated mercury ointment (10 per cent.), or lotions of potass. sulphurat. and zinc sulphate.

(d) ACNE KELOID

A hypertrophic inflammatory condition occurring on the back of the neck just below the hair margin.

Ætiology and Pathology.—The disease occurs in young adult males. Very little is known of the cause. It occurs at a point where the collar rubs the back of the neck, and friction appears to play a part in its production. The condition has been studied closely by Adamson, who can find no evidence of previous comedo formation. Though he considers that it is produced by a combination of trauma and bacterial infection, he does not consider that the acne bacillus or the *Staphylococcus pyogenes* plays any part in its formation.

Symptoms.—The condition commences with small firm nodules, which gradually increase in size and eventually merge into one continuous mass, closely simulating a keloid.

Treatment.—Adamson recommends X-rays as the only satisfactory method of treatment.

DERMATITIS DUE TO FUNGI

1. RINGWORM

Ætiology and Pathology.—Tinea or ring worm is the name given to certain inflammatory affections of the skin produced by the growth in it of certain of the hyphomycetes or moulds. These fungi grow for the most part in the horny layer of the epidermis or its appendages, the hairs or nails, and by their growth produce an inflammatory reaction. The fungi which are commonly seen in this country belong to three genera—the *microsporon*, the *trichophyton* and the *epidermophyton*, the latter of which is characterised clinically by not attacking the hairs. To these must be added a fourth, which, though of the same family, is not usually included under the term "ringworm," namely *favus*, the fungus of which belongs to the genus *Achorion*. The fungus of ringworm is transmitted to man either from another human being or from certain animals, some fungi being only found in man. The types which are common in one country are not necessarily so in another, and in tropical zones a very large variety occur which are not considered here, but are dealt with fully in works on tropical medicine. The *microsporon*,

or small spored ringworm, attacks almost entirely children under the age of about 16, while the epidermophyton is not frequently seen in young children. The trichophyton, however, attacks children and adults indiscriminately. The genera can usually be distinguished without difficulty from one another both clinically and by examining the hair or scales under the microscope in liq. potassæ. The different species can, however, only be distinguished by their cultural characteristics. The same ringworm fungus grows differently on different media, and in order to compare cultures the fungus is by tacit agreement grown on what is known as Sabouraud's "proof medium," for the reason that this observer has collected and illustrated in his book, *Les Teignes*, a very large number of the known ringworm fungi. The common nomenclature of the fungi is that adopted in this work.

Ringworm is found on the scalp, where it almost universally travels along the hairs into the hair follicles, on the beard region, where the hairs are often but not always affected, or on the glabrous skin, where it usually remains confined to the surface horny layer. The nails are also sometimes attacked.

(a) RINGWORM OF THE SCALP

Ætiology and Pathology.—Ringworm of the scalp, *tinea tonsurans*, is essentially a disease of childhood, the adult scalp being so rarely attacked as to be considered a curiosity. In this country about 90 per cent. of cases of scalp ringworm are produced by the microsporon fungus, the large majority of these being produced by a human species, *Microsporon audouinii*, the rest (not more than about 5 per cent.) by the microspora of the cat, dog and sometimes other animals. Another 10 per cent. or so of cases are due to trichophyton fungi of which there are several species. The microsporon fungus first attacks the horny layer on the surface of the scalp; it reaches the hair shaft at the mouth of the follicle and grows down on and beneath the cuticle of the hair, destroying the cuticle and fibrillating the hair, and finally terminates in a fringe of mycelial processes just above the expansion of the bulb of the hair. The mycelial processes on the surface of the hair give off small round spores, which are packed so closely together that, when examined in liq. potassæ under a $\frac{1}{2}$ -inch objective, they are seen to form a thick mosaic sheath round the hair. As a result of the damage produced, the hairs first lose their elasticity and then fracture. This fracture usually takes place about $\frac{1}{2}$ -inch above the mouth of the follicle.

Symptoms.—*Microsporon ringworm.*—In the bulk of cases the disease begins with a small circular scaly patch on the scalp. Very soon the hair on the patch is noticed to be thinning. Several patches may appear simultaneously. On close examination with a lens, these circular patches are found to be covered with fine, branny scales of a greyish colour, the follicles are prominent, giving the patches a nutmeg-grater-like appearance, and numerous broken hairs are seen. At the edge some hairs may be found unbroken but bent at sharp angles, as though a sort of greenstick fracture had occurred. These hairs and the stumps are often covered with a whitish powder, which is the spore sheath referred to above.

On pulling one of the stumps with forceps the former will come away, but usually breaks off above the hair bulb, leaving the latter behind; a great deal of perseverance is necessary to remove the stump intact. The hair

thus removed and examined in liq. potass. has the appearances mentioned above, and in addition the fibrillation of the hair will be noted, especially the irregular fracture of the distal end. In old-standing cases the regular circular outline of the patches may be lost, the whole scalp having a moth-eaten appearance, and stumps being scattered irregularly over large areas. The microsporon ringworms contracted from animals have similar appearances.

Endothrix ringworm.—The fungus of this type is not contracted from animals. The clinical appearances may be similar to microsporon ringworm, but two other types are seen. In some cases no patches are present, but a general thick scurfiness of the scalp occurs. On very careful search with a lens isolated stumps may be found scattered all over the scalp. In other cases sharply defined bald patches occur, which on inspection show no stumps, but every follicular opening is filled with a small black spot. By the careful use of pointed epilation forceps, such as those devised by Whitfield, one or more of these spots may be removed, and on examination the fungus can be demonstrated. This type is called *black-dot ringworm*, and has to be distinguished from alopecia areata.

When the stumps from an endothrix ringworm are examined in liq. potass. under the microscope the spore sheath is found to be absent, the fungus being entirely inside the hair and the cuticle intact. The fungus itself consists of longitudinally running mycelial filaments, which are divided up into small square, round or oval segments, the whole having a ladder- or chain-like appearance.

Ectothrix ringworm.—The fungus which produces this type is of animal origin, and generally produces a much more inflammatory type of lesion than the other varieties. In the majority of cases suppuration occurs, the fungus itself being responsible for pus formation. These suppurating ringworms are spoken of as *kerion celsi*. The affected area is much swollen and red, and often raised considerably from the surrounding skin. The swelling is boggy to the touch, and often gives the sensation of fluctuation, which to the uninitiated suggests abscess formation. On the surface pus is seen to exude from numerous follicular openings, but broken hairs are also seen. These if examined in liq. potass. show fungus, both within and without the hair; the cuticle is destroyed and the mycelium has similar characters to the endothrix fungus, the spores being arranged in chains and not packed together, as in the microsporon type.

Diagnosis.—This is usually simple, the presence of the stumps containing fungus being diagnostic. In cases where stumps are few in number, great help can be obtained by examining the child's scalp under a mercury-vapour lamp, screened by what is known as "Wood's glass." In microsporon ringworm the affected stumps fluoresce brilliantly and can be readily seen. This method is particularly valuable in determining whether a case is cured after treatment. Fluorescence does not occur in endothrix ringworm. From favus the diagnosis is made by the presence of yellow favus cups. From seborrhœic dermatitis the diagnosis should not be difficult if it is always remembered that a scurfy head in a child must always be considered to be ringworm until this has been excluded. Great care has, however, to be taken to make a thorough search for stumps in the endothrix cases. In alopecia areata a smooth, shiny centre with, perhaps, a row of scattered stumps at the periphery of the patch is found. These stumps, however, are club-shaped, are very thin

as they enter the scalp, and when pulled out always come away with a shrunken bulb attached. No fungus can be seen on microscopic examination.

Treatment.—The cardinal fact to remember in the treatment of scalp ringworm is that up to the present no means has been discovered of killing the fungus in the hair follicles. It is, therefore, necessary to epilate the hairs in order to obtain a cure. This can be done by three methods: by X-rays; by producing sufficient inflammation in the affected areas to make the hairs fall out; or by the administration of thallium acetate internally. This second method is the way Nature cures some cases. In kerion the suppuration is sufficient to loosen the hairs, and all that is necessary is to assist this process by hot fomentations and epilation with forceps. In the ordinary microsporon type, however, the production of the necessary inflammatory reaction is not so easy. Various irritants have been used—the most satisfactory of which is croton oil. The application of this, however, requires great care, and is not suitable for ordinary out-patient practice. Probably the best application available at present is an ointment of equal parts of common salt and soft paraffin. The scalp is shaved and washed daily with soap and water, the healthy portion then smeared with a weak mercurial ointment, such as 2·5 per cent. white precipitate ointment, and the salt ointment rubbed vigorously into the ringworm patches. After a time the patches inflame, and the hairs loosen and fall out. Cure by this method in fairly localised cases takes 2 or 3 months if the treatment is vigorously and conscientiously carried out; otherwise it may take 12 or 18 months to effect a cure. X-ray treatment is generally preferable. The method used is that devised by Adamson and Kienbock, and consists in treating the scalp from five different points with an epilation dose, the points being so arranged that the whole scalp is uniformly irradiated. A modified four-area method introduced by Schreuss is gradually becoming more popular. This should cause all the hair to fall out in 3 weeks, and a complete cure should take place.

It has recently been shown, chiefly owing to the work of Buschke and his associates, that, if thallium acetate in a single dose of 8 mgrms. per kilo body weight be administered orally, the scalp hair will fall out after about 18 days, leaving the eyebrows and eyelashes unaffected. This method has now been used in a large number of cases of ringworm, and gives satisfactory results. It does, however, often produce well-marked toxic symptoms, chiefly severe joint pains and gastro-intestinal disturbance, and some fatal cases have been reported as a result of accidental overdosage. Its final beneficial results are probably not equal to those of X-rays. It should only be given to children who are perfectly healthy.

(b) RINGWORM OF THE BEARD

Symptoms.—Ringworm of the beard, *tinea barbæ*, occurs in two types: (1) the superficial, scaly type, and (2) the suppurative type. The former begins as a small scurfy patch, which spreads slowly in ring fashion and resembles the scaly type on the scalp. The hairs are usually attacked, and if removed fungus can be demonstrated in them and also in the scales. The fungus is usually of the endothrix type, and as such is transmitted from man to man. It is not infrequently caught in the barber's shop, and is one of the three forms of "barber's rash."

The suppurative type produces an irregular lumpy swelling of the affected part. The "lumps" are soft and boggy to the feel, and pus may be seen exuding from various follicular openings; the case bears a close resemblance to *kerion celsi*, but has not the same sharp circular edge, being more irregularly distributed. The hairs are attacked by the fungus, which in this case is generally of the *ectothrix* type, and is usually transmitted from animals, being frequent among grooms and cattlemen.

Diagnosis.—The scaly variety must be distinguished from seborrhœic dermatitis and the pityriasisform type of streptococcal infection. This is easily done by the presence of fungus in the hairs and scales of ringworm.

The suppurative type may be confused with the staphylococcal sycosis; but the latter never forms the tumour-like masses which are seen in ringworm, while again the presence of fungus will settle the diagnosis.

Treatment.—The same principles apply as in scalp ringworm. For the scaly type X-rays form the most certain form of treatment. Alternatively 3 per cent. salicylic and 5 per cent. benzoic acid ointment should be rubbed in daily and the hairs epilated, a few at a time, with forceps. The hair should be kept out short.

With the suppurative variety hot fomentations and epilation with forceps should be used.

(c) RINGWORM OF THE GLABROUS SKIN

This can be divided into four types. (1) *Tinea circinata*, the small ring- and disk-like patches seen about the face, neck, body and limbs; (2) *tinea cruris*, *eczema marginatum* or *dhobie itch*, seen chiefly as sheet-like patches in the inner side of the thighs, and on the perineum and scrotum; (3) the *eczematoid ringworms* of the hands and feet; and (4) the *pustular body ringworms*.

Symptoms.—1. *Tinea circinata*.—This condition may occur by itself or in combination with scalp ringworm. In the latter condition it usually occurs on the neck or face. In the microsporon cases of human origin the lesions take the form of small disks, usually not larger than a threepenny bit, which show little tendency to grow, and no tendency to clear in the centre or to form rings. The patches are of a pale pink colour, and are covered with branny scales, in which mycelial filaments can be demonstrated by examining them under a $\frac{1}{8}$ -inch objective in liq. potassæ. The other varieties of microsporon and the *ectothrix trichophyton*s show a much greater tendency to form rings and to attain a larger size. In these cases the earliest spots are similar to those described above, but as they spread the centre loses its scaliness, becomes a paler colour, and eventually the skin resumes its normal character. The spreading edge presents the same branny scaling, and often small pinhead-sized vesicles and pustules. As before, mycelial filaments can be demonstrated in the scales. Itching is often present to a greater or less degree. In rare cases these rings are very numerous, and concentric rings may form. This is well seen in some tropical varieties, such as *tinea imbricata*, where the whole body is covered with concentric ring-formations.

2. *Tinea cruris*.—This condition, also known as *dhobie itch*, is produced by the genus *Epidermophyton*. It is so named because of the commonly held view that clothes are infected by the washerman or *dhobie*. Originally a tropical type, it is now extremely common in this country, being much more

often seen in private than in hospital practice, and almost entirely in males. It is usually seen as a superficial, flat, brownish-red patch situated bilaterally on the inner surface of the thighs in their upper third. The patches usually meet on the perineum, and often involve the whole scrotum, and sometimes spread forward into the groins. The patches, which were formerly known as *eczema marginatum*, have a very sharply defined margin, which is very slightly scaly, but no vesicles are present. Scrapings from the scales show a chain-like mycelium. The patches, though usually confined to this region, are occasionally seen on the umbilicus and in the axillæ, and are frequently associated with one type of eczematoid ringworm seen between the toes. There is generally intense itching felt in the patches.

3. *Eczematoid ringworms*.—There are several varieties of this type seen. The commonest is that which occurs between the toes. It occurs first between the little and fourth toe, and is generally bilateral. The skin in the web of the toe becomes thickened, whitish and sodden, and fissuring is prone to occur. It may spread to adjoining interdigital spaces, and on the dorsum and sole of the foot. In severe cases this area becomes covered with vesicles or large blebs, which may become purulent. It is not always easy to demonstrate the fungus in the thickened skin between the toes; considerable time must be given to soaking in liq. potassæ the skin removed, and many slides may have to be made before the search is rewarded.

The most common type seen on the hands consists of rather sharply circumscribed patches of a vesicular dermatitis. They may occur on any part of the hand or fingers, and are generally single and unilateral. They spread slowly, and are itchy. The lesions are usually produced by the trichophyton fungus. The demonstration of the fungus is necessary to distinguish them from other forms of localised dermatitis. When the lesions occur on the palm much thickening of the horny layer is produced, and cracking in the deeper folds may take place.

In another form an acute dermatitis which may involve both hands and feet, is set up, as has been shown by Whitfield. The cases are clinically indistinguishable from the type of acute dermatitis known as dysidrosis or cheiropompholyx, and in all such cases a careful examination must be made for a ringworm fungus.

4. *Pustular body ringworms*.—These occur in sharply defined patches, chiefly on the limbs and neck. The patches are of dull red colour, and sharply raised from the surrounding skin; they have a soft boggy feel, and pus can be seen exuding from the follicles. The fungus in this case is usually of the ectothrix variety.

Diagnosis.—This is only difficult in the acute eczematoid varieties, when it must be distinguished from cheiropompholyx and the localised forms of dermatitis and eczema. This can only be done with certainty by demonstrating the fungus. The circinate patches have to be distinguished from seborrhœic dermatitis and the scaly streptococcal lesions, and on the face from pityriasis simplex. The presence of fungus, and the ease with which patches respond to Whitfield's ointment, as well as the tendency to ring-formation, and asymmetrical distribution, will enable a diagnosis of ringworm to be made.

Treatment.—This is simple in the flat body patches and in *tinea cruris*. The number of antiparasitic remedies is large but the most satisfactory is

fuchsin paint (Castellani). This is painted on the patches once or twice daily, and can be used in the most inflamed cases. Other preparations include ung. ac. benzoici co (B.P.C.) (Whitfield's Ointment) and liq. iodi mitis (B.P.). Most patches will clear up in a week or two with these remedies, but it is well to continue them for some days after the lesions have disappeared in order to prevent recurrence.

Eczematoid ringworm of the toes can be treated with Whitfield's ointment, the sodden epidermis being removed daily after washing with soap and water; it is well to use this intermittently for 2 weeks at a time, powdering the toes well with a bland powder during the alternate 2 weeks, in order that the keratolytic action of the salicylic acid may subside and give a better indication of the results of treatment. In resistant cases 10 per cent. chrysarobin in spirit may be used in addition. These cases are always resistant to treatment, which requires to be carried on for long periods.

The acute eczematoid ringworms of the hands are often made worse by strong parasitocides, and it is generally better to start treatment with a wet dressing of 1 in 4000 potassium permanganate, subsequently trying small areas tentatively with the preparations mentioned above. The suppurative type can also be treated with 1 in 1000 acriflavine, or 1 in 4000 perchloride of mercury dressing, and subsequently with Whitfield's ointment, if not cured by the former methods. Resistant cases respond well to X-ray treatment.

(d) RINGWORM OF THE NAILS

This is fortunately not a very common affection, but occurs with sufficient frequency to be on the look out for it. It may be caused by the *endothrix* or *ectothrix* fungus.

Symptoms.—Usually several but not all the nails are affected. The disease usually commences under the free end of the nail, and travels slowly upwards. The nail bed becomes much thickened, and the epithelium sodden, and can be scraped away. As the disease spreads the nail becomes a greenish-grey colour and separated from its bed; the growing edge can be seen as a yellowish line above the discoloured and separated nail. In other cases the nail becomes soft or brittle and breaks up, exposing the underlying sodden nail bed. Very rarely the sides and base of the nail may be primarily affected. The toe nails are frequently affected in interdigital ringworm of the feet.

Diagnosis.—The diagnosis has to be made from eczema, psoriasis and syphilis. This can only be done with certainty by finding the fungus. Portions of nail near the growing edge should be taken and soaked for some hours in liq. potassæ. The under surface is then scraped and mounted, and a search made; and this may require several preparations before the mycelium is found. Cultures can often be made direct from pieces of nail; but contamination is very frequent.

Treatment.—The nail must be removed, either surgically or by softening in strong potash and scraping it away. Afterwards one of the stronger anti-trichophytic remedies can be applied. Norman Walker recommends covering the affected nails with lint soaked in Fehling's solution and applying a rubber finger-stall for 24 hours or longer, so as to remove the nail completely. The solution must not be applied to the surrounding skin.

2. FAVUS

Favus is a disease due to the growth of a fungus allied to ringworm, belonging to the genus *Achorion*. It differs from the former in forming thick, yellow, circular cups which cause local scarring and atrophy of the hair follicles. It is a much rarer disease in this country than formerly; but cases are still occasionally seen.

Symptoms.—Favus attacks the *scalp*, the glabrous skin and the nails, and has been recorded on the mucous membranes. On the scalp it appears as a collection of pea-sized or slightly larger circular yellowish crusts standing up from the skin and having a central depression, through the centre of which the hair projects. This is the *favus cup* or scutulum. Very large areas of the scalp may be involved in the process, the whole having a honey-combed appearance. Where the disease has been cured, scars and permanent alopecia are left. In section the yellow cup is seen to be made up of masses of mycelium radiating from the centre. Favus on the *glabrous skin* shows a somewhat similar appearance, a collection of bright yellow cups forming a massive crust, the whole being surrounded by an inflammatory zone. When seen in this country the lesions are generally very few in number and on the exposed parts, but in some countries where the disease is common the whole body may be covered with great masses of favus scutula. Favus of the glabrous skin in this country is often of mouse origin, and a different species to the scalp favus. Favus of the *nails* has somewhat similar characteristics to that of ringworm of the nails.

Treatment.—The only satisfactory treatment for favus of the *scalp* is X-rays. The risks of alopecia mentioned in the treatment of ringworm need not be considered here, as alopecia will result in any case from the disease. As a preliminary to X-Ray treatment the crusts should be removed and the scalp cleaned up with appropriate antiseptic applications.

In *body* favus the scutula must be removed, and the patches treated with either Whitfield's salicylic and benzoic ointment or a 4 per cent. chrysarobin ointment.

Favus of the *nails* is treated in the same way as ringworm of those parts.

3. MONILIA INFECTION

A good deal of attention has recently been paid to lesions closely resembling those produced by the ringworm fungi, but attributable to the growth of yeast-like organisms resembling those found in thrush. The lesions are chiefly found in moist situations, such as the groins, under the breasts and between the toes. The same fungus has been found to be responsible for a sodden condition between the fingers, to which the name *erosio blastomycetica interdigitalis* had formerly been applied. It has also been found in the nail folds, producing a curious bolster-like swelling of these structures, and has also attacked the nails themselves.

The *treatment* of these conditions is similar to that employed in ringworm.

4. TRICHOPHYTIDES

Of recent years a variety of generalised eruptions have been described in association with cases of fungus affection. These have been shown to

be produced in a way analogous to that in which the tuberculides are produced in cases of tuberculosis (see p. 1463). In certain fungus affections the skin becomes sensitive to the toxin of the fungus, as can be demonstrated by intradermal injection of extracts of the fungus concerned. It is presumed that either the fungus itself or its toxins enter the circulation and that eruptions at distant sites are thus produced.

The eruptions vary considerably in type; the lichenoid variety, consisting of numbers of pinhead-sized papules scattered over the trunk and analogous to the lichenoid tuberculide, is the commonest, but eczematous, scarlatiniform, morbilliform and urticarial eruptions have been described, and also lesions resembling erythema multiforme and erythema nodosum. Many of the vesicular eruptions of the palms and soles, associated with interdigital ringworm of the feet, are believed to be trichophytides.

The eruptions are described as microsporides, trichophytides, epidermophytides, favides and levurides, according to the nature of the primary affection, the last named being associated with monilia infections.

The diagnosis rests on the presence of an existing or recently pre-existing fungus infection, together with a proved cuti-sensitiveness to the toxin of the appropriate fungus.

No special treatment is required beyond that required for the primary affection, together with palliative treatment of the lesions.

5. TINEA VERSICOLOR

This is a superficial infection of the horny layer with the *Microsporon furfur*, and is frequently seen among hospital out-patients.

Symptoms.—It usually forms very thin, greenish-yellow patches or a continuous sheet over the chest and abdomen; but may cover larger areas of the body. It is said to occur chiefly in people who wear thick woollen underclothing and perspire freely. If the patches are scraped scales can be removed, and these examined in liq. potassæ show thin mycelial threads with large round spores among them.

Treatment.—The treatment is the same as for other body ringworms, salicylic and benzoic acid ointment or a sulphurous acid lotion causing rapid cure. The underclothing should, however, be sterilised, or reinfection will occur. Precautions against over-clothing should also be taken.

6. ERYTHRASMA

This is an uncommon disease in this country, and is due to the infection of the horny layer with an extremely small fungus, the *Microsporon minutissimum*.

Symptoms.—The affection occurs as superficial, reddish-yellow patches and plaques, more or less symmetrically arranged, chiefly in the groins and axillæ.

Diagnosis.—The malady is to be distinguished chiefly from tinea cruris, and this can readily be done by noting the size of the mycelial elements under the microscope. In erythrasma they are so small as to require a $\frac{1}{12}$ -inch objective, and under it appear as small bead-like chains, with masses

of spores intermingled, while in tinea cruris chain-like mycelium can easily be seen under a $\frac{1}{8}$ -inch objective.

Treatment.—The treatment is the same as for pityriasis versicolor.

7. LEPOTHRIX

This is a not very uncommon affection of the axillary hairs in which they become surrounded with dark reddish concretions.

According to Castellani, this affection is caused by a bacillary-like fungus, *nocardia tenuis*, acting in symbiosis with a red pigment-forming coccus, *micrococcus castellanii*.

The treatment consists in dabbing the affected hairs twice daily with alcoholic formalin (2 per cent.), and rubbing in at night a 2 to 5 per cent. sulphur ointment. Calamine lotion may be used to allay any irritation caused by the treatment (Castellani).

DERMATITIS DUE TO ANIMAL PARASITES

The affections of the skin due to animal parasites are of a mixed variety, but for general purposes may be classed under the superficial dermatoses. Animal parasites produce their effects on the skin either by puncturing and injecting an irritating substance or by burrowing in the skin; but what have chiefly to be taken into consideration are the secondary effects produced by the irritation these creatures produce. In tropical countries the number of animal parasites which produce skin lesions is very large; it is proposed here, however, to consider only those seen commonly in this country.

1. BITES AND STINGS

The common flea, the bed-bug, gnats and the pediculus family are the common biting insects seen in this country, while of the stinging insect bees, wasps, hornets and ants may be mentioned. Excluding pediculi, which require more detailed description, the lesions produced by all these insects are wheals of varying size, depending on the particular insect, and also on the susceptibility of the person attacked. The lesions are familiar to all, and require no detailed description.

Treatment.—As most of these stings are due to an acid irritant, the application of weak solution of ammonia and other alkalis gives most relief. In the case of the bee the sting should be removed if still in the skin.

2. PEDICULOSIS

Three forms of pediculi attack man: the *Pediculus capitis*, the *P. vestimentorum* or *corporis*, and the *Pediculus* or *Phthirius pubis*.

The first two are merely varieties of the same species—the *Pediculus humanus linnaeus*.

PEDICULOSIS CAPITIS.—**Ætiology.**—This condition is caused by a small insect, 2.5 to 3 mm. long, with an oval body consisting of a narrow thorax and wide abdomen, to the former of which are attached six legs, each being provided with a hook-like extremity, with which it hangs on to the hairs. The head is small, oval, and provided with two antennæ, a powerful mandible

and a proboscis with which it punctures the skin in order to suck the host's blood. This variety is found among the scalp hairs, chiefly in female children of the lower classes. Pediculi breed with great rapidity, laying their eggs on the hairs. The eggs are contained in a chitinous, ovoid cell, with a movable lid or operculum, and are known as nits; they are laid from the scalp outwards, and each is stuck on to the hair by a drop of cement extruded by the female as she moves along the hair. Nits can only be removed by unthreading them from the hairs.

Symptoms.—Itching is the only symptom produced by the *P. capitis*, and this is due to an irritating substance injected by the insect when it bites. A large number of infested individuals feel no itching; they are, however, a source of danger, as they infect others. If the itching is severe, scratching follows, and this frequently causes impetigo contagiosa, which is most marked at the back of the scalp, but may spread to the vertex, eventually involving the whole scalp and matting the hair down among thick crusts. Similarly it may spread to the back of the neck and shoulders, and involve large areas of the body. Even when impetigo is absent, the presence of scratch marks on the back of the neck and shoulders is almost diagnostic of *P. capitis*.

Diagnosis.—All cases of impetigo of the scalp, especially in children, should be examined for pediculi. The diagnosis is easily made by finding the pinhead-sized, white, shiny oval bodies attached to the base of the hairs, and in bad cases the transparent little insects themselves can be seen scuttling about among the hairs.

Treatment.—The insects are easy to kill, but the nits are more resistant. The favourite method is to saturate the scalp with paraffin and tie it up for 12 hours; this has the disadvantage of being messy, and is not free from danger if the head is brought too near a naked light. Whitfield's method of saturating the hair with 1 in 40 phenol and then tying the hair up in it for half an hour is very efficacious, especially when much impetigo is present. After this, the crusts can be removed and the nits combed out, and a weak ammoniated mercury ointment applied.

PEDICULOSIS VESTIMENTORUM.—**Ætiology.**—The causative parasite has exactly the same anatomical character as the preceding, but is usually slightly larger, up to 3 to 4 mm. in length. It is not very common in civil life, being only seen in the habitués of the casual ward and the common lodging-house. In war-time, however, it becomes one of the chief causes of sick wastage of armies, being almost universal in its incidence and causing an enormous amount of skin disease.

The insect lives chiefly in the clothes, coming on to the body in order to feed; it is chiefly found, therefore, in those parts of the clothing which come into most intimate contact with the body. In civil life the *Pediculus vestimentorum* is rarely seen, but its nits may be found in the seams of the under-clothing of infected persons. Occasionally in heavily infested people nits may be found on the axillary, pubic and perineal hairs.

Symptoms.—The skin lesions in this condition are mainly those produced by scratching. Closely placed, small, red macules may occasionally be seen, the results of the insect bites, but this is unusual. The scratch eruption has a characteristic distribution and type. In civilians, it is most marked about the back of the shoulders and around the waist and upper part

of the buttocks. In soldiers, it is even better marked on the legs and about the knees, owing to wearing the puttee. The lesions in earlier cases are papules, surmounted by hæmorrhagic crusts and linear excoriations. In cases of longer standing, areas of eczematization and lichenification occur, and the skin becomes irregularly pigmented. Septic complications are not very common in civil life, but in the field are the rule. Boils and linear, gutter-shaped ulcers, described under ecthyma on p. 1432, are extremely common under these latter conditions, chiefly on the legs.

Diagnosis.—This has chiefly to be made from scabies, but the presence of the burrows and the distribution of the rash—described in detail in the article on that disease (p. 1444)—should enable a diagnosis to be made. From senile pruritus the diagnosis can only be made by finding lice or their nits.

Treatment.—Disinfection of the clothing and bedding of the infected person is all that is required, except in those who harbour nits on their hairs, in which case the latter should be cut short or shaved. Most local sanitary authorities will carry out the necessary disinfection if duly notified; the methods employed scarcely come within the scope of this work. Local lesions can afterwards be treated with sedative lotions and creams, and impetiginous lesions as already described (p. 1421).

PEDICULOSIS PUBIS.—**Ætiology.**—The *Pediculus* or *Phthirus pubis* has a different appearance from that of the above-mentioned varieties; the body is shorter, wider and almost triangular in shape. It is usually about 1·5 mm. long and about the same width, and is provided with six legs, which are more curved than in *Pediculus humanus* and are also provided with hook-like extremities. This louse can move with considerable rapidity along the hairs, but has very limited powers of movement on a flat surface. When found among the hairs it is seen clinging with its legs to two adjacent hairs. Its eggs are laid in the same manner as with other varieties.

The pubic louse is found almost exclusively in the pubic and perineal hair, but in severe cases the hair in front of the abdomen, chest and thighs may be infested, as may also the axillary hairs, the beard, the eyebrows and eyelashes. It is extremely rare on the scalp. It is usually transmitted during coitus.

Symptoms.—There are two main symptoms, itching and the presence of small bluish stains on the skin. The itching is often intense and may lead to loss of sleep, but is localised to the area attacked. Scratch lesions are not very common, doubtless owing to the protection afforded by the stout pubic hairs; they do, however, occur. The bluish stains found on the skin in regions infested by the crab-louse are now known to be produced by the bites of the insect. They are 4 to 10 mm. in diameter, not raised above the skin, and do not disappear on pressure. They are known as *maculæ cæruleæ*.

Diagnosis.—This is made by finding the louse and its nits attached to the base of the hairs.

Treatment.—The best results are obtained by clipping the hair short and rubbing in 5 per cent. betanaphthol ointment. Ung. hydrarg., phenol lotion (1 in 40), and petrol are also used, but the former of these may set up a severe dermatitis if not carefully used. On the eyelashes, the insects and their nits should be removed by forceps.

3. SCABIES

Ætiology.—Scabies is a disease caused by a spider-like, acarine parasite, the *Sarcoptes scabiei*. The acari form a large group of animal parasites which attack man and the lower animals. The parasite generally found in man (var. *hominis*) is a special variety and is not contracted from animals. Various other acari, however, which attack animals may also attack man, but they do not produce identical symptoms.

The *Sarcoptes scabiei*, commonly spoken of as the acarus, is a minute round body, just visible to the naked eye, and of white shining appearance. The body bears eight legs, which differ in the two sexes. In both sexes the two anterior pairs bear suckers; in the male the third pair bear long bristles and the fourth pair bear suckers, while in the female both hind pairs bear bristles. The female is larger than the male, and burrows in the horny layer of the skin to lay her eggs. If undisturbed the female may live for 2 to 3 weeks and lay up to about 30 eggs. The eggs are laid in the burrow and the young hatch out there, the complete cycle from egg to mature acarus being completed in about 10 days. The larvæ, however, hatch out in 3 to 3½ days.

The female acarus has certain favourite sites for burrowing, namely, the genitals, the fronts of the wrists, the web and sides of the fingers, the ulnar border of the hand, the backs of the elbows, the anterior axillary folds, the nipples in women, the umbilicus, the sides of the gluteal cleft and lower part of buttocks, the front of the knees, the ankles and the dorsum of the feet. In infants the palms and soles are also frequently affected.

Symptoms.—The eruption of scabies is of two kinds—the acarine burrows and the follicular papular eruption. The burrows occupy the sites named above. They are seen most clearly on the hands, where they usually form thin, sinuous lines, from a millimetre up to a centimetre in length and occasionally even longer. The burrow is generally easily seen, as dirt accumulates in it, but quite often it can only be recognised by a lens. The oldest part of the burrow has a splay mouth, while at the other end the small white body of the acarus, with a black spot in its fore part, can be easily seen with a lens and often with the naked eye. Frequently a clear vesicle or vesicles are seen beneath the burrow, but as a rule on the hands no redness is present unless secondary infection has occurred. When blisters are present, secondary infection is frequent, and pustular, weeping and crusted areas are produced. In other sites vesicles are not common, but a large pea-sized papule usually underlies the burrow, and the burrow itself and its acarus are not so easily seen; these lesions are frequently seen on the penis, scrotum and anterior axillary folds, and are usually diagnostic.

The follicular papular eruption is arranged in smaller or larger circles round the areas where the burrows occur. The main distribution is on the anterior aspect of the body, from the nipples to the knees, and in a semi-circle around the anterior axillary fold. The back is free, except in severe cases, down to the top of the gluteal cleft, but scratch lesions occur on the lower part of the buttocks, where ecthyma is often a complication, and on the back and inner parts of the thighs. On the limbs the eruption occupies both front and back of the forearms, up to about the centre of the arm, and

also occurs around the ankles. The lesions are first pinkish or whitish elevations of scattered hair follicles, but soon they become covered with bloodstained crusts from scratching. Linear scratch marks are rare in scabies. It is not clear what produces the scratch eruption in scabies, but the work of J. W. Munro suggests very strongly that the follicular lesions are produced by the acarine larvæ, and the distribution of the lesions adds confirmation to this view.

In old-standing cases almost the whole body may be affected, though the face and scalp are practically never attacked in adults; but in small children even these areas may suffer.

Diagnosis.—In well-marked cases no difficulty arises, as the burrows can be seen, but in treated cases the diagnosis may be very difficult and a diagnosis from pediculosis may have to be made. Also the two conditions may occur together. The distribution and character of the rash will usually settle the point, but a careful search with a lens for burrows and acari should always be made.

Treatment.—This depends more on the carrying out of detail than on the actual parasitocides used. Three things are necessary, namely, the opening of the burrows by scrubbing, the subsequent application of a parasiticide to the body, and the disinfection of clothes and bedding. The body should be soaked in a hot bath, then rubbed with soft soap, and finally scrubbed with a brush, particular attention being paid to areas where burrows occur. After this sulphur ointment is rubbed in all over the body (face and scalp excepted), and the patient again dresses, his clothes having been disinfected in the meanwhile. On the two following days the ointment is again rubbed in, but no bath given (as it tends to increase the liability to sulphur dermatitis), on the fourth day nothing is done, and on the fifth day the patient has a bath—to wash off the ointment—and puts on clean things, all dirty linen being sent to the wash. If any dermatitis from the sulphur arises, lin. calaminæ, to which 2 per cent. liq. pic. carb. is added, may be smeared on, and if there is much sepsis appropriate treatment can then be applied. Another satisfactory method is to paint the body, after the bath and before drying, with a lotion composed of benzyl benzoate, soft soap and industrial spirit, in equal parts. This is allowed to dry, and the painting repeated at once. Twenty-four hours later a cleansing bath is given. Other preparations which have been used with success are ung. potassii polysulphidi (B.P.C.) (Marcoussen's or Danish ointment), 5 per cent. betanaphthol, and 12 per cent. balsam of Peru ointment.

B.—THE DEEP INFLAMMATORY DERMATOSES

Under this heading are included those inflammatory conditions which start in the dermis or hypoderm, and only involve the epidermis secondarily. It is often easy to decide clinically whether an inflammation starts in the dermis or in the hypoderm, and strictly these conditions should be described separately; but as the same exciting cause may often produce either condition, it is simpler to describe them together.

The causative irritant may reach the point attacked in three ways—namely, (1) through a crack or puncture in the epidermis, (2) by the lymphatics, or (3) by the blood stream. In the first group are included those cases in which certain chemical poisons are introduced into the skin by the bites and stings of insects (already dealt with on p. 1441), and cases in which micro-organisms are introduced into abrasions, as in the case of erysipelas from the streptococcus (p. 19), syphilitic chancre from the *Spirochæta pallida* (p. 193), soft sore from Ducrey's bacillus, lupus vulgaris and lupus verrucosus from the tubercle bacillus (pp. 1460, 1462), and actinomycosis, sporotrichosis, etc., from certain fungi. The second group includes certain lesions produced by bacterial irritants, such as are seen in the lymphangitis abscess in tuberculosis and the sporotrichial gummata. The third group includes the drug eruptions and other dermatoses, which are labelled toxic eruptions and which are presumably due to chemical poisons circulating in the blood, and also eruptions due to the circulation of micro-organisms, such as are seen in the syphilides and tuberculides. For convenience of description it is proposed to deal with the majority of deep inflammatory dermatoses under two headings—(1) the toxic eruptions, and (2) eruptions produced by living organisms. It must be understood, however, that in the present state of knowledge the ætiology of many of those included in the former group is still very obscure. There is also a third group of dermatoses whose characters make it difficult to place them in either group, and these have, therefore, been described as (3) *dermatoses of unknown origin*.

TOXIC ERUPTIONS

It is practically impossible to produce experimentally in animals any of the toxic eruptions, owing to the fact that no animal has a skin comparable to that of man. Consequently, all our experimental knowledge of toxic eruptions has to be derived from the observed effects of drugs and food-stuffs on the human skin. It is, therefore, proposed to consider first the eruptions produced by these substances.

1. DRUG ERUPTIONS

These fall into two great classes—those produced by non-protein-containing and those produced by protein-containing drugs. Extracts of organs given by the mouth rarely, if ever, produce eruptions and are, therefore, not specially considered.

(a) NON-PROTEIN-CONTAINING DRUGS

These include all the ordinary galenicals.

Two classes of eruption are produced by non-protein-containing drugs.

(1) Non-specific eruptions, which may be produced indiscriminately by many different drugs, and (2) specific eruptions, which are peculiar to certain drugs.

(1) NON-SPECIFIC ERUPTIONS

Symptoms.—These are generally erythematous, urticarial or purpuric. The erythematous rashes may be scarlatiniform, morbilliform, or, more

rarely, of the erythema multiforme type ; sometimes the lesions are vesicular. Urticarial lesions are usually of the simple urticaria type, but occasionally the giant forms are seen. Purpuric lesions are often erythematous at the start and develop hæmorrhages later. It is difficult to classify drugs into any special groups by the reactions they produce, but it may be noted that the under-mentioned types of eruption may be produced by the drugs named :

Erythematous.—Acetanilide, alcohol, arsenic, aspirin, barbituric acid and its derivatives, balladonna, benzoic acid, cantharides, capsicum, chloral, chloralamide, chlorbutol (chloretone), chloroform, copaiba, cubeba, digitalis, ipecacuanha, mercury, opium, phenazone (antipyrine), pilocarpine, phenacetin, quinine, rhubarb, salicylic acid and the salicylates, stramonium, strychnine, sulphonal and turpentine.

Urticarial.—Antimony, arsenic, barbituric acid and its derivatives, benzoic acid, chloral, copaiba, digitalis, opium, phenacetin, pilocarpine, quinine, salicylic acid and the salicylates, santolin, turpentine and valerian.

Purpuric.—Arsenic, chloral, chloroform, copaiba, ergot, hyoscyamus, iodoform, mercury, phosphorus, quinine, salicylic acid and the salicylates, stramonium and sulphonal.

(2) SPECIFIC ERUPTIONS

Symptoms.—Certain drugs give rise to eruptions which are characteristic of the drugs. Arsenic, bromides, iodides, phenazone (antipyrine), phenolphthalein, mercury, silver and gold are the most important.

Arsenic.—In addition to simple erythematous and urticarial lesions, an acute generalised exfoliative dermatitis may develop. This is especially seen after injections of arsphenamine (salvarsan). Herpes zoster also occurs. Pigmentation, especially about the trunk, though it may be more or less generalised, is seen in chronic arsenical intoxication. It usually presents a fine reticular pattern. Hyperkeratosis occurs chiefly on the palms and soles ; it may be diffuse or occur in localised, corn-like projections. Occasionally these localised hyperkeratoses develop into epitheliomata. Excessive sweating of the palms and soles (hyperidrosis) may occur, and the nails may become striated and brittle.

Bromides.—Two main types of specific eruption are seen. Bromide acne is a follicular hyperkeratosis, often closely resembling acne vulgaris and seen in the same situations, but often more extensive, involving the legs and arms, as well as the face, chest and back. It is seen chiefly in epileptics who have taken bromide for some time. The other form is the so-called "anthracoid" form, which is most commonly seen in infants and children ; in the former the drug is often conveyed in the mother's milk. Nodules and tumours varying in size from a pea up to an inch or two in diameter are found, chiefly on the face and legs. The tumours are of a deep red colour and studded with minute pustules ; in the larger lesions the surface is often crusted, and in some cases ulceration occurs. The lesions may develop and persist for a considerable time after the drug has been discontinued.

Iodides.—The most typical lesions produced by iodides are papules which look like vesicles and bullæ, but when pricked only blood escapes. They are sometimes spoken of as "pseudo-bullæ." They are common on the face and extremities, and often appear after taking quite small doses of iodides,

and within a very short time, even as quickly as 24 hours. They are most common in patients suffering from nephritis. These lesions may increase rapidly in size and produce large tumour-like masses, studded with pustules or with a crusted or ulcerated surface, and when occurring in patients who are seriously ill may hasten a fatal termination from septic absorption. In the early stages the cases have been mistaken for small-pox. An acne similar to that produced by bromides is also seen.

Phenazone (Antipyrine).—In addition to producing the more generalised types of eruption phenazone may produce large erythematous reddish or purplish patches, situated discretely over the body, of sharply circular outline and giving rise to a sensation of burning. When they subside they leave a very marked pigmentation, which disappears very slowly.

Phenol-phthalein.—This drug, now largely used as an aperient and contained in many proprietary remedies, occasionally produces an eruption similar to the last named. The patches are of dull purplish colour and come out on face and limbs, but often also affect the mucous membrane of the mouth. The lesions belong to the group known as “fixed eruptions,” as they tend to recur at the same site if the drug is repeated.

Mercury.—This drug occasionally gives rise to a severe erythema in the groins and axillæ, and also on the palms and soles. There may be also purpuric spots and vesicles. Other symptoms of mercurialism, such as nephritis and ulcerative stomatitis, may also be present.

Silver.—Long-continued ingestion of this drug produces a peculiar slaty-grey pigmentation of the skin, generally universal, but most marked on the exposed parts. It scarcely comes under the heading of inflammations, but is included for the sake of convenience.

Gold.—Injections of gold may give rise to a general exfoliative dermatitis, similar to that of arsphenamine.

(b) PROTEIN-CONTAINING DRUGS

These include serums and vaccines. Vaccines rarely cause marked cutaneous eruptions, but when they occur they are of similar nature to those produced by sera. Serum eruptions form a very interesting group, as it is reasonable to suppose that their method of production is closely analogous to that of those toxic eruptions whose ætiology is obscure. For it has been assumed that these are due either to absorption of poisonous proteins produced by the body or to the toxins of pathogenic bacteria which are present in the body.

Serum eruptions are supposed to be an anaphylactic phenomenon and, though this seems to be a reasonable explanation in those cases where the rashes follow a second injection of a foreign protein given at least 14 days after the first, it does not fit in so well for cases in which the eruption follows the first injection of serum. In these cases a specific allergy must be assumed to exist.

Symptoms.—Any of the above-mentioned non-specific eruptions may develop, but there is a greater tendency for the lesions to be of the *erythema multiforme* type. Often the rash is very extensive, the trunk, face, and limbs being covered with disk-like, sharply circumscribed, infiltrated red lesions; these often become bullous, and hæmorrhages may occur in the

centre of the lesions. In other cases ringed lesions occur, which spread peripherally and clear in the centre—*erythema gyratum*. Not infrequently these lesions are associated with fever, and pain and swelling in the joints, and gastro-intestinal disturbances, such as diarrhoea and vomiting and albuminuria. In other cases the lesions are more of an urticarial nature, with transitory, very itchy wheals and swelling of the skin of the face. These eruptions usually come out about a week or 10 days after the injection of the serum and clear up in about the same time.

Similar eruptions sometimes develop in persons vaccinated against small-pox, though in addition a true vaccinal eruption, in which the lesions have the characters of the vaccine vesicles, may develop.

Urticarial lesions develop in certain individuals who are sensitive to certain food-stuffs, after ingestion of these substances. They will be considered more fully under Urticaria (p. 1451).

(c) THE TREATMENT OF DRUG ERUPTIONS

The first thing is to stop the drug causing the eruption. In the erythematous and urticarial types local soothing lotions are indicated, of which the most useful are lotio evaporans (B.P.C.), or phenol min. 120, lot. calaminæ (B.P.C.) ad fl. oz. 12. Alkaline and bran baths are often very useful.

In the serum eruptions, calcium chloride or lactate is recommended and may be given in 10-grain doses three times a day. Quinine, grs. 1 or 2 three or four times a day, is sometimes of value. In the more severe cases the patient must be kept in bed.

2. THE ERYTHEMATA

The term erythema may be used to signify any transient redness of the skin, such redness being frequently produced by external irritants; and these have already been dealt with under the superficial inflammatory dermatoses. When, however, the term is used to describe a composite clinical picture, two main types have to be considered, namely, the non-infiltrative and the infiltrative.

(a) THE NON-INFILTRATIVE ERYTHEMATA

These include two classes, the congestive and the inflammatory. The congestive type has little or no dermatological importance. It is seen in blushing, which is a pure vasomotor phenomenon, and also in such transitory rashes as that seen during ether administration. The inflammatory type includes the rashes seen in scarlet fever, measles and rôtheln, drug eruptions just referred to, and certain other toxic conditions.

Ætiology.—One of the commonest varieties of erythematous rashes is that occurring as the result of septic absorption from a wound, and many of the cases of so-called surgical and puerperal scarlet fever belong to this group. They also occur in ptomaine poisoning and in other infections of the gastro-intestinal tract, but there always remain a number of cases in which no cause can be found.

Symptoms.—Erythematous rashes are usually of either the scarlatiniform, morbilliform or figurate type. The scarlatiniform cases differ from

true scarlet fever in the absence of other typical signs, such as high temperature combined with rapid pulse, the date of appearance of the rash, the presence of sore throat and the characteristic appearance of the tongue. The morbilliform erythemata differ from measles in the absence of coryza, conjunctivitis and respiratory catarrh. The temperature is atypical and Koplik spots are not found. The figurate variety consists of patches or groups of disk-like lesions which tend to spread peripherally and clear in the centre so as to leave circinate and gyrate patterns. It is seen most commonly in drug eruptions, and can hardly be confused with anything else. In all these varieties as the eruption subsides there is a tendency to scaling, usually of the fine branny type, and this may often be extensive; the glove-like scaling of the palms and soles, seen in scarlet fever, may also occur in the simple types of erythema.

Hæmorrhage may sometimes take place into the erythematous patches, especially when these occur on the lower limbs. Most cases of simple purpura really belong to the erythema group. The mucous membranes may be affected similarly to the skin, and other general symptoms, such as diarrhoea, vomiting, fever and albuminuria, may be present.

Treatment.—Attempts must be made to find and remove the cause. All possible septic foci, such as pyorrhœa, tonsillar sepsis and chronic appendix trouble, should be dealt with. The bowels should be made to act freely and a light diet ordered. Cases should generally be put to bed, and always when there is fever or any marked constitutional symptoms. In cases of streptococcal infection, sulphapyridine may be given.

(b) THE INFILTRATIVE ERYTHEMATA. ERYTHEMA MULTIFORME

Ætiology.—Erythema multiforme may occur as a drug eruption, and especially as a serum eruption, as already noted, but it generally appears without any special cause being determinable. It occurs especially in young adults and may often recur, some cases doing so year after year at regular intervals. Individual attacks may clear up rapidly, but in many cases fresh crops appear, and the disease may go on for weeks or months. The joint swellings which sometimes accompany the skin lesions led to the supposition that the condition was of rheumatic origin, but it is probable that these are only a manifestation of a similar affection of the synovia of the joints.

Pathology.—Microscopic examination shows a dilatation of the vessels of the dermis with a leucocytic exudation. There is much local œdema. The epidermis is œdematous, and fluid may accumulate beneath the horny layer or less frequently beneath the epidermis.

Symptoms.—The lesions of erythema multiforme differ from those mentioned in the last section in forming raised infiltrated lesions, which vary in size from a pea to a five-shilling piece or larger, and which have a well-defined distribution. They are usually found on the backs of the hands, wrists, and forearms and on the face, but are not infrequently found on the palms, and may also involve the trunk and lower limbs. In the milder cases they consist of red papules and patches with a sharply-defined border and are usually completely circular in outline. In the more severe forms hæmorrhages occur in the centre, or they become surmounted by bullæ. There is some tendency to slow peripheral extension, with clearing up of the centre,

so that ringed lesions may be formed. In these a play of colours may be noted, the outer red ring surrounding a purple hæmorrhagic ring, which in its turn surrounds a brownish pigmented centre; these lesions are sometimes called *erythema iris*. In rare cases the bullæ so predominate as closely to resemble a pemphigus. The subjective symptoms are often slight, but sometimes itching and burning occur. Lesions may appear on the mucous membranes. Pain and swelling in the joints are not infrequent, and gastro-intestinal disturbance may occur, as may also fever and albuminuria.

Treatment.—Care must first be taken to remove any possible cause. Of the drugs which are of value are calcium lactate, the salicylates and quinine. The former is best given in 240 minim doses on alternate nights. Local treatment is not really necessary, but calamine lotion or linament may be applied to relieve itching or burning.

Erythema nodosum is closely related to *erythema multiforme*, and is dealt with on p. 296.

3. GRANULOMA ANNULARE

This is a very chronic, raised, ringed eruption, of dead white colour, seen chiefly on the dorsum of the hands.

Ætiology and Pathology.—The cause of this condition is unknown, but various intermediate types between it and *erythema multiforme* have been noticed, and this suggests a relationship with the latter condition. Histologically a dense cellular infiltration, associated with degeneration of the collagen bundles, is found in the deeper layers of the dermis, especially in the neighbourhood of the sweat coils.

Symptoms.—The earliest lesions are small white nodules, seen most commonly on the back of the finger joints. Such lesions often occur in groups. They spread slowly, the centre of the group becoming flattened, and surrounded by a raised, white festooned margin, which can be seen to be made up of nodules of the same character as those of the original lesions. The disease is very slowly progressive, and may last for months or years if not treated. The lesions are generally localised to the hands and wrists, but are occasionally seen in other parts of the body, such as the nape of the neck, the buttocks, elbows and knees. In some cases subcutaneous fibrous nodules have been described in the neighbourhood of the elbow joints and elsewhere. Subjective symptoms are generally absent.

Treatment.—Internally, quinine and the salicylates may be given. X-rays will often cause the lesions to disappear, but no other local treatment has much effect.

4. THE PURPURAS

The purpuras form a group which are closely related to the erythemata and are very often erythematous at the start. In them, however, the blood vessel walls are damaged and hæmorrhage occurs into the dermis. They may be of toxic or of bacterial origin. They are considered in detail on p. 810 *et seq.*

5. THE URTICARIAS

The urticarial eruptions are characterised by the presence of wheals, or localised areas of œdema. These are usually transitory in character and

are accompanied by severe itching. Several different forms are recognised—(1) Simple urticaria, (2) factitious urticaria, (3) giant urticaria, (4) papular urticaria, and (5) urticaria pigmentosa.

Ætiology.—As is pointed out above, urticaria can be produced by the ingestion of certain drugs and by injections of foreign sera. It can also be produced by the ingestion of certain food-stuffs in susceptible persons; for example, porridge, strawberries, shell-fish, eggs and milk. It also occurs after the consumption of decomposing food. As far as is known it is not due to the direct attack of any micro-organism, though syphilitic urticaria has been described. It is clear that it may be produced not only by protein poisons, but by non-protein poisons circulating in the blood. The actual mechanism by which the lesions are produced is not altogether clear. At one time it was thought to be a pure vasomotor neurosis, and that the poisons mentioned acted on the vasomotor centres; but in recent years it has been demonstrated that the lesions are true inflammations, and, therefore, it is probable that the action of the poison concerned is a local one. At the same time external stimuli, such as friction, seem, in many cases, to play a part in determining the points where the poison acts. Lewis believes that the lesions are produced by the liberation of a histamine-like substance from the tissue cells. It must also be noted that urticarial lesions may be directly produced by the injection of poisons into the skin. This is well seen in the bites and stings of insects, and the stings of plants, such as the nettle. In a large number of cases of urticaria, however, it is difficult to find any cause, and these are usually considered to be auto-toxic. The auto-toxin may be generated in the intestinal tract, or in infected foci, such as septic tonsils and teeth and inflammatory trouble in the pelvis. In the giant urticaria cases there is usually a considerable functional element present, and these cases are considered to be vasomotor neuroses. Nothing is known of the ætiology of urticaria pigmentosa.

Symptoms.—*Simple urticaria.*—This is the form most frequently met with in adults. It may occur in an acute or in a chronic form. In the former the eruption appears suddenly, is often accompanied by general symptoms, such as fever, diarrhoea and vomiting, and subsides more or less rapidly. In the chronic type the eruption appears in crops; the individual lesions run a more or less rapid course, but fresh crops continue to come out at intervals and the condition may persist for many weeks, months or even years.

The lesions of simple urticaria are in their earliest stages pale pink papules or patches, varying in size from a pea to an inch or two in diameter. In a short time—sometimes a few minutes, at others an hour or so—the central part of the patch becomes a dead white colour, is firm to the touch and raised a millimetre or two from the surrounding skin. The lesions are intensely itchy. They may be few in number or very numerous; sometimes the whole body may be covered with patches of all shapes and sizes, and figurate patches are common. The eruption is most common on the trunk, but any part of the body, including the mucous membranes, may be affected.

Factitious urticaria.—This is a condition of the skin in which the slightest trauma, such as a slight scratch, will bring out a wheal. This condition is often present in simple urticaria, but frequently exists apart from any spontaneous eruption. The condition is sometimes referred to as *dermatographism*, as it is possible to produce letters in urticarial wheals on the patient.

Giant urticaria.—In this condition the lesions are not so much wheals as circumscribed patches of oedema. They are particularly liable to occur about the face—the eyelids, cheeks and lips often swelling up quite suddenly—burning or itching being an accompanying phenomenon. The mucous membranes are not infrequently attacked, and in a few cases sudden cedema of the larynx may produce dangerous asphyxial symptoms. The lesions are very prone to recur, and these recurrences may persist for years. This condition is sometimes spoken of as *angio-neurotic cedema* (see p. 1073).

Papular urticaria.—This type may be a distinct disease or only a variant of simple urticaria, but is that commonly seen in infants and young children. It is not very commonly seen in breast-fed infants, though it does occur, but otherwise is chiefly seen in the first two years of life; it may in some cases, however, persist, with intermission, up to about 7 years of age. The lesions appear, just like those of the adult form, as pink oval patches, usually about $\frac{1}{2}$ inch in diameter, but instead of the bulk of the whole patch being converted into a wheal only a central pinhead-sized wheal is produced. Itching is intense and often paroxysmal. When the lesion is scratched the central papule becomes inflamed and a bloodstained crust is formed on its summit, and it persists after the surrounding pink zone has disappeared. The cases usually present discrete pinhead-sized papules, covered with bloodstained crusts, suggesting a parasitic origin. Not infrequently vesicles surmount the wheals and occasionally quite large bullæ are found.

The eruption comes out in crops, especially at night, and chiefly on the extensor aspect of the lower limbs, the buttocks and the extensor aspect of the forearms, but may occur almost anywhere on the body. The children seem to suffer little in general health, though sleep at night is often lost, its effects being often more obvious in the parents.

Urticaria pigmentosa.—This is a rare condition and it is still a question whether it should be grouped with the other urticarias. It is chiefly a disease of infancy and childhood, but a certain number of adult cases are on record. The condition may appear within the first few days of life, and in a few cases lesions are said to have been present at birth.

The lesions usually appear as wheals, $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter; and as these disappear a yellowish-brown colour is left, and the lesion may remain raised above the surrounding skin or may flatten down to a pigmentary macule. These pigmented lesions generally persist for years. Itching may or may not be present. Sometimes marked factitious urticaria can be elicited. It would appear that occasionally simple urticarial lesions may leave behind pigmentation, but the condition described above is something quite distinct. In true urticaria pigmentosa there is generally a great increase in the mast-cells of the dermis which is absent in ordinary urticaria leaving pigmentation.

Diagnosis.—Simple urticaria may be confused with dermatitis herpetiformis and with the premycotic stage of mycosis fungoides. In the former, small deep-seated vesicles usually occur; but as they are not always present the diagnosis may be difficult. From the latter a diagnosis cannot be made with certainty, though when occurring in old people and persistent, mycosis fungoides must be considered.

The giant forms must be distinguished from erysipelas and acute erythematous eczema. In the former, high temperature is present, the lesion has

a sharply defined, slowly spreading margin, and is often blistered. In the latter, the eruption is extensive and symmetrical, the skin is red, and vesicles are frequently present. Chronic erysipelas of the lip is a persistent, slowly increasing condition and usually arises from a persistent crack in the lip, while urticaria has a sudden onset and disappears again.

Papular urticaria in children is most frequently confused with scabies. The diagnosis can be settled by the presence of burrows and the finding of acari in the latter condition. In the vesicular form cases may be confused with varicella, but the course of the eruption is quite different.

Urticaria pigmentosa is not likely to be confused with any other condition.

Treatment.—If a cause can be found it must be removed: articles of diet known to cause the eruption must be avoided and all possible septic foci dealt with. If the cause is not clear, various types of food must be stopped one by one in order to exclude a possible source of trouble. Recently a cuti-reaction to various foodstuffs has been devised to detect the causative agent, and it is possible to desensitise patients from the particular poison to which they are susceptible. Apart from this, mild purgation and the administration of intestinal antiseptics, such as salol, ichthammol, creosote and calomel, can be recommended. Some patients improve on tonic drugs, such as iron, arsenic and quinine. Calcium chloride or lactate given over prolonged periods is efficacious in some cases. In others complete freedom from work and even rest in bed are necessary. In cases of unknown ætiology, non-specific protein therapy, such as injection of whole-blood, milk or peptone, is of great value (see p. 1418).

In the papular form in children excessive intake of sugar plays a part in a proportion of cases, and by a rigid cutting down of jams, sweets, etc., relief is often obtained.

In the giant form nerve sedatives, such as valerian and the bromides, are of value.

Locally, anti-pruritic lotions are most useful. Solution of coal tar and subacetate of lead, min. 120 of each to fl. oz. 8 of water; or liq. potass. hydroxid. min. 60, glycerin min. 60, to fl. oz. 8 of water, may be used and can be applied frequently; alkaline and bran baths also give considerable relief. In children a teaspoonful of liq. picis carbon. added to a warm bath before going to bed is a valuable remedy, and in some cases sulphur ointment grs. 15 to 1 ounce has proved useful; but lotions are usually better tolerated.

There is no known treatment which affects urticaria pigmentosa.

6. PRURIGO

Prurigo of Hebra is a condition which is rare in this country, but is not uncommon in Eastern Europe. It is apparently closely connected with papular urticaria, but most authors consider it a distinct affection. It begins usually in the first or second year of life, by the appearance of intensely itchy, pinhead to lentil-sized papules on the extensor aspects of the limbs, chiefly on the legs and forearms; these soon become covered with bloodstained crusts, and eventually the whole of the skin of the affected area becomes thickened (lichenified), pigmented and excoriated. The lesions may eventually involve the whole of the limbs, but the flexures usually escape. The trunk, neck and face may become eczematized and lichenified. The glands

in the groins and axillæ become much enlarged. The milder cases (*prurigo mitis*) may eventually respond to treatment and get well about the time of puberty, but the more severe cases (*prurigo ferox*) persist throughout life, the patient eventually succumbing to the disease.

Treatment.—There is no specific treatment. Baths and sedative lotions and ointments, together with sedative drugs, to relieve itching and to ensure sleep, should be employed.

7. DERMATITIS HERPETIFORMIS

Dermatitis herpetiformis or Duhring's disease is a condition characterised by the appearance on the body of crops of erythematous or urticarial papules or patches, usually surmounted by herpetic vesicles or bullæ, and giving rise to intense itching.

The evidence for placing this disease among the toxic eruptions is not conclusive, but is sufficiently suggestive to make it justifiable. In this connexion may be mentioned its occurrence as one of the rarer toxæmias of pregnancy (*hydra gestationis*), and its close clinical resemblance, in some cases, to the urticarias and the erythemata.

Ætiology and Pathology.—The disease may occur at any time of life ; it is rare in infancy and childhood, although cases have been reported ; it is fairly common in young adult life, but most cases occur in middle life. Both sexes are pretty equally attacked. A special form occurs in association with pregnancy, and is apt to recur with each pregnancy. The malady does not appear to be associated with the presence of any particular micro-organism in the body, and cultures from vesicles and bullæ are sterile in their early stages. Blood cultures are also negative.

It has been considered by some to be due to some nervous derangement, and it is true that it is sometimes associated with neuroses ; it is probable, however, that these are a product rather than a cause of the disease. It is probable that the condition is produced by an unknown toxin.

Microscopically lesions show a dense, cellular infiltration of the superficial part of the dermis, chiefly around the vessels. There is always considerable superficial œdema, which in the vesicular and bullous cases collects under the epidermis, lifting it from the underlying dermis ; the fluid of the vesicles contains a large number of eosinophil corpuscles.

Symptoms.—The eruption is essentially polymorphous, that is to say, all the types of lesions mentioned above may be present in the same case at the same time. Most commonly the lesions look like irregularly figurate urticarial wheals which are surmounted by numerous shotty vesicles. In other cases the lesions are more frankly erythematous, while in others larger vesicles or bullæ form, either with or without an underlying erythematous or urticarial patch. In all cases, except when the bullæ are large, there is intense itching in the lesions, with the result that they are scratched, and small bloodstained crusts or excoriations are seen mixed up with the other manifestations of the disease. The limbs and the trunk are most frequently affected, but any part of the cutaneous surface may be involved, though rarely the palms and soles, and the mucosæ are attacked in a considerable percentage of cases. There is a great tendency for the lesions to recur, fresh crops coming out at frequent intervals, and the disease may

persist for years; the writer has under his care a case of over 40 years' duration.

The general health usually suffers very little, in spite of the fact that sleep is often disturbed. Gastro-intestinal symptoms, such as diarrhoea and vomiting, may occasionally occur, and in fatal cases lesions have been found in the gastro-intestinal tract, which possibly account for the above-mentioned symptoms. An increase in the eosinophil corpuscles in the blood occurs in the majority of cases.

Diagnosis.—From erythema multiforme it can be distinguished by the irregular distribution, the shape and the polymorphic character of the lesions in conjunction with the intense itching which occurs; from urticaria by the presence of vesicles and bullæ, which are almost unknown in simple urticaria, though not infrequent in the papular variety, and by the persistence and recurrence of the lesions; and from pemphigus by the itching, the polymorphous character of the lesions, and the comparatively slight effect on the general health.

Treatment.—This calls for much patience on the part both of the patient and the physician. In the first place a careful examination, both clinical and bacteriological, must be made to find any focus of disease. Of the internal remedies most reliance has been placed on arsenic, and in some cases the eruption ceases when a certain dose is reached; but this is by no means always the case. Arsenic may be given by the mouth as Fowler's solution or Asiatic pill, or by injection, the cacodylate of soda, enesol and arsphenamine (salvarsan) being the favourite preparations. The dose should be small to start with, and increased to the limit of tolerance; and the drug should be discontinued if no definite result is obtained. Aperients and saline lavage of the bowel are satisfactory in severe cases; in others quinine, salicin and sodium bicarbonate have proved successful. In any case attempts should be made to check the itching. Alcohol and coffee should be stopped, and also all excess of sugar in the diet. Phenacetin and phenazone are useful, and sedatives may be given occasionally at night. The injection of certain non-specific protein substances, such as the patient's own blood, horse serum, sterilised milk or peptone, has occasionally produced a cure.

Local remedies consist mainly of anti-pruritic applications. The most useful is a lotion containing 2 to 3 per cent. phenol, 1 per cent. liq. potass. hydroxid., or 2 to 5 per cent. liq. picis carbonis. Alkaline and bran baths are valuable, especially in the bullous cases—in which cases also weak (5 per cent.) sulphur ointment sometimes acts well.

8. PEMPHIGUS

An inflammatory condition of the skin, characterised by the eruption of blisters usually occurring in crops, and associated with constitutional symptoms.

Four different varieties are recognised: (1) acute pemphigus, (2) chronic (pemphigus vulgaris), (3) pemphigus foliaceus, and (4) pemphigus vegetans. Acute pemphigus is now known to be a definite bacterial infection, and should not strictly be included in this group; but it is placed here for convenience of description. In the other three varieties the cause is unknown, but it is believed that they are of toxic origin.

(a) ACUTE PEMPHIGUS

Ætiology.—This is a rare condition which occurs almost entirely in butchers, and appears to be due to inoculation of some abrasion with a pathogenic micro-organism, the diplococcus of Pernet and Bulloch. The lesions are not produced by local spread, as in impetigo contagiosa, but are distributed through the blood stream, and symptoms of acute toxæmia occur simultaneously with (or even before) the appearance of the eruption. The diplococcus can be obtained from the bullæ, as well as from the blood.

Symptoms.—The disease commences acutely with fever, malaise, nausea and vomiting. Bullæ then appear suddenly on the apparently normal skin; they are usually very numerous, and as big as a pigeon's or hen's egg. They may burst, leaving a red oozing surface. The lesions usually involve the mucous membranes of the mouth and throat, and even of the intestinal tract, causing pain and difficulty in swallowing, diarrhœa and vomiting, and secondary bronchitic and broncho-pneumonic complications. In a large proportion of cases a general septicæmic condition supervenes and the patient dies, but in a few milder cases the lesions dry up and the patient recovers. Rarely cases may become chronic.

Treatment.—The cases are so infrequently seen that little can be said on this point. The main principles are to keep the patient in bed, puncture the vesicles, and apply mild antiseptic dressing, e.g. 1 in 4000 potassium permanganate, with a thin layer of paraffin gauze between it and the skin, or 1 in 1000 acriflavine emulsion. Quinine is recommended as the best internal remedy. No observations have been made, as far as the writer knows, with regard to specific therapy.

(b) CHRONIC PEMPHIGUS

Symptoms.—The affection, which is also rare, is characterised by the appearance of crops of bullæ in various parts of the skin and mucous membranes, each bullæ appearing on the skin without any pre-existing erythematous or urticarial lesion. The eruption is not associated with itching, but the general health suffers, thus differing from dermatitis herpetiformis. Further, the bullæ are usually sterile in their early stages, though both the *Staphylococcus pyogenes albus* and the *Bacillus pyocyaneus* have been found; but these are almost certainly secondary infections.

The bullæ usually dry up in the course of a week, leaving an erythematous and scaly patch, which subsequently disappears; but fresh crops of blisters constantly come out, and this state of affairs may persist for months and years. The general tendency is for the disease to continue, while the general health steadily deteriorates, and finally death supervenes.

Diagnosis.—This has to be made from dermatitis herpetiformis and from the bullous form of erythema multiforme. The main points in the diagnosis of the former have been considered in a preceding paragraph. In erythema multiforme the lesions are especially distributed on the extremities, and some of them show the definite coin-shaped erythematous patches. The attack usually lasts only a week or two.

Treatment.—This is very unsatisfactory. Arsenic in full doses has

given good results, and quinine and salicin are also recommended. Suramin (antrypol, germanin Bayer 205) appears to give good results in some cases. Gastro-intestinal antiseptics and colon lavage are useful. Intramuscular injection of horse serum, or of the patient's own serum, may be tried. Local treatment is the same as for acute pemphigus.

(c) PEMPHIGUS FOLIACEUS

This is probably only an extensive type of chronic pemphigus.

Symptoms.—Bullæ appear frequently and over large areas of the body, and as a result set up a condition resembling generalised exfoliative dermatitis. When this stage is reached fresh bullæ are not properly formed, owing to the permeability of the improperly formed horny layer, abortive flaccid lesions constantly appearing on the affected areas. Crusting, scaling and a tendency to warty formations, together with much pigmentation, are present. The trunk, neck, face, scalp and limbs may all be attacked, but usually the hands and feet are much less affected.

The general health is usually much affected; but this is not always so. The writer has had under his observation a case of 20 years' duration who, apart from the skin condition, was otherwise well. Usually, however, cases end fatally in a year or two.

Diagnosis.—The only condition likely to be confused with this is generalised exfoliative dermatitis; but in this latter condition bullous formation is absent.

Treatment.—This is the same as for chronic pemphigus.

(d) PEMPHIGUS VEGETANS

Symptoms.—The lesions in this type frequently first appear in the mucous membrane of the mouth, but may appear on other parts of the body. When fully developed they are chiefly localised to the flexures of the axillæ, elbows, groins, knees and around the anus and vulva. The initial lesion is a flaccid blister, which on rupture develops fungating granulations from its base, which discharge much fetid secretion. The patient's health suffers rapidly from septic absorption, and he usually succumbs in the course of a few months.

Diagnosis.—The condition when fully established is characteristic, but in the early stages the lesions might be mistaken for syphilitic mucous tubercles. The diagnosis can be settled by the presence of other syphilitic lesions, by finding the spirochæta pallida, and by the Wassermann reaction.

Treatment.—No treatment is known to influence the course of the disease. Local antiseptics are required for the lesions, weak perchloride of mercury and eusol being the most likely to be satisfactory. Otherwise treatment is on the same lines as for pemphigus chronicus.

9. DERMATITIS EXFOLIATIVA

Synonym.—Pityriasis Rubra.

This is a generalised inflammation of the skin, characterised by redness and profuse scaling. There are many types of this condition, and divers

causes. It is customary to divide dermatitis exfoliativa into primary and secondary varieties. The former occurs without any pre-existing dermatosis, while the latter really represents the generalisation of some other skin inflammation, such as eczema, seborrhœic dermatitis or psoriasis. When such diseases generalise there is a tendency for them to take on the character of a primary exfoliative dermatitis, presently to be described.

The primary variety occurs in its most striking form in salvarsan poisoning, and may also occur in such diseases as leukaemia and mycosis fungoides—diseases closely related to one another. There still remain, however, a number of cases in which no cause can be found, and which we are probably justified in considering as toxic eruptions.

Symptoms.—The cases due to arsphenamine may be taken as typical of the group. In these the eruption usually commences as bright, scarlatiniform patches on the flexor aspect of the forearms and on the abdomen and chest. The rash spreads rapidly, so that in a day or two the whole body is covered. At first it is found to consist of distinct pinhead-sized macules, chiefly around the follicles, but soon it becomes one continuous red sheet. Almost immediately the eruption begins to scale; the scales may be of the fine branny type, or large like fish scales. The amount varies in different cases, but is often very considerable, the bed having to be swept out several times a day. The onset is not always as described; sometimes the initial eruption is an urticaria, or even like an acute erythematous eczema, involving the face and forearms, and in a case recently under the writer's care it was erythematous-vesicular at the commencement. Once the eruption is fully established it usually takes 2 to 3 months to disappear. In an uncomplicated case the rash gradually subsides, scaling ceases, and the skin assumes its normal colour, though some thickening may remain for a considerable time. The flexures of the limb and the neck usually are the last to clear. The hair is frequently completely shed, but grows again later, and the nails may also be lost temporarily, though this is less frequent. An irregular thickening of the nails is, however, more common. At the onset there may be fever, malaise and intestinal disturbance; later, in the course of 3 to 4 weeks, bronchitis and broncho-pneumonia may supervene, and sometimes cause a fatal issue. Nephritis may also occur, and may cause permanent renal changes. If careful nursing is not provided the skin may become infected, and septic absorption may occur. There is always much enlargement of the lymphatic glands, which are soft and spongy, and conjunctivitis is sometimes present.

Most of the idiopathic cases run a similar course; but several different varieties have been described, among which the condition known as pityriasis rubra of Hebra is apt to be associated with visceral complications and with skin atrophy, and runs a very fatal course.

Diagnosis.—The primary cases must be distinguished from those due to leukaemia and mycosis fungoides. In the former the blood picture will probably clear the diagnosis, and in the latter the severe itching, which usually accompanies it, and which is usually absent in the simple exfoliative dermatitis cases.

Prognosis.—This is always uncertain, and should be very guarded. Cases may clear up in 2 or 3 weeks, or may persist for years, with gradually increasing prostration ending in death.

Treatment.—In the arsphenamine cases sulphur appears to be the best remedy. It may be given as sodium thiosulphate by intravenous injection. The main internal remedies must be directed to maintaining the general health, and to countering complications as they arise. Complete rest in bed is indicated in all cases, however mild they may at first appear, and the warmth of the body must be maintained. Warm bran baths may be given if no fever is present, and dusting the skin with talc powder makes the patient comfortable. Local septic complications must be dealt with by mild antiseptic creams or pastes.

ERUPTIONS DUE TO BACTERIA AND FUNGI

Having dealt with the deep inflammatory dermatoses produced by toxins circulating in the blood, it is now necessary to consider those which are caused by living organisms reaching the skin by the same channels. Three of these form a very important group of dermatoses, namely, tuberculosis, syphilis and leprosy. The two latter have been dealt with on pp. 193 and 121 respectively. There are, however, certain others which require notice. It is probable that the eruptions of certain specific fevers may be due to the presence of the infecting organisms in the tissues; this is known to be the case in the rose spots of typhoid fever. The erythematous and purpuric eruptions sometimes seen in malignant endocarditis and other septicæmic and pyæmic conditions are also probably due to the direct action of the streptococcus, while it will be necessary later to describe the cutaneous manifestations produced by the gonococcus when it enters the blood stream.

1. TUBERCULOSIS CUTIS

The tubercle bacillus may attack the skin in several different ways. The commonest variety is a superficial granulomatous formation known as lupus vulgaris; this variety can apparently be produced both by local inoculation and through the blood stream. Lupus verrucosus, a variant of this type, is generally a local inoculation, and is accompanied by warty overgrowths. Miliary tuberculosis of the skin may accompany general miliary tuberculosis, and local tuberculous ulcers may also form, but are chiefly seen on the mucous membranes. In addition, an infection of the skin may occur when a tuberculous abscess, either from a suppurating lymphatic gland or bone, bursts through the skin, and this is spoken of as scrofuloderma. There are also groups of cutaneous and subcutaneous tuberculous lesions, produced by bacilli circulating in the blood, which are called tuberculides, and include several varieties, the lichenoid or lichen scrofulosorum, the acneiform or acne scrofulosorum, the papulo-necrotic, and the gummatous (erythema induratum or Bazin's disease).

(a) LUPUS VULGARIS

Pathology.—The lupus nodule is composed of a group of ordinary miliary tubercles, such as are seen in the lungs and elsewhere. It consists of groups of epithelioid cells, surrounding giant cells, often with peripherally arranged

nuclei, the whole being surrounded by a dense mass of round cells. Tubercle bacilli have been demonstrated in the lesions, and inoculation into guinea-pigs will produce tuberculous lesions.

Symptoms.—Lupus vulgaris usually attacks the face ; but it is not uncommon to find patches on other parts of the body. In this latter site it may be symmetrical. Usually on the face it is asymmetrical. It is most frequent on the nose or cheek, and frequently begins in childhood. The earliest lesion is a small, dull-red pinhead-sized spot, to which other similar spots are soon added, the whole being surrounded by an erythematous zone. On pressure with a lens, however, these original spots can be distinctly seen as yellowish points or nodules compared in appearance to apple jelly. They are very soft, and if a pointed match is applied to one of them it sinks easily into the nodule. These patches may slowly spread so as to involve considerable areas, and may persist for a long period without ulceration. In other cases, however, ulceration may supervene, and considerable destruction of tissue take place, especially on the nose, which is often completely destroyed up to the edges of the nasal bones. The bone itself is not attacked ; but the cartilage may completely disappear. If healing occurs, a soft, superficial scar is produced ; but fresh nodules are liable to appear in it. Any part of the face may be attacked ; but the scalp usually escapes. The glands in the neck may enlarge and occasionally break down, but this is uncommon. Facial lupus vulgaris is frequently complicated by similar lesions in the mucosa of the nose and mouth, and these situations are sometimes the primary seat of the disease. On the mucous membranes the nodules are not visible, but sharply defined, raised, rather warty-looking patches occur. It is commonly seen on the inner aspects of the cheek, gums, palate and nasal mucosa ; but the pharynx and larynx may be involved.

On the body the patches may attain great dimensions ; they often spread at the margins and heal in the centre, forming irregular gyrate patterns. As a rule they are of the non-ulcerating type ; but in some cases ulceration occurs. In some cases, too, considerable contraction of the skin results, leading to deformity ; in others the lymphatic vessels become blocked, and a condition of elephantiasis may supervene.

The disease usually spreads very slowly, and lasts a great number of years. Some cases remain practically stationary almost indefinitely. A few, however, spread rapidly and defy treatment.

In old-standing cases there is a definite tendency to the development of carcinoma. This is of the squamous type, and does not as a rule give rise to secondary carcinomatous glands. It can, therefore, generally be removed locally.

Diagnosis.—Lupus vulgaris is most easily confused with lupus erythematosus (p. 1466). In the former definite nodules are present, and there is a tendency to ulceration ; the disease usually begins in childhood and persists for many years. In the latter the disease is non-ulcerative, has a great tendency to be symmetrical, rarely appears before adult life, and is especially liable to occur in the middle-aged. The scalp is often attacked, while this is rare in lupus vulgaris. In lupus erythematosus thick adherent scales, fixed down by epithelial plugs, form, and the scar is often covered with pits of varying size ; in lupus vulgaris the scale is of a very superficial type and easily removed, and there are no plugs.

From tertiary syphilis the diagnosis may occasionally be difficult, and the two conditions may be combined. The absence of nodules, the tendency to form sharply cut, rather deep ulcers, the presence of a positive Wassermann reaction, and the rapid response to anti-syphilitic remedies will generally settle the diagnosis.

Rodent ulcer is distinguished by its appearance fairly late in life, by its firm rolled edges of pearly white colour, and by its tendency to involve the bony structures of the face.

Treatment.—Of prime importance is the general health of the patient. Good ventilation, sunlight, warm clothing and plenty of good food are necessary if cases are to do well. In the absence of a good supply of natural sunlight excellent results are obtained by exposing the body to the rays of the carbon-arc or mercury-vapour lamps. As to drugs, cod-liver oil, malt, iron and arsenic are often of considerable value.

Local measures should also be taken to destroy the lesions. Small patches may be excised; but this method is only rarely applicable. If the patches are non-ulcerating, and not too extensive, excellent results are obtained by the Finsen light; but this method is rarely available, and is very slow. The nodules may be destroyed by the application of 20 per cent. ac. salicylic plaster with cresote, or 10 per cent. pyrogallie acid ointment rubbed in daily until a violent reaction is produced. Adamson has recommended rubbing acid nitrate of mercury into the patches, and this gives very satisfactory results. In the larger ulcerating patches a preliminary scraping, followed by the application of acid nitrate of mercury or zinc chloride stick, is usually satisfactory. The use of X-rays and CO₂ snow has nothing to recommend it, and the former is very dangerous if given over prolonged periods. On the other hand, some satisfactory results have been reported after treatment with "Grenz" rays.

(b) LUPUS VERRUCOSUS

This is due to the local inoculation of the tubercle bacillus, and is chiefly seen in those who handle infected meat, and in those who conduct autopsies. It is also known as *verruca necrogenica* or *post-mortem wart*.

Symptoms.—The lesions usually occur on the hands, chiefly the dorsum and on the knuckles. The earliest lesion is a small, red, firm papule, which spreads slowly. The centre soon becomes raised and warty; but there is always a well-marked inflammatory zone around this warty growth. Serum and pus may exude between the papillæ of the wart, and the whole may be crusted. The lesion is generally single, and may attain several inches in diameter. Rarely numerous lesions are present.

Treatment.—Small lesions are best excised. Destruction by the actual cautery, or by diathermy, may be practised in some cases. In this variety a pastille dose of X-rays may flatten down the warty growth, and this may be followed by the use of salicylic acid or mercurial plasters, or by painting with acid nitrate of mercury.

(c) LOCAL TUBERCULOUS ULCERS

These occur usually as complications of tuberculosis of other organs. They are frequently present on the mucous membranes, or around the orifices

of the body. Little can be done except palliative treatment if they are numerous ; but isolated ones can be destroyed by one of the methods already described.

(d) SCROFULODERMIA

This term is applied to secondary infection of the skin from the bursting of deep-seated tuberculous abscesses. The lesions either take the form of a thick crust overlying an area of unhealthy-looking granulation tissue, or are purplish-red shiny areas surrounding a sinus.

The term is also applied to single or multiple subcutaneous abscesses, not infrequently seen in children, which contain pus, in which numerous tubercle bacilli can be found. They are sometimes called *tuberculous gummata*.

Treatment.—The treatment of the underlying condition is essentially surgical and where possible the affected skin should be excised, otherwise scraping, followed by painting with acid nitrate of mercury, is the best treatment.

(e) THE TUBERCULIDES

These lesions, which are due to the lodgment of tubercle bacilli in the peripheral capillaries, with the production of a local inflammatory reaction, differ from the foregoing tuberculous diseases of the skin in that there is no tendency for the individual lesions to spread. They are thus comparable to the secondary syphilides, and like them are of several different types.

THE LICHENOID TUBERCULIDE.—**Symptoms.**—This condition, also known as *lichen scrofulosorum*, is chiefly seen in young children with glandular tuberculosis. The lesions come out in crops, chiefly on the trunk, and are arranged in circular or oval groups, made up of pinhead-sized acuminate follicular papules. These lesions may be of the same colour as the normal skin, or of bright red colour. There is usually a small crust on the summit of each papule, or sometimes a small pustule. The disease lasts from a week or two to many months.

Diagnosis.—In lichen spinulosus there is less obvious inflammation, and a horny spine projects from the centre of the papule, which can be removed by forceps.

The small follicular syphilide occurs in adults, and is associated with other syphilitic phenomena.

Treatment.—No special treatment of the skin has any effect. The treatment is that for glandular tuberculosis.

THE ACNEIFORM TUBERCULIDE.—**Symptoms.**—This condition, also known as *acne scrofulosorum*, occurs in children and adolescents who are suffering from some form of tuberculosis, and chiefly affects the buttocks and thighs, but may be more extensive. The lesions are lentil-sized, acuminate, follicular papules and pustules, and are generally distributed discretely. They are of bright red colour and pustular or crusted.

Diagnosis.—The affection is sometimes difficult to diagnose from staphylococcal folliculitis ; but the individual lesions run a much slower course, and are usually more numerous and not painful.

Treatment.—This is the same as for the lichenoid tuberculide.

THE PAPULO-NECROTIC TUBERCULIDE.—Symptoms.—In this variety of tuberculide the lesions are small lentil- to pea-sized nodules starting deep in the dermis or in the hypoderm, eventually softening and bursting through the skin with the production of a small rather indolent ulcer. After healing, pitted scars are left. The lesions are usually numerous and come out in crops, which may continue to appear over a period of some years. The parts affected are chiefly the distal extremities of the limbs, *e.g.* the backs of the hands and feet, the sides of the fingers and the extensor aspects of the forearms and legs. Lesions in these regions have been named *folliculitis*. Somewhat similar lesions have been described on the face and termed *acutis*, but it is still not quite clear that they belong to the same group. The eruption nearly always occurs in patients who have some other manifestations of tuberculosis, and occurs chiefly in young adults.

Diagnosis.—These cases can be distinguished from *erythema multiforme* by their deeper site of origin, and by their tendency to ulcerate and produce scars.

Treatment.—As for other tuberculides. No local treatment has any effect on the lesions.

THE GUMMATOUS TUBERCULIDE.—Symptoms.—This condition, which also goes by the name of *erythema induratum* or *Bazin's disease*, is not uncommon, and is almost entirely confined to the legs, especially the calves, and is usually bilateral. It occurs chiefly in girls and young women between the ages of 15 and 25. The initial lesion is a deep-seated nodule, from a pea to a hazel-nut in size, starting in the subcutaneous fat. The nodule slowly increases in size, involves the skin, which becomes purplish in colour, and eventually softens and bursts. The ulcer thus produced has a ragged edge and an unhealthy purplish-red base, often covered by a dirty greenish slough. These ulcers are very sluggish, and take weeks or months to heal. Fresh lesions are constantly forming, and a dozen or more lesions may be found simultaneously on the two limbs. There is often considerable pain in the lesions.

Diagnosis.—From syphilitic gummata.—In this condition the lesions are less numerous—indeed often single—and are rarely so symmetrical. The lesions are usually painless. The edge of the ulcer is sharper and more cleanly cut, and the base is cleaner, or has a characteristic wash-leather slough. Other stigmata of syphilis may be present, and the Wassermann reaction is positive.

In older patients, mainly women, similar nodules occur, which do not break down and ulcerate. These are probably of the same nature but have been called *hypodermic sarcoids*, and must not be confused with *cutaneous sarcoids* described under *sarcoidosis*.

Treatment.—The ulcers heal readily if the patient is kept in bed, but are liable to recur when she gets up again. General tonic treatment should be given, and in many cases arsphenamine has proved to be a potent remedy. Locally antiseptic baths and dressings are required.

2. LUPUS ERYTHEMATOSUS

An inflammatory condition of the dermis, usually chronic but occasionally running an acute course, characterised by the presence of circumscribed

red patches, with or without adherent scales, and which on recovery leaves scars.

Ætiology.—This is still unknown. It has for many years been thought to be due to toxins of the tubercle bacillus and is frequently associated with tuberculosis, but cases occur in which this disease cannot be traced. Lately a good deal of attention has been called to focal sepsis as a cause, but the evidence is no more conclusive than for tuberculosis. It is thought that it may be produced by more than one variety of toxin, or that it may be due to a specific organism as yet undiscovered. There can be little doubt that exposure to sunlight tends to bring out the lesions. The disease is chiefly found in middle age, but may begin before the age of 20. It is more frequent in women.

Pathology.—The chief change in the skin is an infiltration in the neighbourhood of the vessels of the dermis with round cells, which may destroy the hair follicles and sweat ducts. In the epidermis there is a hyperkeratosis, which is especially marked at the follicular openings, so that horny plugs are formed.

Symptoms.—Two main types are seen—(1) The erythematous, and (2) the scaly or fixed type.

1. *The erythematous type.*—This is less frequent and has a greater tendency to be generalised. It may run an acute course or may develop into the scaly type. The lesions are chiefly seen on the cheeks and form circumscribed disk-like lesions, raised and slightly infiltrated and of a pale red to a purplish-red colour. These often show patulous follicular openings on the surface. They may also occur as diffuse flat non-infiltrated sheets of redness. This type is very apt to be associated with lesions in other parts of the body, particularly the backs of the hands and fingers, the arms and forearms, the chest, neck and ears. Occasionally an almost universal eruption appears. The patches may sometimes become bullous and hæmorrhage may occur into the bullæ. These disseminated cases may be associated with acute visceral diseases, such as pneumonia, pleurisy and nephritis, and even in the absence of these high fever may be present. Usually in this type of case, if the patient survives, the eruption clears up without much scarring.

2. *The scaly type.*—This is by far the commonest variety, and is generally very chronic and localised, but may occasionally be acute and generalised. The lesions are chiefly seen on the nose, cheeks, ears and scalp, but are not uncommon on the backs of the hands. They are very apt to be distributed symmetrically in the shape of a bat's wing on the nose and two cheeks. The lesions are usually irregularly shaped red patches, often sunk below the surface of the skin, and covered with greyish scales, which are extremely adherent. When removed horny plugs are seen to penetrate into the epidermis, and when the patches clear up a depressed scar is left, often with numerous pits on its surface. When the scalp is attacked the hair is lost permanently. The mucous membranes may be attacked, the most frequent sites being the vermilion border of the lips and the palate.

Pain sometimes occurs in the patches and sometimes they itch, but generally no local sensations are present. The patient's health is usually below par, and there is often considerable neurosis, but severe constitutional symptoms are usually absent.

The course is exceedingly chronic, the patches often persisting for years in spite of treatment.

Diagnosis.—The condition is differentiated from lupus vulgaris by the absence of nodules, the symmetry of the lesions, the absence of ulceration and the age of the patient; from erysipelas, by the slowness of the spread and the absence of high fever; and from erythema multiforme, by the chronicity of the lesions and the presence of destructive effect on the skin shown by scarring.

Treatment.—The acute erythematous cases should be kept in bed and complications treated. All possible sources of focal infection should be removed. The drug which appears most to influence cases is quinine, which should be given in full doses. Local treatment is not usually required in the acute erythematous cases. In all cases, especially the very acute types, sulphapyridine is worthy of trial.

In the chronic scaly cases, quinine and general tonic treatment are indicated. Good results have been obtained by the intravenous injection of gold compounds, such as myocrysin, krysolgan, triphal, solganal, or sanocrysin. It is advisable to keep the doses of gold preparations small compared with those given for tuberculosis. Intramuscular injections of bismuth salts have also given good results. Rest in bed is always beneficial, and the patient should not be allowed to go out in a strong wind or in the hot sun, as these aggravate the condition.

Local treatment is chiefly directed to removal of the scales and the production of a mild inflammatory reaction in the patches. For the former *ac. salicyl.* ointment, 3 to 5 per cent., or plaster, 5 to 10 per cent., may be employed. For the latter 5 to 10 per cent. pyrogallie acid, painting with pure carbolic acid, or applications for a few seconds of CO₂ snow.

3. GONORRHOEAL KERATOSIS

The lesions in this condition are probably produced by gonococci circulating in the blood stream, although they have not been demonstrated.

They occur in patients suffering from gonorrhœal arthritis and other manifestations of general gonococcal infection, and usually appear on the palms and soles, though other parts of the hands and limbs may be affected.

The lesions are red patches covered with cone-shaped horny thickenings, and are generally numerous. In addition, a general hyperkeratosis of the palms and soles may occur.

Treatment.—General treatment for gonorrhœa is required, together with *ung. ac. salicylic.* locally.

4. SPOROTRICHOSIS

In addition to those bacterial conditions which attack the skin by way of the blood stream, a certain number do so by way of the lymphatics. Lymphangitis with abscess formation from pyogenic organisms is well known, and the same condition in tuberculosis has already been described under the title of "scrofuloderma." Actinomycosis is another such condition, and has already been dealt with (p. 185). Somewhat similar conditions to the

two last mentioned may be produced by certain fungi, of which the only one which requires special notice is sporotrichosis.

Symptoms.—Infection may take place through a crack in the skin, usually on the hand or foot. From this a lymphangitis starts, which spreads up the affected limb, and subcutaneous cold abscesses soon appear at points along the affected lymphatics. These eventually burst and leave indolent fungating ulcers, which show little or no tendency to heal spontaneously. A good deal of pus or yellowish fluid exudes.

Diagnosis.—Cases are usually diagnosed as tuberculosis, and a certain diagnosis can only be made by obtaining the fungus in culture. This should be done on Sabouraud's proof medium and incubated at room temperature.

Treatment.—The lesions usually disappear under large doses of potassium iodide administered internally.

ERUPTIONS DUE TO FILTRABLE VIRUSES

Certain affections of the skin are now known to be due to filtrable viruses. Among these are :

- (1) Herpes zoster (see p. 1583).
- (2) Herpes simplex.
- (3) Herpes preputialis.
- (4) Verruca vulgaris.
- (5) Molluscum contagiosum.

It is more convenient to deal with warts and molluscum contagiosum when discussing tumours of the skin (pp. 1481 and 1482), while zoster is described elsewhere (p. 1583).

1. HERPES SIMPLEX

Herpes Simplex or Herpes Febrilis is a condition to which some people, and especially children, are prone whenever they develop a slight febrile attack or even a slight cold.

Ætiology.—The disease is produced by a filter-passing virus which, when injected into rabbits, produces a fatal form of encephalitis, and is closely related to the virus of encephalitis lethargica.

Symptoms.—The lesions consist of small groups of vesicles, on an inflamed base, which come out chiefly in the neighbourhood of the mouth. They are irregularly distributed, have no relations to any nerve trunks, and are generally bilateral. They disappear in the course of a week or so, after crusting over, and leave no scars. In one type, seen especially in children, recurrent attacks occur on the cheek, often at regular intervals and without any special cause. These attacks also clear up and leave no scars. A recurrent type is also found affecting the buttocks in adults.

Treatment.—A bland protective ointment, such as zinc cream or Lassar's paste, or a dusting powder, such as bismuth subgallate, is all that is required. In recurrent cases, small doses of X-rays given during the quiescent period appear to diminish the liability to fresh attacks. Treatment by vaccines prepared from the virus have been tried, but results are uncertain.

2. HERPES PREPUTIALIS

This is the name given to small crops of two or three to half a dozen or more small vesicles which sometimes appear on the under surface of the prepuce. The vesicles quickly rupture and leave behind pinhead-sized ulcers which are painful. There is no tendency for these ulcers to increase in size. This latter feature helps to differentiate them from both syphilitic ulcers and soft sores.

Treatment.—This is the same as for Herpes Simplex.

ERUPTIONS DUE TO ERRORS OF METABOLISM

XANTHOMA

Xanthoma forms an interesting link between the inflammations due to chemical toxins circulating in the blood and those due to bacteria, for in this condition lesions of a granulomatous nature are produced around a deposit of a chemical substance in the tissues. Apart from this condition, all the granulomata whose nature is known are produced directly by bacteria; tubercle, syphilis and lepra are the best known examples.

Three clinical varieties of xanthoma are recognised: (1) xanthoma tuberosum, (2) xanthoma diabetorum, and (3) xanthoma planum.

Ætiology.—Xanthoma tuberosum and diabetorum occur in patients who for some reason or other have some disturbance of lipoid metabolism, often shown by an excess of cholesterol in the blood serum. This is why one form is seen in diabetics. The cholesterol becomes deposited in the tissues and causes a reaction, chiefly among the fixed connective-tissue cells of the dermis, particularly the endothelial cells, and a granuloma not unlike that seen in tuberculosis is produced. Histologically the tumours of xanthoma consist of large cells, arranged around the vessels, containing droplets of a cholesterol-fatty-acid-ester and some fat. Around these cells a varying degree of connective-tissue hypertrophy may occur.

Symptoms.—*Xanthoma tuberosum.*—In this condition numerous discrete yellowish nodules appear in the skin. These increase in size and may form tumours as big as an orange. They are most commonly seen on the extensor aspects of the limbs, especially on the elbows and knees, where they may form large firm tumours, but they may be seen on any part of the skin. The bones, tendons, viscera and mucous membranes may also be involved. The colour varies from a bright yellow to an orange or red. The disease is usually seen in young adults. It does not generally affect the general health, but is occasionally associated with jaundice. The lesions are very persistent.

Xanthoma diabetorum.—The lesions are usually smaller and more numerous; they are lentil-sized lesions and come out in crops, usually on the buttocks and extensor surfaces of the limbs. They disappear rapidly under appropriate treatment for diabetes.

Treatment.—In the diabetic cases, the underlying disease must be treated. For xanthoma tuberosum no definite treatment can be laid down, but a diet which contains as little fat as possible should be prescribed.

Xanthoma palpebrarum.—**Ætiology**.—This has been considered to be a fatty degeneration of the fibres of the orbicularis palpebrarum muscle, but in some cases an excess of cholesterin in the blood has been demonstrated.

Symptoms.—The lesions consist of flat, yellow, slightly raised patches, which are often symmetrically placed, on the eyelids near the inner canthus. They may be as small as a pin's head or may involve almost the whole eyelid. They produce no symptoms. They mostly occur in old people.

Treatment.—They can be destroyed by electrolysis or by caustics, or removed by excision.

C.—INFLAMMATORY DERMATOSES OF UNKNOWN ORIGIN

In this group are included certain dermatoses with well-defined characters which entitle them to be considered clinical entities, but whose ætiology is entirely obscure. The following diseases are included under this heading: psoriasis, parapsoriasis, pityriasis rubra pilaris, lichen planus, scleroderma and sclerema neonatorum. It must not be assumed that because these conditions are grouped together that they have any relationship to one another.

PSORIASIS

A very common condition characterised by the presence of red, scaly papules and patches of characteristic appearance on various parts of the body and unassociated with any disturbance of the general health.

Ætiology.—The disease frequently begins towards the end of the second decade, and is not infrequently seen in children from about 7 years of age and upwards, but is very rare in small children. Both sexes are equally affected. On the other hand, the first attack may occur in advanced age. It has been attributed to parasitic agencies, toxins of bacterial and metabolic origin, and to neuropathic causes, but there is very little evidence to support any of these views. There is no doubt that in some cases a strong family history can be made out.

Pathology.—Histological examination shows a great overgrowth of the epithelium, with downward growth of the interpapillary processes, and corresponding elongation of the papillæ. The horny layer is badly formed (parakeratosis), and collections of leucocytes can be found between the horny cells. There is a cellular infiltration around the papillary vessels and those of the subpapillary layer.

Symptoms.—The malady is a chronic one and may come and go throughout life. Usually attacks occur at quite irregular intervals, but in some cases they may appear at definite seasons—some appearing in the summer, others in the winter. The extent also varies greatly in different cases, some only having a few patches, others being covered with lesions.

The sites of predilection are the extensor aspects of the limbs—especially of the elbows and knees—the trunk—both back and front, but especi-

ally the waist region, the scalp and, more rarely, the face, nails and palms and soles.

The lesions begin as pinhead-sized papules, and are from their very beginning surmounted by a small silvery scale. The individual lesions usually spread centrifugally and may eventually attain great size. Usually, however, they join with other patches and so form plaques, which may, for instance, cover the whole back in one continuous sheet. The same type of scaling persists even in the largest patches, though in chronic treated cases the surface of these patches may appear to be highly polished; on scratching, however, with a sharp instrument the silvery scales are immediately apparent. The whole mass of scales can, with care, be removed in one continuous sheet, and underneath is found a shiny, dry red surface which, on examination with a lens, shows the dilated papillary vessels as tiny red points.

The arrangement of the lesions varies. In some cases the body and limbs are studded with lesions the size of a small pea or a threepenny-piece (psoriasis guttata); in others the lesions are larger (psoriasis nummulara); in some the centre of the lesions clears up, leaving rings (psoriasis circinata), and the rings may run together, forming gyrate figures (ps. gyrata). Occasionally the crusts are very thick (ps. rupioides), and this is especially the case on the scalp, where the hairs prevent the scales from falling off.

In some cases the lesions remain small and confined to the follicles (follicular psoriasis), and these may occasionally group into patches. They may also come out along scratches on the skin. When the nails are affected, either small pinhead-sized pits may be produced, or the whole nail may be forced up by lesions occurring in the nail bed, the nail eventually breaking up and thick masses of scales being found beneath it. The palms and soles are less frequently involved, but when they are affected circumscribed red patches form, associated with scaling and fissuring in the deep folds. In these regions also a pustular form of psoriasis has been described, consisting of sharply defined red patches studded with minute pustules, usually sterile, imbedded in the patches. The mucous membranes are not affected.

The lesions vary from a pale to a dark red in colour, and on clearing up usually leave little or no pigmentation, though in very chronic patches, especially on the legs, some pigmentation may remain for a time.

There are usually no subjective sensations, but occasionally itching is present. The general health is not affected.

Diagnosis.—This disease may resemble the secondary *papulo-squamous syphilide*. It differs, however, from this condition in the fact that the lesions are scaly from the start; that when the scales are removed no infiltration can be felt, and that the surface left is smooth, dry and studded with numerous small red points; that pigmentation is generally absent or little marked; that the lesions are mainly distributed on the extensor aspects of the limbs, and that the scalp may be extensively involved without loss of hair; and that other signs of syphilis, such as general adenitis and involvement of the mucous membranes, are not present. The Wassermann reaction and the effect of treatment will generally confirm a clinical diagnosis.

In *seborrhæic dermatitis* the scales are greasy, the patches spread by aggregation of follicular papules, and the scalp, face and centre of the chest and back are chiefly affected. The lesions respond quickly to sulphur, which is not the case with psoriasis.

In *eczema* itching is marked, when the scales are removed a moist surface is left, and the lesions are made up of aggregation of papules and papulovesicles.

In *psoriasis rosea* the scaling is usually in the form of a collarette, the lesions are of a pale pink colour, and the limbs are little affected, especially below the elbows and knees.

In *lichen planus* some typical papules can almost always be seen, the lesions have a characteristic lilac or purple colour, the flexor aspects of the limbs are most involved, and itching is generally intense.

Treatment.—Internal treatment is considerably employed, but it is difficult to estimate its value. Arsenic is the most valuable drug, and should be given in increasing doses up to the limit of tolerance, but should be entirely discarded if no effect is produced. Liquor arsenicalis, min. 3, t.d.s. and upwards, and pil asiatica are chiefly employed. Arsenic should not be given when lesions are coming out rapidly. In these cases salicin, gr. 15, t.d.s., and thyroid, gr. 1, t.d.s., have been recommended by Crocker and others.

Local treatment is the most efficacious, and chrysarobin gives the best results, but is messy, stains linen permanently, and is liable to set up a severe dermatitis if used carelessly. It is best applied in 5 per cent. to 10 per cent. ointment rubbed into the patches daily, after the scales have been removed in a hot bath with the aid of soft soap. Dithranol (derobin, cignolin), is a less messy chrysarobin substitute, and used as an ointment, in the strength of gr. $\frac{1}{2}$ to the ounce, is a very efficient remedy. If this treatment is carried out thoroughly for 3 or 4 weeks the patches will disappear. This treatment is best carried out in hospital or in a nursing home.

Pyrogallic acid ointment 10 per cent., oil of cade ointment 20 per cent., or an ointment consisting of solution of coal tar 12·5, ammoniated mercury 6·25, salicylic acid 6·25, simple ointment to 100. If the patches become inflamed they are best treated temporarily with linimentum calaminæ. Isolated resistant patches may be treated by X-rays, but this cannot be often repeated. For the scalp, the scales should be removed with soft soap, and the patches painted with equal parts of solution of coal tar and industrial spirit. This latter solution is useful in psoriasis of the nails, after the nail has been cut away and the scales removed.

PARAPSORIASIS

This is a term applied to certain rare forms of resistant erythematous lesions which occur on the body. Three types are recognised: (1) parapsoriasis en gouttes, (2) parapsoriasis en plaques, and (3) parapsoriasis lichenoides.

Ætiology.—Nothing is known of the ætiology of these conditions.

Symptoms.—*Parapsoriasis en gouttes* occurs as pea-sized or slightly larger red spots, covered by fine branny scales, chiefly on the upper part of the trunk. In some cases necrotic lesions occur.

Parapsoriasis en plaques occurs as symmetrical patches, oval or linear, of pale yellow or red colour, with a shiny surface or covered by fine scales, and occurring chiefly on the legs, thighs and lower trunk.

Parapsoriasis lichenoides forms a reticular pattern, chiefly on the extensor

aspects of the upper limbs. The lesions are red or purplish in colour and the surface is either shiny or covered with fine scales.

Treatment.—These cases are very resistant to treatment, but should be dealt with on much the same lines as psoriasis.

PITYRIASIS RUBRA PILARIS

This is a rare disease characterised by the appearance of follicular papules, with horny spines, which tend to involve the whole cutaneous surface and eventually produce a generalised dermatitis resembling pityriasis rubra of Hebra.

Ætiology.—This is very obscure. It is thought by some observers to be a follicular form of psoriasis, but at present there is no conclusive evidence. The disease occurs in both sexes and at varying periods of life, but sometimes in the very young.

Symptoms.—The lesions are of two types: red follicular papules with horny spines, which are chiefly seen on the extensor aspects of the limbs, and especially on the dorsum of the hands and fingers; and red scaly plaques or sheets, which involve the scalp, face and trunk. Either of these types may predominate. Where the former type occurs the skin presents the appearance of a nutmeg grater. Hyperkeratosis of the palms and soles develops, with fissuring of the deeper folds, and the nails become pitted and brittle. Ectropion followed by conjunctivitis may occur. The mucous membranes usually escape. The malady does not seriously affect the general health, and subjective symptoms are usually absent.

Treatment.—No specific treatment is known. Thyroid has been recommended, but the results are very uncertain. Bran and alkaline baths, followed by inunctions of ac. salicyl., grs. 10, glycer. amyl., adip. lanæ hydros., āā gr. 240, seem to give the best results.

PITYRIASIS ROSEA

A widespread eruption of pinkish macules and papules of round or oval outline, with branny scaling, which does not usually extend to the periphery of the lesion.

Ætiology.—This is still obscure. It was originally thought to be of parasitic origin, probably owing to the resemblance of the lesions to ring-worm, but no definite parasitic has been discovered. It is possible that it may be of the nature of an acute exanthem, as it occasionally has a sudden onset with slight fever and malaise. One attack is said to confer immunity, though this is not absolute, and it seems to occur at times almost in epidemic form. It has been thought by others to be a toxic eruption. The disease occurs chiefly in children and young persons, but no age is exempt.

Symptoms.—The eruption usually comes out suddenly, and the onset may, though this is not usual, be accompanied by slight fever, malaise and sore throat. In some cases the general outbreak is preceded by the appearance of a single patch, the "herald" patch, which may appear a week or 10 days before the general eruption. The lesions are usually most profuse

on the trunk and central portions of the limbs, the distal parts of the latter escaping. The face, neck and scalp may be affected, but this is not common.

The lesions are pinkish macules or papules, mainly macules, which vary in size from a lentil to patches a couple of inches across. They tend to be arranged in lines parallel to the ribs, and the larger patches are oval. When the lesions reach the size of a pea, central scaling commences, and as the lesions grow the scales tend to form a collarette, with the free edge directed towards the centre. The colour of the portion within the scales changes to fawn and eventually returns to the normal skin tint. Sometimes pinhead-sized follicular papules of skin colour are seen among the lesions in considerable numbers. In rare cases vesicles and bullæ form.

Subjective symptoms are usually absent, but sometimes itching is a prominent feature. The rash usually lasts about 4 weeks and then disappears, but it may persist for several months.

Diagnosis.—Seborrhœic dermatitis is distinguished by its greasy scales and by its distribution; secondary syphilis, by the presence of other syphilitic lesions; and ringworm, by the small number of lesions, their asymmetry, and the presence of fungus in the scales.

Treatment—No internal treatment is known to affect the disease. In the early stages a simple coal tar and lead lotion can be used, as this allays itching if present. Once the rash is fully developed a daily warm bath, followed by the application of 3 per cent. salicylic acid in linimentum calcis, will usually cause the lesion to disappear. If there is fever the patient should be put to bed.

LICHEN PLANUS

An intensely itchy eruption characterised by the presence of angula papules of pinkish or lilac colour tending to be localised in special areas.

Ætiology.—The disease occurs chiefly in adult life and is very rare in young children. Two views are held as to its ætiology. By some it is thought to be of nervous origin, following shock, mental anxiety, worry, etc. It is true such a history is often obtainable, but Graham-Little has pointed out that it did not appreciably increase during the War of 1914–1918, which might have been expected if such was the main factor in its production. The other view is that it is of toxic origin, but there is no direct evidence on this score.

Pathology.—The microscopic anatomy of the papules is very characteristic. There is a circumscribed, dense, round-cell infiltration in the upper part of the dermis beneath the papule, and the epidermis is much thickened. The papillæ are flattened out. The granular layer is irregularly thickened and there is a hyperkeratosis, most marked at the orifices of the hair follicles and sweat ducts.

Symptoms.—The most common variety is the *localised type*. The lesions are chiefly found on the flexor aspects of the forearms and wrists, the inner aspects of the thighs near the knees, and on the front of the shins. The trunk, especially the lower part, the palms and soles, and the penis are also occasionally involved. The mucous membrane of the mouth is frequently attacked. The lesions on the skin are discrete, lentil-sized papules, raised

sharply from the skin, with polished shiny surface and usually of lilac colour. They have a curious and typical angular outline, due to the fact that they are bounded by the fine lines of the skin, and sometimes they are definitely umbilicated. Frequently the papules are arranged in lines along scratch marks. Occasionally patches are formed by the aggregation of papules and resemble rather closely patches of psoriasis. On the palms the lesions are generally circular, vary in size from a pea to a threepenny-bit, and the horny layer over them is much thickened.

In the mouth irregular dead white patches are found, usually on the tongue or inner aspect of the cheeks.

Several other types of lichen planus are seen. In one variety the lesions form rings and gyrate figures (*lichen planus annularis*), while in another atrophy occurs (*lichen planus atrophicus*). In a large number of cases the horny layer is much thickened (*lichen planus hypertrophicus*), and this is especially seen on the legs, where warty patches occur (*lichen planus verrucosus*). Occasionally the lesions are arranged in a single line, sometimes following the course of a nerve (*lichen planus linearis*), and very occasionally bullæ and vesicles may occur. Rarely cases are met with in which the papules are few in number, and very much hypertrophied, forming dome-shaped tumours, which itch intensely (*lichen obtusus*).

Another variety is an *acute generalised* type in which a large number of lesions are scattered diffusely over the trunk and limbs. In this type the papules are pale pink in colour and not so raised as in the chronic forms. Even in these cases the face and scalp almost invariably escape.

In association with the above-mentioned lesions small groups of follicular papules with horny spines may be found. The same condition is sometimes found independently of lichen planus and has been called *lichen pilaris* or *spinosus*. Whether these latter cases have the same origin is still unsettled. Graham-Little has reported the association of this type with atrophic alopecia.

Lichen planus lesions are almost invariably accompanied by intense itching and often by marked neurotic manifestation, but otherwise the health remains good, though slight fever may accompany the acute cases.

The disease runs a very chronic course and is sometimes very resistant to treatment.

Diagnosis.—This is usually easy, as the lesions are very characteristic. The diagnosis from psoriasis has already been dealt with. From lichenification (*lichen simplex chronicus* of Vidal), which is produced by friction on the skin, the diagnosis is made by the fact that the latter only occurs in circumscribed patches, and that the typical discrete papules of lichen planus are absent.

Treatment.—In the acute generalised cases rest in bed is essential, and it is of the greatest service in chronic cases, materially shortening their course. Arsenic is looked upon as a specific, but may require to be pushed; arsphenamine (salvarsan) has been recommended by some authors. As in psoriasis, arsenic is not advised in cases in which the eruption is coming out, these cases doing best on mercury; liq. hydrarg. perchlor., min. 60, t.d.s. If the irritation is very bad, bromides or some hypnotic at night may be required, and for this symptom lumbar puncture has been recommended.

For local treatment anti-pruritic lotions and ointments are required; of these ichthammol, oil of cade, coal tar, phenol and menthol are most useful.

For the hypertrophic patches, ac. salicylic plaster, followed by X-rays or CO₂ snow, is the most satisfactory method of treatment.

SCLERODERMA

This is a condition of hardness and rigidity of the skin, caused by a degeneration of fibrous tissue, which is probably of inflammatory origin. It is met with in two forms—(1) Generalised scleroderma, and (2) localised scleroderma or *morphœa*.

Ætiology.—It is chiefly a disease of young adult life and is more common in women than in men. It has been variously attributed to a tropho-neurosis, to alterations in endocrine secretion, to an endarteritis, and to a primary hyperplasia of the fibrous tissue of the skin. Recent work by Dowling and Griffiths suggests that a relationship exists between this disease and thyrotoxicosis.

Pathology.—There is a degeneration of the fibrous tissue bundles in the dermis and subcutaneous tissue, with replacement of fat by fibrous tissue in the latter. There is also an exudation of cells around the vessels of the dermis, with some endothelial proliferation. The epithelium may be flattened by pressure and excess of pigment may be present. Degenerative changes may also be seen in the muscles in some cases.

Symptoms.—1. *Generalised scleroderma.*—This condition may appear rapidly or slowly. In both cases a disturbance of the general health, such as fever, joint pains, neuralgia or itching, may precede or accompany the attack. Stiffness of the parts involved is often the first symptom, and this may spread rapidly or slowly till it produces fixity of the joints, followed by progressive wasting of the muscles. Breathing may become difficult, owing to fixation of the skin of the chest, and taking of solid food may be prevented by the involvement of the cheeks and mouth. The skin appears swollen and glossy and is very hard; the deeper structures are fixed and the furrows of the skin disappear. The colour of the skin may be normal or waxy in appearance. The lesions are usually symmetrical and the mucous membranes may be affected. These cases may occasionally clear up spontaneously, but often end fatally.

Occasionally the disease begins in the hands. The skin is drawn tightly over the fingers, fixation of the joints occurs and atrophy supervenes, so that the fingers become pointed. This type is called *sclerodactylia*.

2. *Localised scleroderma or morphœa.*—In this type the lesions vary from the size of a pea to large patches involving almost all the back or front of the trunk. Patches may be pinkish in colour and raised, with a smooth polished surface and with a sensation of rigidity; or slightly depressed below the surface, very rigid and fixed, and often surrounded by a lilac border, and occasionally occurring in bands; or again they may be of dead white colour, with more or less irregular edges, and of normal consistence. This latter type is one of the forms of so-called *white-spot disease*.

These cases run a very chronic course, often of many years, and are resistant to treatment.

Diagnosis.—From sclerema neonatorum, by the age of the patient and the fact that this condition is confined to the subcutaneous fatty layer.

Treatment.—In generalised cases, the patient should be kept warm, and massage and hot air baths given. Cod-liver oil internally is of value. Thyroid is largely given in all varieties, but its action is very uncertain. In morphœa, local fibrolysin injections have been given with success. X-rays are claimed to be beneficial. Treatment, however, is unsatisfactory.

SCLEREMA NEONATORUM

This condition has no relationship to the foregoing. It occurs in newly-born infants, and is characterised by hardening of the subcutaneous fatty layer in certain parts of the body.

Ætiology.—It has been attributed to hardening of the fat, owing to lowering of body temperature, but this is certainly not an essential factor. There is evidence of a deposit of crystals in the tissues, and of a well-marked proliferation of the reticulo-endothelial cells in the neighbourhood of these deposits. This is probably due to a reduction in the olein in the fat, but how this comes about is not yet known.

Symptoms.—The affection usually begins within a day or two of birth. It occurs symmetrically and chiefly affects the calves, thighs, buttocks and back. The subcutaneous fat becomes very hard, and does not pit on pressure. The edges are well-defined. A certain number of cases die, but in the less severe cases the patches disappear in a month or two. A generalised hardening of the fat occurs all over the body in infants suffering from severe diarrhœa, but this condition appears to have no relation to that just described.

Treatment.—The child should be kept warm and given plenty of nourishment. Cod-liver oil is said to be very beneficial.

D—THE LYMPO-GRANULOMATA

These cases form a bridge between the inflammatory dermatoses on the one hand and the new-growths on the other. The lesions in many ways resemble the granulomata produced by bacteria, and in other ways resemble sarcomata. The following conditions are included: (1) Sarcoidosis, (2) leukæmia cutis, (3) lymphadenoma cutis, and (4) mycosis fungoides.

SARCOIDOSIS

Synonyms.—Besnier-Boeck-Schaumann's Disease; Lympho-granuloma benigna (Schaumann).

Dermatologists have been familiar for many years with two conditions of the skin which from their histological characters have been thought to be tuberculous. The first, which consists of a symmetrical granulomatous condition affecting chiefly the nose, cheeks, ears and fingers, was described by Besnier in 1889 under the name "lupus pernio," and differentiated by him from lupus vulgaris and lupus erythematosus, with both of which conditions

it has some features in common. The second condition, described by Boeck in 1897, was characterised by the presence, generally on the face, of granulomatous swellings, single or multiple, of varying size and of a translucent appearance, often showing small yellowish points closely resembling the "apple jelly nodules" seen in lupus vulgaris. To these lesions Boeck gave the name of "cutaneous sarcoids." In 1917 Schaumann showed that these two conditions were only clinical types of the same disease, and he further showed that they were merely cutaneous manifestations of a pathological process which affected many other organs in the body, the lymphatic system, bone-marrow, lungs and other viscera. It is now recognised as a diffuse reticulo-endotheliosis, somewhat resembling, though clearly distinct from, Hodgkin's disease. Schaumann has suggested that the condition should be called "lympho-granulomatosis benigna," but at the present time the name "Sarcoidosis" has been tacitly adopted in this country.

Ætiology.—The condition is not very common in the British Isles but more frequent on the European continent, especially in the Scandinavian countries. It affects both sexes equally, and is most frequent between the age of 20 and 40. It is very rare before puberty but isolated cases have been described even in infancy.

There is much controversy as to the cause of the condition. The histological architecture, described below, has long suggested a tuberculous ætiology, but certain features have raised doubts on this point. The absence of caseation in the lesions; the fact that the vast majority of the cases give negative tuberculin reactions; that acid-fast bacilli have not been found in the lesions, except in one or two cases in which the clinical diagnosis was by no means certain; and the very indecisive results of animal inoculations experiments—these are points which make the acceptance of the tuberculous hypothesis difficult. Schaumann has obtained the bovine tubercle bacillus from the sputum of some patients suffering from this disease, and has pointed out that active tuberculosis sometimes develops in cases of sarcoidosis and that when this happens the characteristic lesions disappear. He holds the view that sarcoidosis is an indication of a particular phase of immunity to the bovine bacillus, and supports the view of Jadassohn that the high percentage of negative tuberculin reactions is not due to passive anergy, indicative of an absence of tuberculous infection, but is due to an active anergy occurring in a certain phase of this infection.

Certain South American dermatologists have noted the close resemblance of sarcoidosis to tuberculoid leprosy, and have suggested that it may be a form of leprosy. The geographical distribution of the two diseases, however, does not correspond, and this seems to rule out this hypothesis. Lastly other observers think that the disease is an entity due to specific organism not yet discovered. This theory is at present largely based on negative evidence. It is only possible at the present time to say that the ætiology of sarcoidosis is unknown, but that it is difficult to ignore certain similarities to tuberculous infections.

Pathology.—Although the pathological changes were first only described in skin lesions, it is now known that similar changes may occur in many organs of the body. They have been found in the mucous membranes, the lymphatic system, the lungs and other viscera, the bone-marrow and certain other structures. The lesions consist essentially of nodules of epithelioid cells,

often though not always showing a few giant cells in the centre, surrounded by dense bands of normal connective tissue. Sometimes a zone of lymphocytes is found around the epithelioid cells, but these are often very sparse. Fibrous and elastic tissue is entirely destroyed by these granulomatous deposits, and bone is absorbed when invaded. The nodules show no signs of undergoing necrosis or caseation, but in older nodules fibrous tissue infiltration may occur; in healing nodules the cellular structure is gradually replaced by fibrous tissue.

As is mentioned above a very high percentage of cases show a negative tuberculin reaction, a percentage higher than is found in a series of apparently healthy individuals, and this has suggested the possibility of an active anergy and is thought by Jadassohn to be due to the presence of excess of anti-cutins in the blood and indicative of a tuberculous infection.

The blood picture usually shows little abnormal, but a monocytosis has been described in some cases.

Symptoms.—*Skin.*—Skin lesions are of several types and can roughly be classified into (1) the small nodular sarcoid, (2) the large nodular sarcoid, and (3) lupus pernio. In the small nodular sarcoid the lesions come out more or less symmetrically, and are most common on the face but may be found, often in considerable numbers, on other parts of the body, especially the shoulders and upper limbs. The nodules vary in size from a pinhead to a pea, are smooth dome-shaped papules of yellowish-brown colour, translucent in appearance.

The large nodular sarcoids are often present singly or in small numbers, and are not always symmetrical in arrangement. Again, the face is the site of predilection, the forehead, nose and prominences of the cheeks being the usual sites, but they are found also on other parts of the body. The lesions vary in size from a pea to plaques an inch or so across. They are raised above the skin, smooth and shiny in appearance, reddish to purplish-brown in colour, and soft to the touch. On glass pressure they exhibit translucency, and often "apple jelly" nodules are visible. These differ from those of lupus vulgaris in not breaking down under pressure from a pointed match.

Both the small and large sarcoids can disappear spontaneously or as a result of treatment, and in this case some atrophy of the skin results. In the large variety annular lesions, with a pale atrophic centre and a flattened purplish margin, are sometimes met with.

In lupus pernio the lesions form less well-defined infiltrations arranged more or less symmetrically, the face again being most affected. The nose and cheeks show marked swelling with ill-defined outline and are of a dull purple colour, similar to that seen in chilblains, though the skin is warm and not cold. The ears are also swollen and purplish in tint, especially the lobules. In this condition the skin of the hands and feet are also frequently affected, similar ill-defined purplish swelling involve the fingers and toes, and dorsum of hands and feet; this infiltration may extend up the forearms and legs. Lesions of the large and small sarcoids may be present at the same time. In these cases infiltrations of the subcutaneous tissue are sometimes present, especially on the forearms and arms.

Rare cases of generalised erythrodermia have been described in sarcoidosis. The skin becomes red and scaly over considerable areas of the body, or the eruption may be universal. A similar type of eruption is some

times seen in lymphadenoma or leukæmia. Nodules and diffuse infiltration of the mucous membranes, chiefly in the nose, but also in the pharynx, larynx and mouth have also been described.

Lymphatic system.—Palpable enlargement of accessible lymphatic glands is often present, though this enlargement may be limited to certain glands, of which the epitrochlear are the most common. When examined microscopically these glands reveal the same changes as are seen in the skin. Enlargement of the peribronchial glands is even more common, and can usually be seen in a radiogram of the chest. Of other lymphatic tissue the tonsil is frequently affected, and biopsies of the tonsil are sometimes useful for diagnostic purposes.

Bones.—In many cases of lupus pernio, swellings of the fingers and toes suggestive of tuberculous dactylitis have been observed. These, however, never showed any signs of breaking down. Radioscopic examination has shown that these swellings are mainly due to deposits in the connective tissue surrounding the small bones of the fingers and toes, but it has also demonstrated that in the bones themselves areas of rarefaction and cystic spaces can be seen, sometimes so extensive that the surrounding compact bone of the phalanx may collapse. This change, which may occur in cases where the fingers look quite normal, affects mainly the phalanges and heads of the metacarpal and metatarsal bones, but may occasionally occur in the long bones and even the spine is known to have been affected. These changes are of considerable diagnostic importance.

Lungs.—Patients suffering from sarcoidosis rarely show any symptoms of pulmonary involvement, such as cough or expectoration. Since, however, radioscopic examination of the chest has become a routine, changes have been demonstrated which are more or less diagnostic of the condition. In addition to enlargement of the hilar glands, mentioned above, most cases reveal evidence of deposits of granulomatous tissue in the peribronchial regions. These changes take the form either of a diffuse mottling due to the presence of small nodules scattered along the bronchi, or a reticulation or marbling, showing a more diffuse infiltration along the bronchi. In no case is there evidence of cavity formation.

Other viscera.—Lesions in most of the viscera have been described. Of these the spleen is most frequently affected and may become much enlarged, reaching down to the iliac crest. The liver may also be enlarged but usually to a less degree. Lesions have also been found in the heart, stomach and kidneys. In the case of the last, albuminuria and hæmaturia may be present.

Nervous system.—Involvement of the peripheral nerves, giving rise to localised anæsthesia, paresis and muscular atrophy, has been recorded. Lesions have also been described in the brain and in the pituitary body.

Ocular lesions.—Small yellow nodules have been found on the conjunctiva but special interest attaches to lesions of the uveal tract. These may take the form of an iritis; of nodules on the iris; of exudate with the anterior chamber; and opacities in the vitreous. White patches have also been observed in the choroid. These eye changes may be found in association with infiltration in the salivary glands, especially the parotid, and the condition known as uveo-parotitis or Heerfordt's disease is now known to be a manifestation of sarcoidosis (see p. 532).

General symptoms.—Patients suffering from sarcoidosis generally appear

to be in remarkably good health. Fever is generally absent but in rare cases persistent fever is present. It is not usually high, about 100° F., but may continue without intermission for months. The pulse in these cases is scarcely raised and the patient's general condition is good.

It should be remembered that in most cases only a few of the lesions mentioned above may be present, and that although the skin lesions are the most prominent numerous cases are now on record in which skin lesions are absent.

Diagnosis.—In cases where skin lesions are present they are usually sufficiently typical to enable a diagnosis to be made, but a biopsy can be done to confirm this. The presence of characteristic lesions seen in radiographs of the chest and hands mentioned above are of further assistance, as is a negative tuberculin test. The absence of necrosis and caseation is a useful sign in differentiating from classical tuberculous lesions, while the absence of follicular plugging distinguishes these cases from lupus erythematosus. The diagnosis from syphilis can be made by a careful consideration of the characteristic symptoms of that disease, assisted by a positive Wassermann reaction.

Prognosis.—It is generally held that the milder types of the disease eventually clear up spontaneously. According to Schaumann, however, some cases eventually develop frank tuberculosis, and in these the symptoms of sarcoidosis disappear. Severe cases may die from involvement of vital organs.

Treatment.—The best results have so far been obtained by the administration of chaulmoogra oil preparations or sodium morrhuate. The latter drug is best given either intravenously or intramuscularly in a 3 per cent. solution: 1 to 3 c.c. being administered weekly. It may be necessary to give the drug over a considerable period of time. There can be no doubt that lesions gradually disappear under this treatment but there is some tendency to recurrence, just as there is in the treatment of leprosy by the drug. Milder cases seem to be cured, but it is not easy to say the same of the more advanced cases. Chaulmoogra oil preparations are administered in the same doses as for leprosy. Arsenic also appears to have some influence on the lesions, and is best given in the form of sulpharsphenamine intramuscularly, doses of 0.3 to 0.45 gm. being administered weekly in courses of 10 injections.

No local treatment has much influence on the lesions, but general arc-light baths appear to be of value.

LEUKÆMIA AND LYMPHADENOMA CUTIS

Symptoms.—In both these conditions itching may be a marked symptom; sometimes it occurs without any cutaneous lesions, while at other times very persistent urticarial or prurigo-like lesions are present. Hæmorrhages may occur into the skin, and in some cases exfoliative dermatitis is present.

The more characteristic lesions are, however, granulomatous infiltrations of the skin, which form tumours, either in certain localities or more or less all over the skin. In the former case the face is most affected, the tumours forming chiefly on the forehead, about the nose and on the cheeks, producing a leonine appearance. The lesions vary from a pea to an orange in size, and

are usually of a dull purplish-red colour. Most of the cases recorded occur in lymphatic leukaemia, but a few have been reported in the myeloid cases. They also occur in Hodgkin's disease, but there is great difficulty in distinguishing aleukæmic leukaemia from Hodgkin's disease, unless the glands have been examined microscopically.

Treatment.—This has been dealt with in the articles on Leukæmia and Hodgkin's disease (pp. 802 and 841).

MYCOSIS FUNGOIDES

A chronic inflammatory dermatosis with a tendency to form granulomatous tumours, which usually ends fatally.

Ætiology.—It is a disease of late middle life, and more common in men. Its cause is quite unknown, but it is probably an infective process and closely related to leukaemia, though no characteristic blood changes have been observed.

Symptoms.—In the early or premycotic stage the most frequent lesions are patches of redness and scaling, associated with intense itching. After a time these lesions become infiltrated and raised above the surface of the skin. Later, tumours appear in these patches, usually about the size of an orange, but not infrequently much larger. The epidermis over them gives way and a fungating mass is produced. These tumours are usually multiple.

Sometimes the initial lesion takes the form of an eczema, an urticaria or a dermatitis exfoliativa, but in all these cases itching is a prominent symptom. In other cases the tumours appear without any pre-existing dermatosis.

The course is slow, and the general health is affected first by loss of sleep and then by septic absorption. Practically all cases eventually terminate in death.

Diagnosis.—This may be very difficult in the premycotic stage. The itching and the persistence of the symptoms in spite of treatment, together with the age of the patient, will help in coming to a diagnosis.

Treatment.—The only treatment known to benefit these cases is X-rays or radium. Either of these will keep the lesions quiescent for a considerable time, but recurrence generally takes place sooner or later. Arsenic and antimony may also be given.

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V. TUMOURS OF THE SKIN

Tumours can be divided into epithelial and connective-tissue tumours, and each of these varieties into benign and malignant.

BENIGN EPITHELIAL TUMOURS

1. WARTS

These are benign epithelial tumours, characterised by an overgrowth of the prickle-cell layer, with or without hyperkeratosis, and produced by an

infective agent, which appears to be a filter-passing virus. The following types are recognised :

1. *Verruca vulgaris*.—This is the common wart which is so frequently met with on the back of the hands, but may occur on the face and other parts of the body. The lesions are raised tumours, varying in size from a pinhead to a filbert, and are usually discrete, but may group to form larger swellings. They have a rough surface, rise sharply from the surrounding skin, and are skin-coloured. Histologically they show great hypertrophy of all the epithelial layers, with downward growth of the interpapillary areas, and a corresponding papillary elongation. They occur chiefly in children. They are inoculable from one spot to another, and from one individual to another.

Treatment.—Isolated lesions are best removed by the application of CO₂ snow for from 15 to 40 seconds, according to their size. They may be burnt away with glacial acetic, trichloracetic, or nitric acid, silver nitrate or the actual cautery. When very numerous, evulsion with a sharp spoon or X-rays are most satisfactory.

2. *Verruca plana juvenilis* are pinhead-sized warts, seen chiefly on the face and hands of children, though they are met with in adults. They have smooth, flat tops, and are usually very numerous.

Treatment.—They are best treated by touching with the galvanocautery, or by magnesium ionisation.

3. *Verruca plantaris*.—This is a wart which occurs on the sole of the foot, and has the appearance of a corn, because it is surrounded by a hyperkeratotic ring, and on account of the pressure on the foot does not stand up above the level of this ring. It is, therefore, often mistaken for a corn. It is usually extremely painful.

Treatment.—The most satisfactory treatment is by X-rays; 1½ to 2 skin units are usually required, the surrounding zone being carefully screened. These warts may also be removed by salicylic acid plaster, followed by touching with acetic acid, by CO₂ snow, or by evulsion.

4. *Verruca filiformis*.—These minute warts are usually about 1 mm. in diameter at their base, with long filamentous processes. They are sometimes found on the face; but chiefly occur on the genitals, and around the anus. They frequently occur in very large numbers, forming cauliflower-like growths. They are often seen in cases of gonorrhœa, and are sometimes known as *gonorrhœal warts*; but they may be present apart from this disease, and are probably due to some other infecting agent. In these warts there is very little overgrowth of the horny layer.

Treatment.—Locally antiseptic applications, such as 1 in 1000 perchloride of mercury in spirit painted on frequently, or silver nitrate 3 per cent. in sp. æth. nitros., will cause them to dry up, or they may be removed with the galvanocautery, or painted with trichloracetic acid.

5. *Keratoma senile*.—These co-called senile or seborrhœic warts are commonly seen on the face, back and chest of old people; but may occur in younger persons. They vary in size from a pea to a filbert, and are only slightly raised from the skin. They are soft to the touch, and have a slightly warty surface. Their colour varies from yellow to a deep black, and they may itch a good deal. The distribution is much the same as for seborrhœic dermatitis.

Treatment.—Washing with soap and water, followed by the application

of 10 per cent. ac. salicyl. ointment will often remove them. Failing this, painting with trichloroacetic acid, freezing with CO₂ snow, or a skin unit of X-rays should be employed.

2. ACANTHOSIS NIGRICANS

This is a rare condition in which warty pigmented growths appear on the neck, axillæ, groins, umbilicus, and flexures of the limbs and on the face. These growths often fungate and suppurate, especially in moist areas. In addition areas of pigmentation and scattered warty growths may occur. The mucous membrane of the lips, cheeks and tongue may be affected. In a large proportion of cases abdominal malignant growths have been found; but in other cases no such complication exists. The nature of the condition is not understood.

Treatment.—No treatment is known to affect the condition.

THE DYSKERATOSES

1. MOLLUSCUM CONTAGIOSUM

In this condition small tumours appear on the skin which, like the warts, are infective and produced by a filter-passing virus. They differ from warts not only in clinical characters, but in the peculiar degenerative changes which occur in the process of horny cell formation.

Pathology.—The most striking feature in a section is the presence between the Malpighian and horny layer of large cells containing large transparent oval bodies. These are known as "molluscum bodies," and at one time were thought to be coccidial bodies, but are now considered to be degenerations of the cell protoplasm.

Symptoms.—The lesions consist of small lentil- to pea-sized bodies of a white or pinkish colour, with a smooth glistening surface. They may appear anywhere on the skin. They are dome-shaped, and have a central pit, in the floor of which the thickened horny layer can be seen. Not infrequently they become inflamed.

Treatment.—By taking a sharpened match, and introducing it into the central depression, the whole horny mass can be forced out, and if the cavity be painted over with pure carbolic acid a cure will result.

2. DARIER'S DISEASE

This is a very rare condition of the skin usually seen in young adults in which an eruption of follicular papules develops on the face, scalp, abdomen, back, and the flexor aspects of limbs. The lesions run together and form warty-looking masses. The disease is slowly progressive, and the lesions are resistant to treatment, but the general health is not affected.

Microscopic examination shows similar changes to those seen in molluscum contagiosum, namely, an irregular hyperkeratosis with formation of "psorosperms" in the region of the granular and Malpighian layers of the epidermis.

Treatment.—This consists of baths and the application of keratolytic agents, such as salicylic acid.

3. PAGET'S DISEASE OF THE NIPPLE

This is a chronic affection which usually develops around the nipple in middle-aged women, but has been described in other parts of the body and in men. It is seen as a sharply defined, red, oozing area involving the nipple, the areola, and the skin around for a short distance, and is almost always unilateral. The whole area has a distinct parchment-like induration. The nipple becomes retracted, and eventually disappears. The condition is associated with carcinoma of the breast, and whether it is primary or secondary is still a matter of dispute.

In this condition, as in the two diseases just referred to, "psorosperms" are seen under the microscope. The surface horny layer is mostly lost, and the deeper layers of the epidermis are much hypertrophied and cedematous, but show no obvious epitheliomatous proliferation.

Treatment.—Amputation of the breast is the only treatment that can be advised.

MALIGNANT EPITHELIAL TUMOURS

1. RODENT ULCER

A slowly growing epithelioma usually single, but sometimes multiple, which may cause considerable local destruction of tissue but does not form metastases.

Pathology.—This variety of epithelioma is usually described as a basal-cell epithelioma. In section epithelial processes are seen penetrating into the underlying dermis and subcutaneous tissue; but the processes are bounded by a regular basal layer of cubical cells, and although degeneration cysts may form in these processes no cell-nests are formed.

Symptoms.—The lesions chiefly occur in old people; but this is not always so. They also rarely begin anywhere but on the face, and then chiefly in the neighbourhood of the eye, or on the nose or cheek. At first a small raised white nodule appears, with small vessels coursing over it. Then, as it spreads, an ulcer forms in the centre, but the raised intensely hard white border persists. If not treated a great deal of tissue destruction occurs; the nose may be destroyed, or the antrum perforated, and the whole of the nasal cavities opened up. In advanced cases practically the whole face is destroyed. Some cases, however, remain superficial, spreading slowly, the older parts healing as the lesion spreads. Multiple lesions are not very rare.

Benign forms are also recognised. In one the lesions are pea-sized or slightly larger nodules, scattered over the face, and of the same type as the early lesions referred to above. Sometimes they undergo cystic change. They were described by Brooke under the name of *epithelioma adenoides cysticum*. Another benign type is seen in which multiple walnut-sized tumours form on the scalp (*Spiegler's tumours*). A third type occurs in the form of multiple psoriasiform patches on the trunk, and has been named by Graham-Little *erythematoid benign epithelioma*.

Diagnosis.—This can always be made in cases of doubt by microscopic examination.

Treatment.—Excision is the best treatment when possible ; but excellent results are obtained by radium, or X-rays. CO₂ snow has been advocated for early cases, and the results obtained are excellent. In advanced cases, which are unsuitable for surgical treatment, diathermy is useful.

2. SQUAMOUS EPITHELIOMA

In this condition rapidly growing tumours form, which ulcerate and cause local destruction of tissue, and also cause secondary glandular involvement. It is chiefly a disease of old age.

Pathology.—Sections show an irregular proliferation of the Malpighian layer, with the formation of cell-nests, and the limiting basal layer is absent.

Symptoms.—The lesions begin as nodules, much like those of rodent ulcer, but they spread much more rapidly and either form irregular deep cut ulcers, without the characteristic edge seen in rodent ulcer, or else they become raised and form mushroom or cauliflower-like growths. The glands may be involved, and general dissemination may occur. The condition may sometimes supervene on pre-existing non-malignant conditions. It may commence in a keratoma senile, in the warty conditions which occur in cases of atrophy of the skin due to exposure to tropical sun, on an old lupus scar, on X-ray dermatitis, in xeroderma pigmentosa, in arsenical keratoses and in tar molluscum.

Treatment.—This is purely surgical, and consists of erasion of the local growth and of the glands draining the area concerned. Radium is now extensively used in treating these growths. In the case of epithelioma complicating lupus vulgaris, however, secondary glandular involvement does not appear to occur, and local destruction with arsenic paste, or by diathermy, gives even better results than excision.

TUMOURS OF THE APPENDAGES OF THE SKIN

1. MILIUM

In this disease pinhead-sized yellowish-white bodies are seen in the skin of the face, chiefly on the cheeks, eyelids and forehead. They are often very numerous. They can be shelled out, and are found to consist of a whorl of epithelial cells. Their origin is unknown, but they are probably derived from the lanugo hair follicles.

Treatment.—These tumours can be destroyed by electrolysis.

2. SEBACEOUS CYSTS

These are painless cystic swellings chiefly found on the scalp, face, ears, back and scrotum. They vary in size from a pea to an orange. When incised they are found to be filled with cheesy matter. They are either due to blocking of the sebaceous duct, or according to some authorities they are of embryonic origin. True *dermoid cysts* of the skin are also found.

Treatment.—Excision is the most satisfactory method of treatment.

3. ADENOMA SEBACEUM

A symmetrical eruption of pinhead-sized, bright red papules, of congenital origin, on the face. It commences very early in life, and is often associated with mental defect—in fact, cases are most often seen in asylums. The lesions are distributed chiefly over the nose and cheeks, and consist of hypertrophied sebaceous glands and numerous capillary vessels.

Treatment.—The lesions can be destroyed with the galvano-cautery, by electrolysis or by diathermy.

Tumours of the sweat glands and ducts are so rare as to need no description here.

CONNECTIVE-TISSUE TUMOURS

1. KELOID

This is a fibrous tumour developing in a scar. The mere overgrowth of a scar is sometimes referred to as a *hypertrophic scar*, while the term keloid is limited to those cases in which the tumour extends beyond the original limits of the scar. In this latter condition processes often grow out in all directions like tentacles, and also in some cases the condition appears to start spontaneously from the normal skin; but there can be little doubt that some small abrasion was present. Small keloids often appear after acne vulgaris, varicella and other dermatoses which lead to scarring. There can be little doubt that this fibrous overgrowth is due to some chronic bacterial infection of the wound—probably a staphylococcal infection.

Treatment.—The best results are obtained by radium and X-rays.

2. FIBROMA—MOLLUSCUM FIBROSUM

Hard fibromata of the skin are rare, and usually occur in pear-sized nodules scattered about the skin. Soft fibromata are common, and are met with as small pedunculated tumours, chiefly on the trunk. They may occur in large numbers, and are then described as molluscum fibrosum. In this condition the tumours vary in size from a small pea up to several inches in diameter. Not only the skin but the mucous membranes may be the seat of these tumours. At times they form huge dependent unshapely masses, which completely disfigure the part from which they arise; this condition is called *dermatolysis*. Not all the tumours are pedunculated—as some are sessile—but all have the same softness. Some definitely surround nerve trunks, and it has been thought that they all develop in connection with the nerve fibres; hence they are often called *neuro-fibromata* or *plexiform neuromata*.

In some cases true neuro-fibromata and molluscum fibrosum lesions occur in combination with pigmented spots about the body. This syndrome is called *Recklinghausen's disease*, and is sometimes associated with mental disturbance.

Treatment.—Nothing can be done except surgical removal of the tumours, and this is only occasionally necessary.

3. LIPOMATA

These are soft freely movable lobulated tumours in the subcutaneous tissue, and may be single or multiple. One variety is very painful and associated with general adiposis, and is referred to as *Dercum's disease* (p. 1480).

Treatment.—Excision is the only treatment.

4. MYOMATA

Small multiple tumours of the size of a pea are sometimes found which have the structure of leiomyomata, and arise from the arrectores pilorum muscles. The lesions are often numerous, grouped and painful.

Treatment.—The cautery, or excision, is the only treatment.

5. MULTIPLE IDIOPATHIC SARCOMA OF KAPOSI

This is a curious condition chiefly seen in old people, and generally in the natives of Eastern European countries ; but cases have arisen *de novo* in this country. The lesions occur chiefly in the region of the ankles, but have also been found on the hands and on the trunk. They are irregularly shaped red plaques raised from the skin, and of firm consistence. Histologically they consist of an overgrowth of fibrous tissue with dilated blood spaces. Whether this condition is of inflammatory origin, or is a species of *nævus* is as yet undecided. The condition does not affect the general health.

Treatment.—No treatment is known to affect the condition.

6. SARCOMATA

Both round- and spindle-celled sarcomata have been found arising in the skin, but are rare. They may be single or multiple, of any size, sessile or pedunculated, and are usually of a purplish-red colour. They tend to break down and produce fungating ulcers, and run a rapid course ending in death unless removed in the early stages.

Treatment.—This is purely surgical.

7. NÆVI

This term should be applied only to certain new formations of congenital origin ; but in practice certain other conditions have been included. They fall into four classes : (1) Vascular *nævi* ; (2) lymphatic *nævi* ; (3) pigmented *nævi* ; and (4) hyperkeratotic *nævi*.

VASCULAR NÆVI.—There are two chief varieties, the capillary and the cavernous *nævi*.

Capillary *nævi*.—These *nævi* are essentially dilatations of the capillary vessels of the papillary and subpapillary layers of the dermis. They form flat red patches of varying size. They may be small pea-sized lesions, or they may practically cover the whole body, including the mucous membranes. When they occur in large patches they are called *port-wine stains*.

The lesions are usually not raised and not infiltrated, the only change being in the colour of the skin, which is red or purple in the affected areas.

Sometimes, however, thickenings occur irregularly throughout the patches. On examination with a lens the capillaries can often be seen. These *nævi* are either present at birth or appear shortly afterwards. They tend to get paler as age advances, but rarely disappear.

Treatment.—These cases are very difficult to treat, especially the more extensive ones. CO_2 is useless, unless the application is sufficiently long to destroy the skin. Radium, though sometimes successful in removing the *nævus*, is inclined to produce atrophy and telangiectases in its place. The best hope in disfiguring cases rests with excision and plastic surgery, though some fair results have been obtained with diathermy.

Cavernous *nævi*.—These are soft or hard tumours, which appear as bright red sharply defined swellings raised above the level of the skin or as purplish indurations in the skin and subcutaneous tissue. The blood can usually be squeezed out of them by pressure. They consist of a fibrous stroma surrounding irregular blood spaces, the whole being more or less encapsuled. The tumours vary much in size and extent, some being as large as a cricket-ball or larger. Any part of the body may be affected, but they are frequent on the face and scalp, the lips and eyelids often being attacked.

Treatment.—Small tumours are best treated with CO_2 snow, one exposure of 15 to 30 seconds being sufficient to remove them. Larger tumours may be dealt with by repeated applications of snow, but they do better with multiple punctures with a fine galvano-cautery at dull red heat. In some situations excision is the best treatment, while radium can often be used with success. Electrolysis was formerly much employed, but is slow and has been superseded by the methods mentioned above. It is often advisable to abstain for a time from active treatment, as there is a tendency for the tumours to disappear spontaneously.

Stellate *nævi*.—These are not strictly *nævi* at all—that is, they are not congenital growths. The cause is not clear, but they may be degenerations or possibly traumatic dilatation of venules. The lesions consist of a central pinhead-sized dilatation of a venule, with a stellate arrangement of dilated vessels running into it. They are seen chiefly in children on the face, but also occur in adults. It has been thought that insect bites may be a determining cause.

Treatment.—If the central vessel is destroyed by a fine galvano-cautery or by electrolysis the lesion will disappear.

LYMPHATIC NÆVI.—Synonym.—Lymphangioma Circumscriptum.

This occurs as a raised circumscribed patch of skin colour, which on close examination is seen to consist of closely grouped vesicles, varying in size from a pin's head to a lentil. There may be a few discrete vesicles surrounding the main patch. In some cases, too, the surface is warty. The patches appear at or soon after birth, but may come out later. Microscopic examination shows dilatation of the lymphatic vessels of the dermis, with or without epidermal hypertrophy.

Treatment.—Excision, cauterisation or treatment by radium are the three methods applicable.

PIGMENTARY NÆVI OR MOLES.—*Nævi* of this class are very numerous and vary considerably in type. They may consist of pigmented patches of varying size and various depth of colour from a pale yellow to a deep black. These may be associated with hairy growths. In other cases smooth lobulated

pigmented tumours may occur on any part of the skin. Some cases have a rough, warty surface, while others are hairy. They may be quite small, no larger than a pea, or may cover large areas of the body, and have a distinct tendency to occupy segmental areas. They may appear at or soon after birth, or may occasionally develop later in life. The histological picture is characterised in all types by the presence of masses or columns of round embryonic cells in the dermis and also in the deeper layers of the epidermis. There is excess of pigment in the cells of the basal layer, in the adjoining Malpighian layer, and also pigmented wandering cells in the upper part of the dermis. The epidermal changes vary with the type of nævus.

There is a slight tendency for these pigmented moles to undergo malignant transformation into *nævo-carcinoma*, which has a high degree of malignancy, giving rise to rapidly generalised metastases.

Treatment.—It is best to leave pigment moles alone unless some definite indication for treatment is present. Free excision with grafting or plastic procedures is indicated in some disfiguring lesions, or in those liable to irritation from friction. CO₂ snow, diathermy or electrolysis may be used in the case of the smaller tumours.

HYPERKERATOTIC NÆVI.—**Synonyms.**—Linear Nævi; Ichthyosis Hystrix.

In this type of nævus the lesions are arranged in lines or bands, usually on the limbs, and often appear to follow the course of certain nerves. They are frequently unilateral, though in the ichthyosis hystrix type they are frequently symmetrical. The lesions consist of thick horny plugs, which can be pulled out from depressions in the skin, and are closely packed together; sometimes great horns protrude from the skin. On microscopical examination an irregular hyperkeratosis is found, with alternating depressions and elevations.

Treatment.—This is very unsatisfactory. Salicylic acid plasters may be used to soften and remove the horny masses, and small areas can be excised or cauterised.

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VI. OTHER MORBID CONDITIONS OF THE SKIN

ANOMALIES OF PIGMENTATION

Pigmentation may be produced by the deposit of blood pigment in the skin, by the excessive production of melanin—the normal pigment of the skin, or by the deposition of such substances as silver, seen in poisoning by that substance.

Blood pigment is found after hæmorrhages have subsided and in congestive conditions.

Increase in melanin occurs in the pigmentary nævi already referred to; in certain internal diseases, such as Addison's disease, diabetes and exophthalmic goitre; and in pregnancy; after the ingestion of arsenic; and locally, after certain inflammatory conditions of the skin, such as sunburn, erythema ab igne, lichen planus and syphilis. It also occurs in a curious condition named vitiligo, the ætiology of which is obscure.

1. LENTIGO

This is the name given to *freckles*, which occur on parts of the body exposed to the sun in certain individuals. The lesions are so well known as to require no description.

Treatment.—This is purely preventive. The application of a greasy preparation, such as lanolin, to the skin before exposure to the sun will protect the face from an erythema; sunshades and veils, especially red or brown, are also useful.

2. CHLOASMA UTERINUM

This is a peculiar yellowish-brown pigmentation which occurs chiefly about the face in women who are pregnant, or suffering from some uterine disturbance. It occurs in ill-defined patches, chiefly on the forehead and on the abdomen. It disappears after the termination of pregnancy, or when the pelvic condition is rectified.

3. VITILIGO

This condition, also known as *leucoderma* and *melanoderma*, may occur at any age or in either sex. The ætiology is quite unknown, both toxic and tropho-neuritic theories having been invoked to explain the phenomenon, but very little evidence is at present forthcoming in favour of either view. It consists of patches of a dead white colour appearing in various parts of the body; they may be quite small, or may in rare cases completely cover the body. The edge of the patches is sharp, and the surrounding skin is hyperpigmented; the texture remains normal. In the white areas pigment is entirely absent, but no other histological changes can be observed. The patient's health is in no way affected, nor can any derangement of any of the organs of the body be made out in the majority of cases.

Treatment.—The only treatment which appears to have any effect is the repeated application of ultra-violet rays, either by the Kromayer lamp or by arc-light baths.

4. ALBINISM

This is a congenital condition in which there is complete absence of pigment in the skin and other epidermal structures. The hair is white, the eyes pink from absence of pigment in the iris, and the skin fails to pigment, even when exposed to the strongest sun.

ATROPHIES OF THE SKIN

Various conditions may cause atrophy of skin, particularly local inflammations, but under this heading certain atrophic conditions are dealt with that have not been considered elsewhere.

1. SENILE ATROPHY

Generalised atrophy of the skin occurs in old age. The skin becomes thin and loses its elasticity; irregular pigmented spots, small telangiectases

and vascular cysts (de Morgan's spots) appear, especially on the face and trunk; wrinkles are very numerous; and the skin develops a yellowish colour. A generalised pruritis may occur. Senile warts are frequently found, and these may be the seat of a localised pruritus. They occasionally become transformed into squamous epitheliomata.

Treatment.—This is purely symptomatic.

2. STRIÆ ATROPHICÆ

These are bands of atrophic skin which develop in areas where the skin has been much stretched. They are seen best on the abdomen, breast and hips of women who have borne children. The lines when first formed are red in colour and about $\frac{1}{2}$ inch in diameter, but as they get older they become greyish-white. It is thought that they are produced by damage to the elastic fibres of the skin by stretching.

No treatment is required.

3. XERODERMA PIGMENTOSA

This is a rare condition of the skin which is hereditary. The ætiology is quite unknown, but there is no doubt that light-rays play a part in the production of the lesions. The affection begins in infancy, and is characterised by the appearance on the face and backs of the hands of macules of yellow and brown pigmentation. The disease is slowly progressive, and in addition to pigmentation other signs of skin degeneration appear, namely, atrophic patches, telangiectases and warty growths. Later, ulceration occurs and epitheliomatous tumours appear on the warty growths.

Treatment.—The patient should be protected from the sun's rays as much as possible. The warty growths can be removed and the ulcer treated antiseptically. Epitheliomatous growths can be checked by radium, but the cases always end fatally.

CONGENITAL CONDITIONS OF THE SKIN

Most of these, such as ichthyosis, the nævi and xeroderma pigmentosa, have been already considered. There still remains one condition which has not been alluded to, namely, epidermolysis bullosa.

EPIDERMOLYSIS BULLOSA

This is a congenital defect of the skin which renders it extremely sensitive to the slightest injury. In those affected, the slightest knock is sufficient to produce a blister. The disease is hereditary, and can often be traced to a considerable number of members of a family. The lesions usually appear first in early infancy, but occasionally they have occurred for the first time later in life. They vary much in degree. In some cases the lesions are slight and cause very little inconvenience, and no disturbance to the general health. In other cases the lesions are numerous, almost all parts of the body being affected at one time or another; teeth and nails develop badly, septic com-

plications are often severe, and these cases usually do not live to adult age.

Treatment—Nothing can be done except by prevention of sepsis and the antiseptic treatment of the lesions when once formed.

DISEASES OF THE HAIR

1. ALOPECIA

Loss of hair occurs in many diseases. It may fall out after acute illnesses, such as influenza and typhoid fever, in inflammatory conditions of the scalp, in secondary syphilis, and in dermatitis exfoliativa. It is also lost locally in scarring conditions, traumatic or inflammatory, as seen in lupus erythematosus or tertiary syphilis. A progressive loss also occurs in senile atrophy of the skin; this, however, may occur prematurely.

ALOPECIA PREMATURA.—**Ætiology.**—The disease is essentially one of the male sex, and usually begins at about the age of 20. The ætiology of the disease is not clear. Two factors appear to be present, heredity and seborrhœa of the scalp. It is quite clear that the latter condition by itself does not always produce baldness, but it appears to accelerate the loss of hair, as might be expected. Heredity seems to be important, especially in those cases where complete baldness occurs at an early age, and there is no doubt that fine hair is more liable to fall out early than is stouter hair.

Symptoms.—This gives rise to a very characteristic type of hair loss which is familiar to every one. The hair gradually gets thin on both temples and on the vertex, and by slow progression these thinned areas eventually meet, leaving the top of the head entirely bald or only covered by a fine down, while the sides and back of the scalp are covered normally. The progress varies very considerably in different individuals, some becoming completely bald in a year or two, while others still have a good crop of hair at 50.

Treatment.—This has mainly to be directed to curing the seborrhœa, and the methods for doing this have been dealt with under that heading. Apart from this, avoidance of tightly fitting hats, and gentle massage with the fingers are the most appropriate remedies. Certain drugs such as pilocarpine, have been thought to have a stimulating effect on hair growths, and rubefacients, such as cantharides, are also much employed. The general health should receive attention.

ALOPECIA AREATA.—In this condition the hair falls out in patches, leaving smooth, shiny, bald areas. There is a general tendency for the hair to grow again.

Ætiology.—The malady affects both sexes and generally occurs in early adult life. It is probable that the disease is an inflammatory condition, but the nature of the irritant is unknown. A somewhat similar loss of hair can be produced by the administration of thallium salts, which lends support to the toxic theory. It was at one time thought to be due to an external parasite, but there is no evidence to support this view. It has also been thought to be of nervous origin, as damage to nerves has produced bald patches over the areas supplied.

Symptoms.—The disease may start suddenly or slowly. In some cases a large circular patch of baldness may occur in a single night, and in these cases the skin may be tender and reddened. Generally, however, a small bald patch appears, which spreads slowly, and other patches may subsequently arise, causing considerable loss of hair and a curious patchy condition of the scalp. Not only the scalp hairs but those of the beard, the eyebrows, axillæ and pubes may be affected, and in severe cases all the hairs of the body, including the eyelashes, may fall out. In one type a band of hair may be lost extending from ear to ear around the margin of the scalp, either in front or behind the head or even in a complete circle.

In the patchy form, the individual patches are characteristic. The centre is usually completely bald and shiny, though new downy hairs may be seen. Around this a row of stumps may be observed. These are club-shaped, like a note of exclamation, being very thin as they enter the scalp and thicker above. When pulled out, a shrunken hair bulb comes out and the hair does not break as in ringworm. The zone outside the zone of stumps looks normal, but if the hairs are pulled upon many loose hairs may be detached.

Course.—This varies in different cases. Usually new hair grows fairly rapidly, and the patches cease to spread. The new hair is usually white when it first appears, but pigments later. In some cases, however, the patches progress as new hairs grow and this may continue for many months. In the band-form hair growth is usually much slower. In the generalised cases the prognosis is not so good, a large proportion losing the hair permanently.

Diagnosis.—This has to be made from ringworm and is generally easy. In ringworm the patch is scaly and covered by stumps, which break easily and have an irregular fractured end. Under the microscope the fungus can be seen.

Impetigo contagiosa of the scalp sometimes gives rise to bald patches. They are numerous, small and usually red in colour, and no stumps are seen.

Treatment.—The general health must be looked to and all possible sources of irritation removed. The teeth must be attended to, tonsillar sepsis treated, and errors of vision corrected. General tonic treatment should be prescribed, and rest from overwork and worry ordered. Thyroid extract has been recommended, but in the writer's experience has sometimes made the condition worse.

Local applications which cause hyperæmia are of most value. Painting the patches with pure phenol, iodine or blistering fluid, or rubbing in oil of turpentine is useful. Various antiseptic lotions, such as perchloride of mercury and resorcinol, have been used with success. High frequency current and ultra-violet rays have given good results when other means have failed. If seborrhœic dermatitis is present it should be treated.

CICATRICAL ALOPECIA.—In this condition, also known as *pseudo-pelade* or *folliculitis decalvans*, progressive loss of hair takes place, and the scalp shows signs of atrophy or scarring. There is a progressive patchy loss of hair occurring over considerable areas of the scalp, and on examination inflammatory lesions are often present round the hair follicles. The denuded areas show obvious scarring, and hair does not regrow on the patches. The disease occurs chiefly in young adults, and though it does not usually lead to complete baldness, very disfiguring patches remain.

Treatment.—This consists in the application of antiseptic ointments and lotions, perchloride of mercury, resorcinol and sulphur being the most useful, but no treatment is very efficacious.

2. HYPERTRICHOSIS

This is the term applied to an excessive growth of hair. It is usually confined to those cases in which a growth of stout hairs occurs in sites usually covered with lanugo hairs, such as the face in women. This may sometimes be very excessive, and the "bearded woman" and the "dog-faced man" are extreme examples, though the latter are often cases of hairy moles. The only conditions that the medical practitioner is likely to have to deal with are those in which stout and dark hairs occur on the chin and upper lip in women. The treatment consists in removal of the hairs by electrolysis, but considerable judgment is often required to decide whether a case is suitable for treatment. Electrolysis consists in passing a current of about 1 milliampere for a quarter to half a minute into the hair bulb by means of a fine needle attached to the negative pole of a galvanic battery. The hair then loosens and can be removed. It is important not to remove hairs too close to one another at the same sitting, or troublesome scarring will supervene.

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VII. TROPICAL SKIN DISEASES

In the tropics many skin diseases occur which are met with in Europe; in addition, there are certain conditions peculiar to hot climates, and it is with these that the present section deals. At the same time it must be realised that skin diseases may be modified by racial immunity, social custom and skin pigmentation. Leucoderma has, for instance, a peculiar and sinister significance, not only on account of the great disfigurement it produces in dark-skinned people, but also because of its superficial similarity to the depigmented patches of nerve leprosy. Again, native custom may modify such conditions as keloid, which may assume a magnitude never experienced in Europe, owing to the fact that primitive people often purposely irritate wounds to produce tribal marks of a keloid nature.

CRAW-CRAW

A West African native name (Kra-kra) applied to any itchy, papular or pustular eruption of the skin. It originates as a papular dermatitis.

Ætiology.—O'Neill found filarial embryos in an eruption resembling scabies, but they were probably *Microfilaria streptocerca*, which Scott Macfie has since commonly found in the skin of West African negroes.

Symptoms.—The papules are hard and horny, occur chiefly in the limbs, and are very itchy: scratching and secondary infection lead to a pustular dermatitis with enlargement of adjacent lymph glands.

Diagnosis.—The condition must not be confused with scabies or coolie itch: no acari are obtained and no burrows seen.

Treatment.—Pustules are opened, ulcers scraped and crusts removed, then disinfected with 1 in 1000 sublimate solution and subsequently dressed with boric acid ointment. Carbolic lotion improves some cases.

PRICKLY HEAT

A form of miliaria associated with excessive sweating in hot climates.

Ætiology.—The condition quickly disappears in cold weather, and is possibly purely a mechanical process due to blocking of the sweat glands with sodden, inadequately cornified cells of the stratum corneum. Bacteria and yeast-like fungi have been incriminated, but these are probably secondary invaders.

Symptoms.—The red eruption consists of small, watery vesicles and inflamed, red papules which feel like grains of sand and may involve the trunk, limbs, forehead or almost any part of the body. The pricking sensations and great itching may be sufficient to prevent sleep.

Treatment.—The underclothes should be frequently changed (twice daily), and antiseptic soaps used in bathing. After a warm bath the application of corrosive sublimate solution (1 in 1000) containing eau-de-cologne is helpful. McLeod recommends the following lotion: R acid. salicyl., grs. xxx., hyd. perchlor. grs. ii., sp. vini rect. 3 ii., aq. dest. ad 3 vi. After this has dried a dusting powder such as zinc oxide, boracic acid and starch in equal parts, or boracic acid and menthol should be employed.

VELDT SORE

This is a chronic, septic, ulcerated sore met with in the tropics and sub-tropics, generally involving the exposed hairy parts of the body.

Ætiology.—The disease has a widespread geographical distribution in hot, dry, sandy or desert country, being known as barcoo rot in Northern Australia, and veldt sore in South Africa: during the war it became known as desert sore, and affected troops in the Near East, especially in Sinai and Mesopotamia. Vitamin deficiency may be a predisposing factor, for there is often a history of living on tinned foods and an absence of fresh fruit and vegetables. Organisms isolated from the lesions include streptococci and diphtheria bacilli; staphylococci are also present, but these are probably surface contaminants. Horse manure may constitute a source of origin for the infecting organism.

Symptoms.—The lesion commences as a painful vesicle, containing yellowish fluid, on exposed parts of the skin, especially the dorsum of the hand, forearm, elbows, knees and occasionally the face. Rupture follows, and the condition ultimately results in a punched-out, circular or oval ulcer with a tough, dirty grey base and thick, bluish indurated edges; it may take many months or even years to heal, leaving a thin scar. Typical diphtheritic paralysis of the limbs and palate was noted in some of the Sinai cases associated with Klebs-Loeffler bacillus (Craig) in the sores.

Diagnosis.—The condition must be distinguished from ulcus tropicum and Leishmanial sores; cultures may reveal the causative organism, which, in some cases at least, is the diphtheria bacillus.

Treatment.—**PROPHYLACTIC.**—Adequate dietary, protection of exposed parts and antiseptic treatment of abrasions should help. *Curative.*—The only specific treatment is anti-diphtheric serum (4000 units); dramatic cure may follow in certain cases. The lesions themselves must have sterile protective dressings: ammoniated or nitrate of mercury ointment often does good. Iron and arsenic tonics and a high vitamin diet should be given, and autogenous streptococcal vaccines are worth a trial in intractable cases.

PEMPHIGUS CONTAGIOSUS

A contagious skin eruption, known also as *Pyosis mansonii*, due to coccal infection, characterised by inflammatory vesicles and bullæ which ulcerate and scab.

Ætiology.—The disease is common in the humid tropics such as Ceylon and Malaya, also in parts of Africa. European children are specially affected. Culture generally shows *Staphylococcus aureus* or *albus*; streptococci may be isolated.

Symptoms.—The condition begins as a minute red speck which is transformed first into a vesicle, then a bulla and later a pemphigus-like blister. The fluid content, which is at first clear, later becomes purulent and, after bursting, the lesions generally dry up, desquamate and heal, sometimes leaving pinkish, slightly glazed spots on the skin. The eruption is mainly confined to the axilla and crutch, but in children may be spread more widely by auto-infection, the whole body, except the face, being sometimes involved (Smith). Constitutional disturbances are minimal.

Diagnosis.—The condition is allied to impetigo contagiosa and may need to be distinguished from early small-pox, chicken-pox and ringworm.

Treatment.—Cleanliness is all-important and auto-infection must be avoided. The parts should be washed with perchloride of mercury (1 to 1000) followed by a dusting powder of zinc oxide, boracic acid and starch (equal parts). Ammoniated mercury ointment is often useful. Sulphapyridine preparations (M. & B. 693) in full dosage is worthy of trial.

TROPICAL ULCER

Ulcus tropicum or tropical sloughing phagedæna is a gangrenous ulceration of the skin and subcutaneous tissues of unknown ætiology, resulting in the formation of sloughing ulcers of great chronicity.

Ætiology.—In contradistinction to veldt sore, this disease is met with in damp, steamy jungle in the tropics. The lower limbs are generally involved, and a history of preceding trauma is the rule. It is common in debilitated and diseased populations, may affect people of any age and either sex, and has occasionally assumed epidemic proportions, as amongst coolies in the tea plantations of Assam. Some regard it as a dietetic deficiency. Fusiform bacilli and a spirochæte named by Prowazek, *Treponema schaudinni*, are commonly present in the ulcer: various cocci, fungi and diphtheroids have also been found. The condition is directly transmissible by inoculation of ulcer material from man to man (Smith).

Symptoms.—Phagedænic ulcers generally affect the dorsum of the foot and the front of the legs, and more rarely the hands and forearms. The disease originates as a serosanguinous bleb which soon ruptures, leaving a dirty grey slough. This process rapidly extends, forming a foul sloughing ulcer, which may attain several inches in diameter, giving rise to pain, and sometimes fever, and occasionally involving deeper structures like muscles, tendons, blood vessels, nerves, periosteum, and even joints. Three stages are recognisable: (1) spreading sloughing ulceration; (2) a stage of tissue equilibrium when destruction and growth of granulation tissue are equalised; (3) healing. Generally these ulcers persist for months, a factor delaying healing being inadequate epithelial proliferation, even after a healthy granulation tissue base has formed. Many cases show a decrease in blood calcium.

Diagnosis.—In the humid tropics diagnosis is generally easy, though varicose ulcers, yaws, syphilitic and blastomycotic ulcers and oriental sore may need differentiation.

Treatment.—Protection of the legs with puttees is very advisable. Curative treatment varies with the stage of the ulcer. Rest, a nutritious diet, calcium, cod-liver oil and general vitamin reinforcement by multivite pellets (B.D.H.) are advised. In the rapidly ulcerating stage sloughs should be removed and ensol dressings or lotions of carbolic or permanganate applied. Good results have been reported following curettage of the ulcers and daily dressing with B.I.P.P. Cod liver oil dressings have also been favourably reported on, and local treatment with tar spread on lint and changed every three days has been found suitable for mass treatment of natives. The application of iodoform powder may be followed by firmly bandaging with elastoplast which is left undisturbed for a week; septic dermatitis sometimes complicates this treatment. Probably the most effective procedure in the chronic stage is complete excision, followed by skin grafting. Rarely in the acute stage of fulminating cases, with rapid sloughing and gangrene, amputation is necessary to save life; even more rarely has it to be done in chronic cases.

TINEA

Ringworm infections abound in the tropics, some being confined to special regions, while others are much the same as in temperate climates. The chief ones are: (1) *Tinea cruris* or dhobie's itch; (2) Hong-Kong foot or ringworm of the foot; (3) *Tinea unguium*; (4) *Tinea imbricata*. The first two are due to the *Trichophyton*, *Epidermophyton inguinale*: they are not peculiar to warm climates and are described elsewhere (p. 1436).

TINEA UNGUIUM.—A mycotic infection of the nails affecting Europeans from the Far East: it may last for years and be associated with ringworm elsewhere. The nail-bed is involved, leading to brittleness, ridging and opaqueness of the nail. Diagnosis is made by demonstrating *Epidermophyton inguinale* in scrapings mounted in liquor potassæ. In severe cases the nails may have to be removed before cure is effected.

TINEA IMBRICATA (Tokelau).—A form of ring-worm mainly indigenous in the Eastern Archipelago and South Pacific, and characterised by non-inflammatory raised brown spots, giving rise to flaky tissue-paper scales which are free centrally, but attached at their peripheral bases, producing

a rosette-like appearance. These circles are about $\frac{1}{4}$ inch in diameter and as adjacent ones form they cause a characteristic festooned appearance. The fungus, *Endodermophyton concentricum*, is readily demonstrable in the scales: it affects the face, trunk and limbs, but the palms, soles, scalp, axillæ and crutch generally escape.

PITYRIASIS VERSICOLOR or *Tinea flava* is common in the tropics, producing pale, yellowish-brown, scurfy patches on the pigmented negroid skin, especially on the face, neck, arms and chest. Castellani holds that the yellow patches met with in his Ceylon cases differed from the brownish patches long recognised as being caused by *Microsporon furfur* in the European disease, and has named the tropical variety *Tinea flava* and the causal fungus *Malassezia tropica*; the black variety, which is caused by *Cladosporium mansonii*, Castellani calls *Tinea nigra*.

PINTA

This is a group of dermatomycoses associated with coloured patches of pigmentation in the skin.

Ætiology.—The disease, also called *caraate* or *mal de los pintos*, is found in tropical America, is contagious and attacks either sex at any age. A variety of fungi are implicated, including *Penicillium*, *Aspergillus* and *Monilia*.

Symptoms.—Patches of pigmentation are first noted on the back of the hands or face, from which they spread elsewhere: they are somewhat rough, dry and raised, and vary in colour with the fungus, red, violet, white and black types all being encountered. The skin may be offensive and itchiness marked. When the scalp is involved the hair may become white.

Diagnosis.—Microscopic examination of material scraped from the pigmented areas reveals the fungi. The patches are not anæsthetic like leprosy, while leucoderma, which the white variety may resemble, fails to show fungi.

Treatment.—As for ordinary ringworm.

PIEDRA

Trichosporosis or Piedra is a disease common in Colombia and British Guinea in which hard, gritty nodosities form around the hair of the scalp; it is caused by the *Trichosporon giganteum* and may be confused with ordinary Trichomycosis nodosa.

CREEPING ERUPTION

Synonyms.—*Larva migrans*, *Myiasis linearis*, *Hautmaulwurf*.

Definition.—A peculiar linear, slightly raised red eruption, gradually creeping forward in a sinuous or straight line, the posterior end fading away.

Ætiology.—The condition may be produced by *Gastrophilus* or other fly larvæ wandering under the skin, but more commonly it is due to nematode larvæ of animals which have accidentally invaded man. The following species have been implicated: *Ancylostoma braziliense*, *A. caninum*, *Uncinaria stenocephala* and *Gnathostoma hispidum*.

Symptoms.—The symptoms vary in different individuals and include smarting pain and intense itching along the raised line which first shows red spots, and later hard round red papules 2 to 5 mm. in diameter ; pustulation may occur. Unless treated the condition persists for a long time.

Treatment.—Freezing the anterior end of the line where the larva is located, with an ethyl chloride spray for 2 minutes, is suitable for the type due to canine ancylostomes. Multiple lesions may be treated with collodion ethyl acetate or salicylic acid, and blisters and pustules with mercurochrome solution. An injection of pure carbolic an eighth of an inch in front of the spreading spot may kill the larva, or if the condition is due to the larva of *Gastrophilus*, this may be cut down on and removed. Recently oleum chenopodii applied locally either pure or diluted with three parts of castor oil has been favourably reported on.

CERCARIAL DERMATITIS

Definition.—An inflammatory condition of the skin due to the passage through it of different species of cercariæ.

Ætiology.—In 1928 Cort in Michigan described a form of dermatitis due to the passage of *Cercaria elva* through the skin and Taylor and Baylis have also found this in England.

Symptoms.—The skin at the site of entry of the cercariæ becomes intensely itchy and smart, then red spots or urticarial wheals appear, these being followed by papules which sometimes go on to pustulation.

Treatment.—No specific treatment is known. The part should be kept clean and dusted with boracic and zinc powder. Calamine lotion combined with lead acetate may reduce the itching.

ULCERATING GRANULOMA

Synonyms.—Granuloma venereum ; Granuloma inguinale ; Granuloma inguinale tropicum ; Ulcerating Granuloma of the Pudenda ; Serpiginous Ulceration of the Genitals.

Definition.—A very chronic ulcerating condition of uncertain ætiology occurring in the tropics, involving the genitals, perineum and groins.

Ætiology.—The disease occurs in the West Indies, Guiana, Brazil, Porto Rico, parts of India and Africa, the Pacific Islands and Northern Australia. Both sexes are affected, but not before puberty, and all races are susceptible. Spirochætes have been reported, and Donovan and many other observers have found a short, oval bacillus specially located within the mononuclear cells ; it is a non-motile, capsulated bacterium of the rhinoscleroma group, but though found with frequency in the lesions there is still doubt as to its real ætiological significance. The disease itself is probably contracted during coitus.

Pathology.—The condition resembles an infective granuloma, and microscopic section of the nodules situated at the edge of the sore shows infiltration with plasma and round cells containing poorly staining nuclei in which phagocytosed bacilli may occur in clumps. The granulomatous tissue is very vascular, while in the older areas fibrosis and scarring are marked. Spread is by direct continuity and the lymphatic system is never involved.

Symptoms.—The disease begins on the genitals as a flat papule which desquamates, leaving a red granulation-tissue surface which bleeds easily: this superficial ulceration extends serpigginously producing offensive pus. As the process advances the older areas cicatrise, but this scar tissue readily breaks down again. The disease is auto-inoculable so that adjacent parts such as the scrotum and thighs, or the surfaces of the labia become infected. Ultimately the whole of the penis, scrotum and groins in the male, and the clitoris, vulva, labia, vagina, perineal and perianal region in women become involved, and, if unchecked, the urethra and rectum as well. Though skin ulceration extends slowly over a period of many years, the process accelerates once the mucuous membranes are involved, and here there is little tendency to heal. Until the terminal phase the general health remains good and the local lesions give rise to a minimum of pain and discomfort.

Complications.—These include recto-vaginal fistula, urethral stricture, septic cystitis and pyelitis.

Diagnosis.—Ulcerations due to syphilis, tubercle or lupus vulgaris may be confused, and where the glans penis is involved with fungating granuloma, epithelioma may be suspected.

Prognosis.—This has greatly improved by modern treatment; formerly the condition was hopeless, lasting for life.

Treatment.—Illicit intercourse, especially with native women, should be avoided. Surgical excision of the early lesions is curative, especially as the ulceration does not extend deeply, but in more advanced cases is not feasible. The modern treatment consists of intravenous injections of tartar emetic which is a specific. This drug is given as in schistosomiasis (p. 311) only a longer course of injections and a greater total dosage, *i.e.* 50 to 60 grains, is generally necessary; in extreme instances as much as 150 grains have been given. Certain pentavalent preparations of antimony, such as stibosan and stibenyl, are reported to be more efficacious, a total dosage of 3–4 g. being necessary to obtain complete cure. Protein shock produced by the intravenous injection of T.A.B. vaccine starting with 50,000,000 per c.c. and gradually increasing to 300,000,000 per c.c. may be employed in addition to antimony: sometimes it stimulates healing in a remarkable fashion.

In certain chronic cases unaffected by antimony, gauze soaked in an aqueous solution of zinc oxide (40 per cent.) is an effective dressing, especially if non-hæmolytic anerobic streptococci be present in the ulcer. Sulphapyridine therapy (M. & B. 693) is still under trial.

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SECTION XX

DISEASES OF THE NERVOUS SYSTEM

AFFECTIONS OF THE CRANIAL NERVES

INTRODUCTION

AFFECTIONS of the cranial nerves may be due to their involvement: (1) by purely local disease at some point between their central origin and their peripheral distribution; (2) by some general toxic or infective disease of the whole nervous system; as, for example, by alcoholic or diphtheritic poisoning; (3) by multiple local lesions of the central nervous system, as, for example, by syphilis or disseminated sclerosis; and (4) in some systemic neurone degeneration, as in progressive muscular atrophy.

In the case of local lesions within the brain stem, the presence of one or more cranial nerve palsies may have considerable localising importance in diagnosis. In their peripheral course, both inside and outside the skull, these nerves may be involved by a great variety of inflammatory processes, by hæmorrhage, or by compressing tumours.

THE OLFACTORY NERVE AND TRACT

The sense of smell includes the appreciation of all flavours, and in its absence taste is reduced to the appreciation of bitter, sweet, salt and sour. The view that only readily oxidisable substances are appreciated by the sense of smell, formerly expressed in these pages, cannot be sustained. Benzene, for example, has a powerful smell yet is chemically very stable. The same is true of chloroform and carbon tetrachloride, both substances with a strong odour. The gas phosgene is a final example. Most conditions of anosmia are due to affections of the nasal mucosa, or occur in such general diseases as syphilis or influenza. More important is unilateral anosmia, which indicates the involvement of one olfactory tract by the pressure of a tumour. Bilateral anosmia is a not uncommon sequel of head injuries in which the olfactory filaments are torn as they traverse the cribriform plate.

OPTIC NERVE

The optic nerve, from its origin at the back of the eyeball to its termination in the optic chiasma, is liable to injury from the pressure of tumours within the skull, or at the back of the orbit, and from inflammatory conditions of

the bone and periosteum as it passes through the optic foramen. It may be affected by a primary thrombosis of vessels, or from thrombosis of the ophthalmic artery in the condition known as carotid hemiplegia. These conditions result in blindness of one eye, which may be partial or complete, and with a varying visual field, according to the degree of the lesion and the manner in which the pressure falls upon the optic nerve. On account of the cutting off of the light-reflex path, the pupil will be moderately dilated and insensitive to light. Notwithstanding the fact that the nerve cells which control the nutrition of the optic nerve fibres are situated in the retina, lesions of the optic nerve of any long duration produce atrophy of the optic disk.

RETROBULBAR NEURITIS

Inflammatory and other local lesions in the substance of the optic nerve between the globe and the chiasma are of very common occurrence. According to their severity they give rise to partial or complete blindness, which often recovers wholly or in part. The central part of the optic nerve is the seat of election for these lesions and, therefore, the visual defect appears commonly in the form of a central scotoma. According to the degree of the visual defect, the pupil tends to be dilated, and to react poorly to light, and dilate badly to shade. These conditions of retrobulbar neuritis are very often followed by optic atrophy of varying degree. When the inflammatory lesion occurs far forwards and impinges upon the optic papilla, papilloedema occurs, and, as this region is sensitive, whereas the optic nerve is not, there is usually pain on movement of the eyeball. The prognosis in this condition varies with the causes, which are as follows :

1. *Syphilis*.—The lesion is a diffuse gumma of the nerve, and sometimes there is thrombosis. It is commonly unilateral, and often recovers, if treated early and energetically, but in severe untreated cases, and when thrombosis occurs, it not uncommonly ends in complete blindness. It is the common cause of complete blindness confined to one eye in tabes, and in other syphilitic conditions.

2. *Disseminated sclerosis*.—Plaques in the optic nerve tracts and chiasma are very common in this disease. When they first form they are swollen oedematous pink patches and cause pressure, and on their subsequent shrinking, this pressure is removed, and the nerve fibres recover to a great extent, and never become completely destroyed. This condition is the cause of the very common transient amblyopia or blindness, which may come on very suddenly. Though the plaques often occur in the tracts and chiasma, yet the visual defect always indicates a lesion of the optic nerve, and therefore it is possible that it is determined by the swelling of the optic nerve as it passes through the optic foramen, and is compressed against the bony canal. It may be unilateral or bilateral, and may recur several times. It always causes some degree of optic atrophy ; but is never followed by complete blindness.

3. *Local septic conditions*.—Before the general recognition of disseminated sclerosis as the common cause of retrobulbar neuritis in young adults, the chief rôle in its production was attributed to local infection, or suppuration in the sphenoidal or other accessory sinuses in the neighbourhood. The natural restoration of vision that marks the subsidence of the retrobulbar neuritis was accordingly often ascribed to the operative procedures designed

to deal with the local infection. It is probable that such local infections are but a rare and exceptional cause of retrobulbar neuritis.

4. *Diabetes*.—In this malady a form of retrobulbar neuritis occurs. It commences with a central scotoma for colour, and the failure of vision may progress to blindness, with optic atrophy.

5. *Other causes*.—Tobacco, if over indulged in, may cause a curable form of retrobulbar neuritis, commonly called "tobacco amblyopia," of which the chief sign is central scotoma, and the symptom mistiness of central vision. It recovers rapidly on the removal of the cause. Alcoholic indulgence, and especially taking of wood spirit and many other poisons such as arsenic, lead, bismuth and quinine, may cause retrobulbar neuritis. A large proportion of cases of retrobulbar neuritis are without discoverable cause; there is evidence that some of these are dependent upon local oedematous reactions within the optic nerve of an allergic nature.

The expansion of the optic nerve within the globe of the eye is visible on ophthalmoscopic examination, and the pathological changes therein occurring afford important indications which cover the whole realm of medicine. Among these changes the most important in connection with diseases of the nervous system are: syphilitic choroiditis, as an indication of the presence of syphilis, papilloedema, which is fully described in the section on intracranial tumours, and optic atrophy.

OPTIC ATROPHY

Optic atrophy is recognised, on ophthalmoscopic examination, by a peculiar opaque whiteness and flatness of the disk, with a very high contrast at the edge of the disk between disk and surrounding retina, both as regards colour and limitation. The lamina cribrosa—the sieve-like cross-latticing of the strands of the sclerotic through which the bundles of optic nerve fibres pass—becomes visible as a stippling of the temporal region of the disk. The vessels of the retina become atrophied, and are seen to be unduly small. In many atrophies the edge of the disk is sharply cut; but when atrophy follows papilloedema the edge is apt to be fluffy, like that of torn cotton-wool. Optic atrophy may be of three kinds—(1) Primary optic atrophy results from an original devitalisation and death of the nerve cells of the retina with their processes, which constitute the fibres of the optic nerve. This is a primary neuronc degeneration, analogous to that of the anterior-horn cells in progressive muscular atrophy. (2) Secondary or retrograde optic atrophy results from lesions of the optic chiasma and optic nerve, and is the constant result of long-continued pressure upon these structures. (3) Consecutive optic atrophy follows the more severe grades of papilloedema and papillitis, in proportion as these are of long standing, and proportional to the amount of exudate, and is due to strangling of the optic nerve fibres by the oedema in the first place, and by the cicatrization subsequently. Severe degrees of papilloedema may, if pressure be relieved, recover perfectly without atrophy or impairment of sight. It may be that there is another factor in the atrophy following papilloedema, and that is the long-continued pressure of a distended infundibulum upon the optic chiasma.

Ætiology.—1. It is of frequent occurrence in familial, hereditary or

congenitally installed diseases in which primary degeneration of neurones occurs, as in cerebral diplegia, amaurotic family idiocy, and hereditary cerebellar atrophy where it is characteristic of Marie's type, and sometimes occurs in Friedreich's type. It is the chief feature of the familial optic atrophies, of which Leber's type, appearing about the twentieth year, is one. It occurred in several members of a family with peroneal atrophy under our observation.

2. It is one of the common manifestations of syphilis of the nervous system, and may occur alone, but much more usually as part of the syndrome of tabes and general paralysis. It is not rarely met with in congenital syphilis. It is commonly associated with local lesions of the chiasmal region, pituitary neoplasms being the most often met with. The optic atrophy of disseminated sclerosis is not a primary optic atrophy, but is secondary to retrobulbar neuritis.

3. It may follow the exhibition of certain drugs, and noticeably the injection of the earlier used arsenic preparations, such as atoxyl, soamin and orsudan. In rare cases it has followed the use of quinine.

4. Optic atrophy also occurs in connection with diabetes, malaria and arterial disease. It is common as a primary condition in later life. Its occurrence in glaucoma from increase of the ocular pressure requires no explanation.

Prognosis.—This is uniformly bad in primary atrophy. When once the degenerative process is installed, the atrophy proceeds to complete blindness, sometimes slowly, sometimes quickly, and seems entirely uninfluenced by any form of treatment.

Secondary optic atrophy is frequently arrested, with the recovery or removal of the cause; but some defect of vision usually remains. Consecutive atrophy from neuritis may be of any degree of severity, from the slightest, which allows of $\frac{5}{8}$ ths vision, to the most complete with utter blindness and loss of light reflex.

OCULO-MOTOR NERVES

The third nerve supplies the two internal muscles of the eye, and all the external muscles of the eyeball, except the superior oblique, which is supplied by the fourth nerve, and the external rectus, which is supplied by the sixth nerve. Complete paralysis of this nerve produces a dilated and inactive pupil with complete ptosis, a downward and outward strabismus and complete loss of upward, downward and inward movements. There is often no diplopia complained of by the patient, because of dropping of the lid. When diplopia is present it is a crossed diplopia, because the strabismus is divergent. There is secondary deviation of the sound eye, and false projection in the visual field.

The fourth nerve supplies the superior oblique muscle. Paralysis produces no obvious strabismus, but in looking outwards or downwards there is a wheel movement of the globe which can be detected by observing the conjunctival vessels when the eye moves. The diplopia is most discomforting, and occurs in every position of the eyes, except on looking up. The diplopia is uncrossed, and the false image is lower than, and with its top tilted toward, the true image.

The sixth nerve supplies the external rectus muscle. Paralysis produces a convergent squint and an uncrossed diplopia. In the case of the skeletal musculature, we are accustomed to distinguish between upper and lower motor neurone paralyses; the essential difference being that in the former case we are dealing with loss of co-ordinated movements, in the latter with paralysis of individual muscles. Disorders of ocular movements may usefully be considered on the same basis of classification. Thus we may contrast the loss of conjugate deviation of the eyes to the right—an upper motor lesion—with paralysis of the left internal rectus the result of a lesion of the lower motor neurone. In the former case, the internal rectus muscle acts normally on convergence, but does not act on attempted conjugate deviation to the right. In the latter case—a lesion of the peripheral nerve supply—the muscle is paralysed in all movements of the eye.

We may thus classify paralyses of ocular movement under the following headings: (1) paralyses of ocular movements, conjugate and supranuclear; and (2) paralyses of ocular muscles, nuclear, stem and root palsies.

1. PARALYSES OF OCULAR MOVEMENTS.—Conjugate palsies consist in the loss or impairment of co-ordinated ocular movements in one or more directions. Since both eyes are equally and simultaneously involved, there is neither squint nor diplopia. The commonest form is that in which there is loss of conjugate movement to one or other side. The lesion in such a case is so situated as to interrupt the association path between the sixth and third nerve nuclei, probably where this path lies in the dorsal longitudinal bundle. If a lesion in this situation be a coarse one (*i.e.* not a simple nerve cell degeneration), there will probably be an associated nuclear or stem palsy, in which case there may be diplopia and squint. Possibly a lesion lying just oral and ventral to the sixth nerve nucleus is capable of causing a pure paralysis of conjugate deviation to the side.

Lesions involving the corpora quadrigemina cause loss of vertical (upward) movement of the eyes, sometimes with dilatation and immobility of the pupils. This combination of signs may be seen in pineal tumour.

Supranuclear ocular palsies differ from conjugate palsies in that with the latter a given movement is totally lost, while in the former, whether or not the movement occurs will depend upon the particular stimulus in action. Thus, in a supranuclear palsy the patient may be unable to deviate his eyes to order, but deviation may be elicitable by labyrinthine stimulation. Or again, the gaze may be unable to follow a moving object, but if the object be still and the head slowly rotated passively, the gaze may remain fixed on the object, and the eyes thus come to take up a position of deviation.

2. PARALYSES OF OCULAR MUSCLES.—The lesion in this case may be in the brain stem (nuclear or stem palsies), or in the peripheral course of the nerve (root palsies). Further, the lesion of the peripheral part of the nerve may involve the nerve between its point of origin from the brain and the point of entry into the orbit; that is, in its intracranial course; or in the region of the sphenoidal fissure; or, finally, within the orbit.

Within the orbit lesions of any of these nerves or of their branches may occur from perforating wounds, blows upon the eyeball or from local syphilitic lesions of a gummatous nature. Injury to the lenticular ganglion, with resulting complete internal ophthalmoplegia, not uncommonly results from a blow on the eyeball. A lesion, confined to the nerve to the inferior oblique,

occurs in rare cases from syphilis, and produces a peculiar attitude of the head, for, since the resulting diplopia occurs only above the horizontal level of the eyes, the patient constantly throws back his head, and looks down his nose to avoid the diplopia.

Lesions at the back of the orbit may involve one or more of the oculomotor nerves, and since the first division of the fifth nerve passes through the sphenoidal fissure with these nerves, and the second division of the fifth nerve is entering the infra-orbital canal at the apex of the orbit, both these nerves are commonly involved in the same lesion. New-growths, including those of the bone, periosteal inflammation and subperiosteal hæmorrhages arising from fracture of the skull are the common lesions. A not infrequent clinical picture may be described. It has been attributed to a periostitis of the orbital bones in the region of the sphenoidal fissure. There is, however, no pathological confirmation of this view, which remains speculative. It has been thought, too, that the condition is closely allied to the common facial palsy from exposure to cold, but here also proof is lacking. It may occur at any age from puberty onwards. The serum reactions for syphilis have always shown that this infection is absent. The condition may arise from exposure to cold or from septic conditions of the nose and its accessory sinuses, and sometimes without obvious cause. The malady commences with pain in the orbit, which is often severe and long lasting. Soon after some proptosis is evident, and there is tenderness on pressing the globe backwards. This is soon followed by signs of involvement of the nerves which pass through the sphenoidal fissure. The sixth nerve is the first and sometimes the only nerve involved, but usually the paralysis of this nerve is followed by that of the fourth, the first division of the fifth, the third, and the second division of the fifth nerve in that order. The final result usually is that of a total ophthalmoplegia with anæsthesia of the upper two divisions of the fifth nerve, unilateral proptosis and tenderness of the eyeball, and often excruciating and lasting pain. When the sixth nerve is involved alone, and there is no spread to the divisions of the fifth nerve, there may be little pain, and the proptosis and tenderness may be little marked. Under treatment with mercury by inunction, salicylates in full doses, warm applications to the eye and counter irritation, the condition commonly recovers in a few weeks in the milder cases, to a few months in the more stubborn ones. It is in their intracranial course that the oculomotor nerves are most frequently involved by disease-processes, or by injury; thus, by acute or chronic meningitis, by toxic or infective neuritis, by compression from tumour, aneurysm, hæmorrhage or fracture of the skull.

Fractures of the skull, may involve the orbit, or the middle fossa of the skull, thus producing ocular palsies. Sometimes a blow on the skull without fracture is followed by an ocular palsy. It is possible that in this instance small hæmorrhages in the brain-stem involving the nuclei may be responsible.

In subarachnoid hæmorrhage, the effused blood in the region of the interpeduncular space may compress the cranial nerves, thus producing ocular palsies. These may also occur in the course of an acute lepto-meningitis. In association with middle ear disease, there is occasionally a localised meningitis at the tip of the petrous bone, which gives rise to a unilateral sixth nerve palsy and to pain on the side of the head and face from fifth nerve involvement. This is the condition known as *Gradenigo's syndrome*. It is

usually seen in children ; and a mastoid operation may be necessary before it clears up.

Formerly, one of the commonest causes of ocular palsies was a gummatous infiltration of the nerves of the base of the brain in neurosyphilis. In some cases of *tabes dorsalis*, however, it is probable that the lesion underlying the defects of ocular movement (squint, diplopia, ptosis) may be due to primary degeneration of nerve cells in the nuclei.

In some cases of alcoholic and diphtheritic multiple neuritis, paralysis of one or more ocular muscles may occur, and also an isolated sixth nerve palsy has been known to follow the spinal administration of stovaine.

In some elderly subjects with atheroma and high blood pressure a sixth or third nerve palsy may develop, and recover after some two or three months. It is probable that in such not infrequent cases one or other of these nerves has been compressed by a tortuous and rigid artery. Thus, during its passage forwards the sixth nerve lies in close contact with the middle cerebellar, the basilar and the internal carotid arteries ; the fourth nerve crosses the posterior cerebral artery ; and the third nerve lies between the last-named vessel and the superior cerebellar artery.

Stem palsies, in which the lesion is in the brain-stem, are usually recognisable from the presence of associated signs due to involvement of other structures. These may be the long projection paths, sensory or motor, association nuclei and reflex centres, or other cranial nerve nuclei. Thus, a lesion in the crus will produce a homolateral third nerve palsy, with a crossed hemiplegia (Weber's syndrome). A lesion in the region of the red nucleus will cause a homolateral third nerve palsy, with tremor of the crossed limbs (Benedikt's syndrome). A pontine lesion will produce associated sixth and seventh nerve lesions on the side of the lesion.

Nuclear palsies may result from a number of inflammatory processes within the brain stem : *e.g.* epidemic encephalitis, disseminated sclerosis, acute poliomyelitis, botulism, chronic alcoholism (Wernicke's encephalitis *hæmorrhagica superior acuta*). Barbiturate poisoning, diphtheria and diabetes may also be associated with ocular palsies of nuclear origin. Finally, there is the rare condition known as *chronic progressive nuclear ophthalmoplegia*, in which there is a primary degeneration of the third, fourth and sixth nuclei. The course of this malady, which is related to progressive muscular atrophy, is slowly progressive, and finally all the extrinsic ocular muscles are paralysed.

PATHOLOGICAL CONDITIONS OF THE PUPIL AND OF ACCOMMODATION.—Myosis or unusual smallness of the pupil is a common sign of syphilis of the nervous system. It is an important sign of paralysis of the cervical sympathetic. It occurs in lesions of the pons below the third nerve nucleus, is often met with in advanced age without pathological associations, and is also a symptom of the morphine habit.

Eccentricity of the pupil and deviations of its form from the circular are important signs of nervous syphilis, and these signs occur also in lesions of the foremost part of the third nucleus.

Inequality of the pupils occurs in connection with all nuclear and peripheral ocular paralyses, and with cervical sympathetic paralysis. It accompanies all defects of vision from lesions of the visual path between the eye and the external geniculate bodies, provided the appreciation of light be unequal in the two eyes. It may be congenital or associated with inequalities

of the refraction of the two eyes, and then has no pathological significance. It is commonly a sign of nervous syphilis.

The Argyll Robertson pupil (reflex iridoplegia) as originally described includes loss of the light reflex, myosis, inequality and irregularity of the pupils. Atrophic changes in the stroma of the iris have also been described. These are best seen in blue-eyed persons in whom the stroma is not concealed by pigment, and they impart a fineness of texture and a pale, washed out tint that, in association with myosis, gives the eye of the tabetic so characteristic an appearance.

The iridoplegia may be due to a lesion in the region of the posterior commissure and the aqueduct of Sylvius, and it is here that some believe the essential lesion of the Argyll Robertson pupil to lie. But the Argyll Robertson pupil of neurosyphilis is almost constantly associated with the other pupillary phenomena noted above, and it is difficult to see how a central lesion could produce any of these. In short, the pathogenesis of this familiar phenomenon still remains obscure.

When reflex iridoplegia is met with apart from neurosyphilis—which is but rarely—it is not accompanied by myosis, or irregularity of the pupils, and the absence of these signs is of diagnostic importance.

It has been said to occur in disseminated sclerosis and in epidemic encephalitis, but the statement is one to be accepted with reserve.

THE MYOTONIC PUPIL.—During the past fifteen years a number of writers (Foster Moore, Holmes, Adie) in this and other countries have described a pupillary phenomenon which has the following components: There is no reflex contraction to light, and there is a very slow contraction on convergence, followed by an extremely slow relaxation and dilatation. In some patients this condition is associated with a total loss of all tendon jerks. The patient is more commonly a young woman in normal health, the phenomenon being discovered usually in the course of a routine examination of the eyes. It constitutes no disability. Serological examination of the blood and cerebro-spinal fluid is always negative, and the condition is not related to neurosyphilis or tabes, with which, however, it may be confused if a complete examination of the nervous system be not made.

Total internal ophthalmoplegia is met with in lesions of the anterior part of the third nucleus, and in lesions of the lenticular ganglion in the orbit.

Wernicke's hemianopic pupil phenomenon is a test for the position of a lesion causing hemianopia. If the lesion is situated upon the visual path where that path contains the light reflex path, the pupil does not react when light is thrown on the blind side of the retina. In other words, this sign is present if the lesion is involving the visual path between the eye and the external geniculate body. When the lesion is between the geniculate body and the visual cortex in the occipital lobe, the pupil reacts equally well from the blind and from the seeing field.

PARALYSIS OF THE CERVICAL SYMPATHETIC

Synonym.—Horner's syndrome.

So far as the eye and orbit are concerned, the sympathetic is the tonic retractor of the lid, the tonic protruder of the eyeball, and the tonic dilator of the pupil, and stimulation of this mechanism results in retraction of the

lid or widening of the palpebral fissure, exophthalmos and wide pupil, while paralysis of the cervical sympathetic produces narrowing of the palpebral fissure (cervical sympathetic ptosis), and a small pupil. It is customary to include enophthalmos amongst the components of cervical sympathetic palsy, but it is extremely doubtful that this is ever present. The excitation condition is seen in Graves's disease; the paralytic condition is of common occurrence in nervous diseases. The cervical sympathetic is also the tonic vaso-constrictor and secreto-motor nerve of the head generally, but disturbance of the mechanism does not often give rise to characteristic or important clinical phenomena. A curious lack of expression is, however, sometimes observable in the face on the side of the lesion. Cervical sympathetic paralysis occurs in the following clinical associations: (1) In all lesions of the cervical cord, especially when the last cervical and first dorsal segments or roots are damaged. It is common in syringomyelia. (2) In lesions of the cervical sympathetic trunk by trauma, pressure, growths, etc. (3) It is very common in tabes and nervous syphilis generally, where it appears as partial bilateral ptosis with small pupils. It appears to be a primary neuronie degeneration in this condition and never improves.

THE FIFTH OR TRIGEMINAL NERVE

Symptoms of Lesions of the Fifth Nerve.—Pain over the sensory distribution of this nerve occurs from irritating lesions and reflexly, if its periphery is irritated. With organic lesions in any part of its course, the pain is followed by sensory loss, corresponding with the part involved. The initial expression of this sensory loss in a progressive lesion of the fifth nerve: *e.g.* compression by an eighth nerve tumour, is loss of the corneal reflex. The peculiar disease, neuralgia, of which the pathological basis has not been as yet discovered, is practically confined to the distribution of this nerve. Herpes zoster over the distribution of this nerve is common, and results from a lesion by a virus infection in the Gasserian ganglion, and is in every respect comparable with that occurring in the distribution of the spinal nerves from similar lesions in the posterior root ganglia. It produces bad scarring, and when affecting the cornea is apt to produce ulceration, very destructive to the eye. It is accompanied by severe pain, which may be persistent for months. It should be borne in mind that the sensory supply to the cornea is entirely from the naso-ciliary branch, via the long ciliary nerves, and that herpes zoster of the cornea is usually accompanied by a small group of vesicles only at the tip of the nose on the same side.

Taste.—It has frequently been argued that loss of taste over the anterior two-thirds of the tongue follows destruction of the Gasserian ganglion and proximal portions of the fifth nerve. Cushing has, however, investigated this subject upon a series of cases of complete Gasserectomy, and has found that in every case the sense of taste was preserved. The path of taste thus seems proved. It is from the glossopharyngeal nucleus via the fasciculus solitarius, portio intermedia, facial nerve, chorda tympani and lingual nerve to the tongue.

Trophic changes.—Lesions of the first division of the fifth nerve are often productive of serious corneal deterioration and ulceration, which may be followed by septic panophthalmitis. These changes, however, have been

proved to be the result of mechanical damage upon the insentient surface. If, for example, after destruction of the fifth nerve for neuralgia, the eye be carefully protected either by covering or sewing it up, these changes do not occur. After a little while, the anæsthetic cornea becomes much less vulnerable, and will stand the wear and tear of ordinary life without disturbance.

Paralysis of the motor function of the fifth nerve occurs in lesions of the nucleus in the pons, or of any part of the peripheral course of the motor division. The signs of such paralysis are not apparent to the patient, who experiences no difficulty in mastication, provided the lesion be unilateral. To the observer, the jaw deviates to the side of the paralysis on opening the mouth, on account of the action of the unopposed external pterygoid of the sound side. The masseter, as felt by the finger on its anterior edge, does not harden on biting, nor do the temporal muscles harden. The floor of the mouth does not stiffen on the paralysed side on forcibly opening the mouth.

Bilateral involvement of all the muscles supplied by the fifth nerve is the rule in all cases of progressive muscular atrophy where the bulbar nuclei are affected.

THE SEVENTH OR FACIAL NERVE

This nerve supplies all the facial muscles of expression. In the petrous bone, it gives off a branch to supply the stapedius muscle. One quarter of an inch above the stylomastoid foramen, it gives off the chorda tympani, which enters a small foramen, the iter chordæ posterius, which leads it to the tympanum, where it crosses the long process of the malleus and enters the temporal fossa by a canal, the iter chordæ anterior, and subsequently joins the lingual, by which it is conveyed to the anterior two-thirds of the tongue and to the submaxillary and sublingual glands.

1. BELL'S PARALYSIS

Synonym.—Common facial palsy.

Definition.—A common variety of peripheral paralysis of the facial nerve, sometimes of uncertain ætiology and sometimes a sequel of herpes of the geniculate ganglion. The paralysis almost invariably recovers, but if the recovery is slow, a very peculiar spasm or facial contracture may accompany or follow the recovery.

Ætiology.—Facial paralysis is rare at the extremes of age and it is most common in early adult life. The sexes are equally affected. Many different views have been held as to the pathogenesis of Bell's palsy, as, for example, that it is the result of a local inflammation of the fibrous tissue forming the deep part of the sheath of the parotid gland, from which a process in the form of a sheath accompanies the facial nerve into the Fallopiian aqueduct, and along which the inflammation extends and compresses the facial nerve in that canal. The proofs adduced in favour of this view are, that facial paralysis is so often accompanied at its onset by pain in the stylomastoid region and behind the mastoid process and by tenderness on pressure, and that in some cases there is very considerable swelling of the deep part of the parotid gland. Moreover, it might be thought that the pathological process begins outside, and subsequently spreads up the facial canal, since the loss of taste in the anterior two-thirds of the tongue, from involvement

of the chorda tympani, is so often not present when the palsy first appears, and develops in the course of a few days, as the inflammatory process spreads up the facial canal, and reaches the region where the chorda tympani leaves the facial trunk. However, it is apparent that this can be no more than speculation, lacking pathological confirmation. On the other hand, it is clear that a proportion of cases of Bell's palsy are a sequel of herpes of the geniculate ganglion. There is also, in the experience of the present writer, a curious periodicity in the appearance of cases of Bell's palsy in the late autumn and early spring. While this may perhaps be attributed to cold weather, it is perhaps more strongly indicative of an infective origin and suggests that geniculate herpes may be responsible for a greater proportion of cases of Bell's palsy than is commonly believed.

Symptoms.—The onset is usually rapid and sometimes even sudden. Pain of a neuralgic character below the ear, behind the mastoid process, or referred to the occipital region, is common, but it does not last more than a few days, and sometimes pain is entirely absent. On deep pressure upon the styloid region behind the ramus of the jaw on both sides, one can almost always elicit the fact that there is tenderness on the paralysed side, and sometimes obvious swelling of this region may be felt. The first sign of the facial paralysis is that the patient feels the face to be stiff when he attempts to move it. Subsequently, the paralysis appears rapidly, and the face is drawn over to the opposite side. The paralysed side is motionless, according to the degree and distribution of the paralysis, if incomplete, and, if complete, is expressionless. The eye cannot be closed, and there is epiphora from paralysis of the tensor tarsi. The paralysis at the corner of the mouth causes difficulty in articulation and escape of fluids on drinking, but the patient soon learns to dodge these disabilities. When the paralysis is partial it is nearly always the lower part of the face which is the most affected. The facial muscles soon become hyperexcitable to mechanical stimuli. In nearly all the severe cases, there is loss of taste over the anterior part of the tongue. It should be remembered that the sense of taste is confined to a very small area on the lateral edge of the tongue, some half an inch behind the tip.

There is never any pain in the distribution of the facial nerve. After a time, which may vary from a few days to two years, the paralysis begins to recover, and invariably this recovery appears in the upper facial region first, and in almost every case becomes complete. We have seen perfect recovery follow complete paralysis lasting 21 months. Bilateral Bell's palsy is not so rare as is supposed. When seen, there is usually a lapse of 4 or 5 days before the second nerve shows signs of paralysis. The paralysis may become complete on both sides, or upon one only. Perhaps less common is recurrent Bell's palsy. The writer has seen several cases of the kind, including one in which the patient has three separate Bell's palsies, two on the right and one on the left side of the face. All recovered.

Treatment.—Those who believe in a fibrositic aetiology of Bell's palsy will base their treatment on this hypothesis. Salicylates and iodides are given internally and mercurial inunction (3 grs. rubbed in over the mastoid region daily for 2 weeks). The local application of warmth and of such a counter-irritant as tincture of iodine may also be used. The patient is probably best kept in the house for the first week and instructed to massage the face gently using olive oil for 5 to 10 minutes daily. In cases which show

early signs of recovery this is adequate, but when after a month or 6 weeks no evidence of recovery is seen, undue stretching of the paralysed muscles may be minimised by "splinting" the face. For this purpose a silver wire, rubber covered where it turns round the lip, may be bent so as to hook round the lip at one end and over the ear at the other, so that the mouth is kept symmetrical during facial movements. Gentle massage may also be continued. In such cases it has been customary to give galvanic stimulation to the paralysed muscles. Since the present writer abandoned this practice many years ago, he has satisfied himself that recovery proceeds as quickly and completely without it, and the distressing facial contractures that are sometimes seen do not occur. In other words, electrical stimulation has no place in the treatment of Bell's palsy. When geniculate herpes is in question, the vesicles and the swollen pinna require the local treatment suitable for this condition.

Facial paralysis from caries of the temporal bone rarely makes any recovery, and it is almost always complete and permanent. To remedy the unsightly and permanent distortion of the face, union of the peripheral trunk of the facial to the central end of the divided spinal accessory or preferably the hypoglossal nerve, has been performed, and with considerable success. Section of the hypoglossal with consequent hemiatrophy and hemiparalysis of the tongue produces no disability with speech, mastication or swallowing. It is not so much that reunion of this nerve restores volitional power to the face, but associated movement does return and also some after-contraction, which restores to some degree the symmetry of the face.

Facial Paralysis from herpes of the geniculate ganglion.—Among the not infrequent causes of facial palsy must be numbered geniculate herpes. Attention was first drawn to this cause by Ramsay Hunt. The herpetic vesicles, preceded by local pain, appear in the external auditory meatus and adjacent parts of the pinna, and sometimes also just behind the pinna and on the soft palate and anterior pillar of the fauces. When the innervation of the last named derives fibres from the geniculate ganglion, the clinical picture of geniculate herpes is apt to be a misleading one if it be not thought of. The patient complains of pain in the ear, and in the throat on the same side. As the eruption develops the fauces on the affected side are red and injected, and several small ulcers (ruptured vesicles) may be seen. At the same time, the vesicles appear in the ear, rupture, and give off a watery discharge which may be mistaken for otorrhœa. The pinna may then swell very considerably. After some days, during which the patient may feel ill and be feverish (temperature of 100° to 102° F.), a facial paralysis almost invariably develops and becomes complete within 12 hours. In milder cases there may be only initial pain in the pinna and the appearance of herpetic vesicles on the pinna without much swelling. It is in the severe cases that an erroneous diagnosis of middle ear disease with otorrhœa may be made and hazardous and unnecessary steps be taken to deal with this. According to Ramsay Hunt facial palsy always follows geniculate herpes, and undoubtedly many cases of this kind, where the herpetic eruption is minimal, escape accurate diagnosis.

Diagnosis.—Care in diagnosis is necessary lest peripheral facial palsy of very unfavourable prognosis should be mistaken for it. The facial palsies which result from lesions of the nerve in the temporal bone, from caries and

from tumour, those due to lesion of the nerve within the skull and from pontine lesions, rarely make any recovery. To this rule the following exceptions must be made: In the peripheral facial paralysis of poliomyelitis, lethargic encephalitis, tetanus and diphtheria recovery always occurs, if the patient survives.

Facial contracture.—In cases of long duration when recovery commences, the face goes into a condition of persistent spasm which causes often a very unsightly distortion of the face, which is very disappointing to the patient, who after waiting many months for improvement, now finds the place distorted in the opposite direction and to a more severe degree than at the onset. No adequate explanation of facial after-contracture has ever been put forward, and no similar condition occurs after the lesion of any other peripheral motor nerve, so far as we are aware. It recovers slowly in the majority of cases. Patients should be warned from the first about the occurrence of after-contracture so that disappointment may be obviated, and at the same time encouraged as to the probability of complete recovery.

The diagnosis is not difficult, and mistaken diagnosis means faulty examination. In disease of the temporal bone, the facial palsy is accompanied by signs of such disease, which should be carefully sought, namely, deafness, perforation of the drum, discharge from the ear, and signs of long-standing otitis.

Lesions within the skull are apt to co-involve the auditory nerve, the fifth nerve of the cerebellum, and the characteristic signs of tumour of the lateral recess are common. In the pons, hemiplegia, hemiataxy and hemi-anæsthesia are likely to coexist.

Course and Prognosis.—Recovery is so usual that it should be promised in every case. The date of recovery is often difficult to forecast. If at the end of a week after the onset there is the slightest trace of any voluntary power in the orbicularis palpebrarum, which is the “ultimum moriens” of the facial muscles, or if any trace of faradic excitability to bearable stimuli remains, then it may be confidently said that recovery will be complete and rapid within 3 months, and that there will be no contracture. Cases in which no complete paralysis occurs in any region of the face usually recover in a fortnight. In complete cases, with complete reaction of degeneration in the muscles, it is difficult to say when recovery will occur or when the effect of contracture will be at an end. Cases which show no loss of taste and, therefore, in which there is no great extension of the inflammatory process up the facial canal, usually recover rapidly. Traumatic facial paralysis from blows upon the side of the face, and obstetrical facial paralysis from the pressure of forceps during delivery, always recover and leave no sequelæ.

2. PERIPHERAL FACIAL SPASM

Synonym.—Facial hemispasm.

Definition.—A unilateral malady of the facial nerve, in which inter-mitting spasm of the facial muscles occurs, exactly like that caused by faradism of the facial trunk. Rarely it is associated with a slowly oncoming facial paralysis, and may follow a facial paralysis due to injury.

Ætiology.—This malady occurs in adults, and the onset is usually in-

sidious and without known cause. It is most often seen in middle-aged women. It is certainly due to a lesion of the peripheral facial nerve trunk, and this lesion seems to be of such a nature as to irritate, and not in most cases to destroy; but in rare cases partial destruction, with the appearance of partial facial paralysis, does occur.

Symptoms.—It commences with twitching of some part of the facial musculature, which occurs at first at rare intervals, and subsequently becomes more and more frequent, so as in some cases to be almost continuous. Commencing locally, it tends to spread so as to involve the whole face in a sudden and hideous contortion. We have seen cases in which the attacks of peripheral facial spasm at first glance almost exactly resembled a Jacksonian fit of the face. The spasms may be so severe and continuous as to keep the eye closed for long periods together, and to interfere greatly with the work and enjoyment of life. The malady is associated with no other symptoms. Cases exist in all degrees of severity, from the mildest, in which an occasional flicker of the face occurs, to the most severe and incapacitating and unsightly malady.

Course and Prognosis.—Some of the cases recover spontaneously, and others under treatment; but when the malady becomes severe and the spasm hardly remitting, it is practically intractable, except by operative interference.

Treatment.—In the milder cases, measures calculated to subdue chronic inflammation, such as mercury, iodides and salicylates, are said to be of benefit. In severe cases, the only remedy which affords relief is the injection of alcohol into the facial nerve either at the stylomastoid foramen, or as it crosses the ramus of the jaw half an inch below the external auditory meatus, or when one division of the nerve only is affected, in any part of the *pes anserinus*.

THE AUDITORY AND VESTIBULAR NERVES

Lesions of the cochlear nerve produce deafness, and in addition pathological changes in its peripheral termination are productive of tinnitus. Except from direct involvement of this nerve or of its terminations in the cochlea, deafness is practically unknown as a symptom of disease of the nervous system. In other words, lesions of the central auditory paths are not as yet recognisable by any known symptoms.

Nerve deafness may be produced by any lesions of the cochlea and cochlear nerve, and is confined to diseases of the temporal bone and labyrinth, lesions of the internervine part of the eighth nerve by tumours, meningitis or pressure, and lesions of the lateral side of the medulla. The deafness is the same wherever the lesion may be, and the position of the lesion is to be deduced from the associated involvement of contiguous structures. Nerve deafness, which characterises lesions of the cochlea and its nerve, is distinguished from deafness due to middle-ear disease by the facts that hearing both by air conduction and by bone conduction is diminished or lost, while in middle-ear deafness the hearing by bone conduction is increased. If a tuning-fork in vibration be applied to the forehead until it is no longer audible, and then presented to the ear, it will not be heard aerially in middle-ear disease, since the aerial conduction is impaired in that condition. But in nerve deafness it is either not heard through the bone when the tuning-fork

is applied, or if heard, when it has ceased to be audible to bone conduction, will still be audible when presented to the ear. This is known as Rinne's test, and it is a reliable one. Weber's test for nerve deafness consists in the application of a tuning-fork to the forehead in the middle line, the patient being asked which ear the sound comes to most. In middle-ear deafness the sound is heard best on the deaf side, and in nerve deafness it is best heard on the sound side. As a symptom of nervous disease, nerve deafness is met with in disease of the lateral region of the medulla, in tumours of the cerebello-pontal angle growing from the eighth nerve, following epidemic meningitis, and in syphilis of the nervous system, especially congenital syphilis.

1. TINNITUS

Ætiology.—Tinnitus or the occurrence of persistent recurring noise referred to the ears may be produced by wax in the ear, by otitis media, or by any other condition of vascular congestion, or by inflammation in the region of the auditory mechanism. It occurs in those who work exposed to deafening noise, as in boiler-makers and riveters, and may be produced by the administration of quinine and salicylates. It is much more frequently indicative of intractable disease of the cochlea, which often ends in complete deafness. Persistent tinnitus is a malady of adult life, the earliest cases occurring after puberty. It is rare for the malady to commence in old age. It begins insidiously, and as a rule without cause, but debilitating influences may precede its onset.

Diagnosis.—The diagnosis of tinnitus presents no difficulty. A careful examination of the ears will discover and cause to be removed any local trouble in the external auditory meatus and tympanum. Moreover, these conditions do not give rise to persistent tinnitus with nerve deafness.

Symptoms and Course.—The sounds commence faintly and often intermittently, and at first may be only perceived in stillness and silence at night, and later become louder and more persistent, and are often absolutely continuous. The slight sounds may be low pitched, a low rumble like a distant wagon, or a faint murmur such as may be heard when a shell is held to the ear. The loud sounds are never low in tone. They may be humming, hissing, rushing or bell-like noises. The common simile used by the patient is that of a hissing kettle, of a gas jet, of a threshing machine, of a steam-engine, or of a room full of machinery. The same patient may have several sounds, sometimes successive and sometimes heard all at once. When the sounds are rhythmical they are usually synchronous with the pulse. In some of the cases labyrinthine vertigo occurs, and the attack may be heralded by an increasing intensity of the sound. The condition of hearing in patients suffering with tinnitus may vary in each case, and from time to time in any one case. In many cases hearing is perfectly normal, and may remain so for years, in spite of increasing tinnitus. One of my patients retained perfect hearing for over twenty years, with increasing tinnitus; but the hearing rapidly declined afterwards. In many cases, however, there is some degree of nerve deafness on one or both sides. In the course of time the deafness increases even to absolute deafness, and in a few of them the noises persist in spite of absolute deafness. As a rule, the noises decrease as deafness becomes severe.

Prognosis.—The prognosis is very uncertain, and in most cases unfavourable. In many cases the noises persist in spite of all treatment, sometimes treatment secures considerable relief, and not infrequently the symptom is removed by treatment.

Treatment.—In the early stages the disease may be much benefited by the exhibition of salicylates and iodides. As a symptom, tinnitus is more affected by bromides than by any other drug, and these should be given in doses of from 10 to 20 grains twice or three times daily. The effect of the bromide is sometimes increased by the addition of from 5 to 10 minims of tincture of belladonna.

2. VERTIGO

Definition.—The word “vertigo,” which by derivation means a “turning,” is used to designate any movement or sense of movement or unsteadiness either in the individual himself (subjective vertigo) or in external objects (objective vertigo) that involves a defect, real or seeming, in the equilibrium of the body. It is a sensation of involuntary movement, either of subject or of external objects. It always involves a slight interference with consciousness, which, in severe vertigo, is often momentarily lost.

Ætiology.—Vertigo is always the result, direct or indirect, of disturbance of the labyrinth, vestibular nerves or cerebellum. It is commonly associated with vomiting and with vasomotor and secretory phenomena, such as “cold perspiration.” The disturbance of the vestibular mechanism which results in vertigo may be set up by multitudinous causes, among which may be mentioned toxic states as in specific fevers, and from the administration of alcohol, anæsthetics and morphine, irregularities of blood supply as in fainting, loss of blood, cardiac feebleness, Stokes-Adams’ disease, and sudden alterations of position and in arterial disease; from visual or bodily disorientation as in diplopia, dancing, swinging, sea-sickness and train-sickness; in anæmic states; in migraine, and as an aura in epilepsy; in diseases of the tympanum, labyrinth and semicircular canals; in diseases of the vestibular nerve and cerebellum, and in conditions of raised general intracranial pressure.

Diagnosis.—The vestibular mechanism is closely connected functionally with the cerebellum, and the symptoms which result from its disturbance are almost identical with those resulting from lesions of the lateral lobe of the cerebellum, and comprise nystagmus to the side of the lesion, vertigo, forced movements, hemiataxy and hypotonus on the side of the lesion. There are two points which serve to separate the two conditions. In the first place, vestibular lesions are usually associated with nerve deafness, which is absent in cerebellar lesions, and secondly, the cerebellar symptoms are only marked in vestibular lesions when the condition is acute, or during acute exacerbations.

Tests for vestibular lesions.—1. Barany’s calorice test is made by irrigating the external auditory meatus with either hot or cold water or air. With an intact vestibular mechanism this causes irritation of the vestibular apparatus with the appearance of nystagmus or lateral deviation of the eyes to the side of the irrigation. When the vestibular mechanism is impaired this test fails relatively or completely.

2. If the patient be rotated either by placing him in a special rotating chair, or by turning him round several times in the standing position, lateral

conjugate deviation of the eyes immediately after the rotation will show nystagmus in the opposite direction to the rotation, if the labyrinth on that side is intact. It will not appear if the functional activity of the vestibular mechanism is deficient.

3. MÉNIÈRE'S DISEASE

Synonym.—Labyrinthine vertigo.

Definition.—A malady in which paroxysmal attacks of severe labyrinthine vertigo occur at irregular intervals, associated with tinnitus and progressive deafness, and due to disease of the labyrinth of a chronic nature.

Ætiology.—The cause of this symptom-complex has always been obscure, and this is not surprising since until a recent careful pathological examination of the labyrinth in two cases of typical Ménière's syndrome by Hallpike nothing was known of its underlying morbid anatomy. Ménière's original hypothesis was that hamorrhage into the labyrinth as the responsible factor, but is inherently improbable and lacks pathological support. According to Hallpike the essential lesion is a gross distension of the endolymph system together with degenerative changes in Corti's organ and the presence of albuminoid coagula throughout the endolymph spaces. He regards these changes as incompatible with an infective origin, and as probably primarily degenerative in nature.

The correlation of these changes with the paroxysmal character of the vertigo can at present only be surmised, but Hallpike believes that the attacks are probably due to rapidly induced bouts of asphyxia of the labyrinthine end-organs caused by rapid rises of fluid pressure in response to small volume increases in the endolymph.

Symptoms.—The attacks set in suddenly with a buzzing noise in the ears, followed immediately with intense vertigo, both subjective and objective. The vertigo may be so intense that the patient feels he is hurled to the ground. He often falls as if shot; sometimes he has time to assume the sitting or lying position, before the vertigo reaches its height. Consciousness is often lost, or seriously impaired, for a few moments only. Spontaneous nystagmus occurs to the side of the lesion, and unilateral cerebellar signs on the side of the lesion. The patient becomes nauseated, and often vomits repeatedly. The skin is pale and covered with a clammy sweat. The patient lies perfectly still, and in terror lest the least movement should bring on more vertigo. The duration of the attack and the time taken in the recovery from an attack vary from a few minutes to 24 hours. Sometimes the attacks are excited by some sudden movement, such as coughing or sneezing, but they are usually without any such antecedent. They may occur during sleep, and wake the patient. The recovery from the attack is usually perfect, the vertigo disappearing; but in some cases slight persistent vertigo remains between the attacks. When Ménière's disease is persistent a slow onset of nerve deafness and signs of slow vestibular destruction follow, and as these signs deepen the attacks become less and less severe, and finally cease when the functions of the labyrinth become destroyed.

The **Diagnosis** of Ménière's disease presents no peculiar difficulty, for the symptoms are highly characteristic, and although the attacks vary in the degree of their severity, from a slight momentary giddiness to a sudden falling, with the most acute cerebellar symptoms, yet the first attack is usually

severe. The rapid disappearance of the symptoms is striking. Vertiginous attacks from all other causes must be excluded. In epilepsy consciousness is usually lost. In Ménière's disease it is momentarily impaired, and there is no convulsion. In acute cerebellar lesions the symptoms are very like those of labyrinthine vertigo, but they are not transitory in a few hours. A careful search of the nervous system for signs of organic nervous disease should in every case prevent any mistake.

Prognosis.—The outlook in Ménière's disease is uncertain. Some cases go from bad to worse in spite of treatment, and progressive deafness ensues with disappearance of the attacks. Many cases, however, recover perfectly with little or no impairment of hearing.

Treatment.—The salicylates seem to have a definite specific effect upon the morbid process, and should be given in doses of 20 grains thrice daily. In the form of aspirin they may be even more beneficial from the sedative effect of the latter drug. The bromides have a wonderful effect in relieving the symptoms, and in averting the attacks, to the extent that it may be said that labyrinthine vertigo may be almost diagnosed by the beneficial effect of bromides upon it. They should be given in doses of from 10 to 20 grains three times a day. Syphilis must be excluded, and if present, treated. Counter-irritation of the mastoid region has been recommended, and can certainly do no harm.

THE NINTH OR GLOSSOPHARYNGEAL NERVE

Lesions of this nerve involve loss of taste over the posterior one-third of the tongue with some unilateral paresis of the pharynx. It is rarely involved alone; but, with the other nerves taking origin in the neighbourhood, by tumours of the lateral region of the medulla.

THE TENTH OR VAGUS NERVE

This nerve is a mixed nerve. The motor fibres supply the voluntary muscles of the soft palate (except the tensor palati), pharynx and larynx in conjunction with the accessory fibres, and the non-striped muscles of the respiratory and alimentary tracts.

The sensory fibres of the vagus supply the respiratory tract, the pharynx and œsophagus. Its visceral fibres supply the lungs, heart and abdominal viscera. No sensibility seems to be supplied to the abdominal viscera by this nerve, since with division of the spinal cord above the offshoot of the splanchnic nerves all sensibility in the abdomen is lost.

LESIONS OF THE VAGUS.—The important signs of lesion of this nerve and its nuclei are pharyngeal and laryngeal paralysis and loss of sensibility. Symptoms indicative of lesions of its complicated and mysterious visceral supply are neither well marked nor well understood, and in unilateral lesions seem to be entirely absent; they are therefore not considered.

Lesions of the vagus in the medulla are common. Syringomyelia, when affecting that region, usually involves the nucleus ambiguus, causing unilateral palsy of palate, pharynx and larynx. Thrombosis of the posterior inferior cerebellar artery which supplies that region of the medulla containing the nucleus ambiguus is likely to produce vagus paralysis of the same side. Pro-

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gressive muscular atrophy, in the form of progressive bulbar paralysis, may affect its cells, as do often polyneuritis and lethargic encephalitis and rabies. Lesions of the nerve roots often occur from tumours of the lateral region of the medulla, and growths outside the medulla from nerve roots and meninges, and here the lesion of the vagus roots is associated usually with those of the glossopharyngeal, spinal accessory and hypoglossal. In the neck perforating wounds and growths may implicate the nerve, and in the thorax tumours, particularly aneurysms and new-growths, are apt to cause paralysis of the muscles supplied by its recurrent branches.

Unilateral pharyngeal paralysis.—This is characteristic of all unilateral lesions of the vagus high up. It is recognised by the low-lying motionless palate and the loss of sensibility of one side of the pharynx, with loss of the pharyngeal reflex on that side. There is no impairment whatever of deglutition.

Bilateral pharyngeal paralysis.—This results from nuclear lesions of the nucleus ambiguus on either side, and is common in diphtheria, polyneuritis, myasthenia gravis and progressive muscular atrophy. The whole palate is low and paretic or paralysed, the voice is nasal, there is nasal regurgitation of liquids, the cheeks cannot be forcibly blown out, and there is difficulty in pronouncing final “b” and “g,” the words “rub” and “egg” becoming “rum” and “enck.”

Total unilateral laryngeal paralysis.—Since the superior laryngeal nerve which supplies the cricothyroid muscle, which is the chief tensor and adductor of the vocal cords, is given off high in the neck from the ganglion of the trunk of the vagus, it follows that total paralysis of the larynx on one side can only result from a lesion of the vagus, between the ganglion of the trunk and the nucleus ambiguus in the medulla. The vocal cord on the paralysed side is motionless in the cadaveric position—that is, midway between abduction and adduction. The larynx is insensitive on the same side. There is some loss of tone of voice but no stridor.

Unilateral abductor paralysis or recurrent laryngeal paralysis.—This occurs from all lesions of the trunk of the vagus below the ganglion of the trunk, and from lesions of the recurrent laryngeal branch. The vocal cord on the side of paralysis lies close to the mid-line. It fails to abduct on taking a deep breath. There is no change of voice; but there may be slight stridor on inspiration—the sensibility of the larynx is not affected.

Bilateral abductor paralysis.—This condition is most commonly seen in the earlier stages of nuclear laryngoplegia, and is most often met with in tabes, sometimes in bulbar paralysis, and we have seen it in disseminated sclerosis. It occurs also in bilateral lesions of the recurrent laryngeal nerves in the thorax, which may occur from aneurysm and new-growths. It is the most dangerous form of laryngeal palsy, as the vocal cords cannot be abducted from close to the middle line, and they tend to open during expiration, but to suck together during inspiration, and for this reason may cause death from asphyxia, or necessitate laryngotomy.

THE ELEVENTH OR SPINAL ACCESSORY NERVE

This nerve may be caught with the vagus by lateral lesions outside the medulla, or by lesions in the region of the jugular foramen; but it is more

often damaged by injuries to the neck, and by operations for the removal of cervical glands. The spinal accessory nerve, as it crosses the posterior triangle of the neck, is very liable to injury, either from blows or from sudden strains, and most of the isolated trapezius palsies are due to local neuritis of the nerve trunk, so arising. Paralysis and wasting of the sternomastoids is conspicuous in most cases of myotonia atrophica. That of the trapezius is often conspicuous in the facio-scapulo-humeral type of myopathy. Both muscles are commonly affected in progressive muscular atrophy.

When the sternomastoid is paralysed there is neither weakness complained of, nor deformity, nor peculiar attitude of the neck, other muscles compensating for its paralysis. The muscle does not harden when turning the head to the side opposite the paralysis, and its reaction to faradism is diminished or lost.

Paralysis of the trapezius, on the other hand, causes great disability in raising the arm above the horizontal level of the shoulder and also difficulty in shrugging the shoulder or approximating the scapula to the middle line behind and therefore also in carrying the extended arm backwards. It produces a very ugly deformity, for the scapula unsupported by the trapezius rotates so that the superior internal angle appears as a hump in the slope of the neck above the clavicle, and there is also winging of the angle of the scapula with the axillary border of that bone horizontal. This paralysis of the trapezius may be confused with that of the serratus magnus, for in both winging of the angle of the scapula is marked. In trapezius palsy, however, the deformity is much more marked, the scapula is farther away from the spine, and is much more rotated. Tests for the movements of these two muscles and the faradic excitability should prevent any confusion.

COMBINED LESIONS OF THE NINTH, TENTH, AND ELEVENTH NERVES

No account of the glossopharyngeal, vagus and accessorius nerves is complete which does not consider their clinical interrelationships. Not only are they closely associated in part of their peripheral course, but their central origins are very intimately connected. It is therefore not surprising that in both central and peripheral nervous lesions two or more of them may be involved. A number of characteristic syndromes have thus been observed and described. We may classify these according to whether the causative lesion is intramedullary, or extramedullary (at the base of the skull, or in the neck).

Intramedullary lesions include thrombosis, nerve cell degeneration (chronic bulbar palsy), and syringobulbia; and its component symptoms (*syndrome of Avellis*) are unilateral paralysis of the palatal, pharyngeal and laryngeal muscles, with a crossed hermanæsthesia of syringomyelic type (for pain and temperatures).

At the base of the skull injuries or new growths involving the jugular foramen may give rise to *Schmidt's syndrome*: unilateral paralysis of pharyngeal, laryngeal, palatal, sternomastoid and trapezius muscles; or to *Jackson's syndrome*, in which a hypoglossal palsy may be added to those of the above syndrome.

Injuries high in the neck may produce *Tapia's syndrome* (first described

in bull fighters from penetrating wounds caused by the bull's horn), which consists in unilateral paralysis of the vocal cord and tongue, the palate being intact.

THE TWELFTH OR HYPOGLOSSAL NERVE

The nerve supplies all the muscles of the tongue, both intrinsic and extrinsic.

Unilateral lesions of the hypoglossal nerve are usually the result of tumours in the lateral region of the medulla, or local lesions just lateral to the medulla, and catching the nerve roots. Hemiatrophy of the tongue is perhaps more commonly seen in tabes than in any other condition. A hemiatrophy also occurs in cases of facial hemiatrophy, where the lower distribution of the fifth nerve is the region affected; but this variety does not involve paralysis of the tongue. Spastic paralysis of the tongue, with well-marked dysarthria and dysphagia, occurs in double hemiplegia and amyotrophic lateral sclerosis. Atrophic paralysis of the whole tongue, with exactly similar defects of articulation and swallowing, occurs when the hypoglossal nuclei are affected, and is commonly seen in progressive bulbar paralysis and sometimes in polyneuritis and myasthenia gravis. The sole physical sign of a lesion of one hypoglossal nerve is atrophic paralysis of one side of the tongue with loss of faradic excitability. The affected side of the tongue shrinks and comes in the end to consist solely of mucous membrane, fibrous tissue and glands. The tongue becomes sickle-shaped, with the concavity on the paralysed side. There is little impairment of movement, and no defect of articulation from a unilateral lesion.

The treatment is that of the condition causing the paralysis.

TRIGEMINAL NEURALGIA

Synonym.—Tic Douloureux.

Definition.—A disease of the fifth cranial nerve, in which no definite morbid changes in the nerve have been discovered, and in which no loss of function, either motor or sensory, occurs in the distribution of the nerve. The chief feature of the malady is the occurrence of pain of varied intensity which tends to be paroxysmal, and is often excruciating. Tenderness over the branches of the fifth nerve is always present during the bouts of pain, and when the third division of the trigeminal nerve is affected there is conspicuous unilateral furring of the tongue when pain is present.

Ætiology.—The malady is first met with at the age of puberty; it is not seen in childhood. In the earlier years of adult life it is often a mild and curable condition, though notable exception to this rule may occur; but as age advances, and especially after the age of 50 years, it tends to be increasingly severe and intractable by any measures save those for the destruction of the affected branch of the nerve, or of the Gasserian ganglion. No causal factors can be adduced. The sexes are equally affected. It is much more common in cold and damp climates than in southern and dry countries. Any debilitating influences, such as overwork, general ill-health and specific fevers, especially influenza, may precede the onset of the malady.

Symptoms.—The chief feature of the malady is pain, which may be general throughout the area of distribution of the nerve, but which is more commonly confined to one of the three divisions of the nerve and often to one branch of a division. It is characteristic for the pain of neuralgia to commence locally, and subsequently to spread in each attack and gradually, in the course of the disease, permanently to invade a larger area. Two different kinds of pain occur, the sharp and paroxysmal, and the dull and continuous pain. The paroxysmal pains are sudden in onset and in cessation. They have a lightning-like character, and are described as piercing, knife-like, or as if the affected region were penetrated by red-hot wires. Often quite spontaneous, these pains may be brought on by touching the surface, by a cold draught, by movement of the face and jaw, or by the act of swallowing, and in this last condition mastication and deglutition may become so difficult as to render feeding the patient a matter of great anxiety. When the paroxysms are occurring in a severe case the patient remains for a period, which may be from a few minutes to several hours, paralysed under the fear of the pain, unable to move a muscle lest a spasm more dreadful than the last should occur. The paroxysmal pains are usually followed, if severe, by a more lasting dull continuous pain often of a boring character, and sometimes such pain becomes absolutely continuous. The skin over the affected region is sore and tender after the paroxysm, and the patient may be unable to bear brushing the hair or shaving the face. The pain may be of every degree of severity, from mild momentary starts to continuous incapacitating pain, interrupted only by excruciating attacks of agony which render life a piteous burden. The distribution of the pain may be anywhere or everywhere in the distribution of the trigeminal nerve. The lightning-like onset of the agony often causes convulsive spasm of the face and of the body and limbs, and from this feature the names "tic douloureux," "spasmodic neuralgia," and "epileptiform neuralgia" arose. The tender points of Valleix are constantly present during the attack, and for some little time after. When the first division is affected, the tender points are found above the supra-orbital notch, over the external angular process, on the upper outer aspect of the nose, and on the globe of the eye. When the superior maxillary division is affected the chief tender point is over the infra-orbital foramen, while other points may be found over the points of exit of the temporo-malar nerves and in the roof of the mouth. When the third division of the nerve is involved, the chief tender points are over the mental foramen, the side of the tongue, and just in front of the external auditory meatus. When the third division is affected, unilateral furring of the tongue, which always occurs when the pain is present and which does not seem to occur with organic lesions of the fifth nerve, nor constantly in any other malady except neuralgia, is seen. Vasomotor and secretory disturbances are common. During the paroxysms, tears and saliva may flow in abundance. The trophic changes which have been described in the skin are usually the result of rubbing during attacks of pain, or of the application of heat or liniments. Local greying of the hair, however, does undoubtedly occur. The clinical picture of trigeminal neuralgia is completed with varying degrees of general physical ill-health, mental apathy and depression, which occur in proportion to the frequency and severity of the attacks, the presence of continuous pain, the ability to

take food and to sleep, and the possibility of taking any interest in life. It is surprising in England how few of the sufferers from severe neuralgia become habitual drug-takers.

Course.—In patients under the age of 40 years the malady is often transient and is completely and permanently recovered from, though even at this age cases occur which are only amenable to surgical interference. But when the malady commences after the age of 40 years, it is the rule for it to become progressively worse. The paroxysms become more severe, and occur at shorter and shorter intervals, continuous pain sets in, sleep and the taking of nourishment become difficult, and useful life becomes more and more restricted.

Diagnosis.—There should be no difficulty in making a correct diagnosis if proper care be taken. In the first place, all local cause for pain in the peripheral distribution of the trigeminal nerve should be excluded. The teeth should be most carefully examined and the jaws skiagraphed for any concealed disease, which should be put right if present. Organic disease of the fifth nerve can be excluded by the facts that such disease cannot long exist without signs of loss of function, which never occur in neuralgia. Diminution of sensibility, which is first marked perhaps by increased tolerance of the conjunctiva and cornea to touch, and weakness of the musculature with deviation of the chin on opening the jaw, and diminution of taste are certain signs of a local organic lesion. Moreover the pain of neuralgia, with its lightning onset and cessation, is hardly imitated by any pain of organic origin. Ocular conditions, such as glaucoma, which may give rise to agonising pain, can hardly be mistaken for neuralgia.

Treatment.—Having in the first place seen that all possible causes of local irritation in the region of distribution of the fifth nerve are absent, or, if present, adequately dealt with, it is essential to improve the nutrition and general physical health with tonic, dietetic and hygienic treatment, and such remedies alone will often cure slight cases. It is important to remember that in its early stages, the malady shows complete remissions of long duration. These remissions do indeed tend to become shorter after some years, but their occurrence suggests that in planning treatment it is essential to consider the circumstances of each individual case. Thus, if a patient who may be expected to enjoy a long period of freedom from pain can be tided over the present attack by medical means, it is clearly not wise to give an alcohol injection. This confers a long period of "cover" from pain which the patient will probably not require, and the premature recourse to injection means that in the end more injections may be called for than would otherwise have been needed.

With this qualification, treatment may be undertaken on the following lines: *Tr. gelsemii* in doses of from 10 to 20 minims thrice daily is an admirable remedy, and arsenic is a useful adjuvant. All the analgesic antipyretics of the coal-tar series are of great value, not only as immediate relievers of pain, but also as curative agents, and among these aspirin is most important. In cases where malaria has been recently present, quinine should never be omitted. In very severe cases, and when operation is to follow, morphine is an invariable temporary relief to the pain. But if persisted in, the beneficial effects of moderate doses soon disappear. In every case except in old subjects, a thorough trial of the above treatment should be made over a

sufficient period to make a competent judgment of its efficiency or inefficacy, as the case may be. When failure is met with, and in old subjects, who will be found to respond little if at all to such treatment, operative relief should be sought. In the first place, the injection of alcohol should be performed, and if this fail, as it sometimes does, on account of anatomical peculiarities of the individual, recourse should be had to the operation for dividing the fifth nerve proximal to the Gasserian ganglion. The permanence of the effect of alcohol injection varies, sometimes lasting relief is obtained; more often, after a period which varies from months to years, some return of the pain occurs. It is, however, a most difficult procedure for the operator, and requires great skill and experience. While absolutely devoid of risk in skilled hands, alcohol injection should never be undertaken by one who has not special training in its performance. The radical operation produces final cure.

GLOSSOPHARYNGEAL NEURALGIA

Definition.—A comparatively rare form of neuralgia within the distribution of the glossopharyngeal nerve. It is strictly comparable with trigeminal neuralgia in the quality and severity of the pain, its paroxysmal incidence, the remissions in its course, its provocation by special stimuli, and finally by the absence of any discoverable lesion in, or loss of function of, the nerve.

Ætiology.—Nothing is known of its ætiology. It is most frequently seen in middle-aged or elderly males. A symptomatic neuralgia of the same distribution is occasionally found in cases of carcinoma of the tongue in which the growth invades the faucial region.

Symptoms.—When fully developed, the malady consists in paroxysms of shooting pain of great severity in the region of the throat and ear. The exciting stimulus is commonly the act of swallowing. But just as in trigeminal neuralgia the pain may at first be confined to a single branch of this nerve, so in glossopharyngeal neuralgia, the pain may for long be confined to the tympanic branch, the pain being felt deep in the ear. This pain does not spread to the pinna. In other cases, pain in the faucial region predominates, the pharyngeal branches being affected. As in trigeminal neuralgia, the patient may enjoy long intervals of freedom from pain. During a paroxysm the patient screws up his face and may hold his head in his hand as does the subject of trigeminal neuralgia.

Diagnosis.—The presence of neuralgic pain of great severity, provoked by the act of swallowing, and in its general characters and behaviour resembling the very familiar and characteristic paroxysms of trigeminal neuralgia, but differing from these in its restriction to the ear and throat, occurring also in the absence of objective signs of a lesion of the cranial nerves: these together are the features which make a diagnosis of glossopharyngeal possible and easy.

Treatment.—In the early attacks, the same forms of medication employed in trigeminal neuralgia may be employed. If the pain does not respond to these, then surgical measures are called for. The tympanic branch of the nerve leaves the main trunk within the skull, so that when pain in the ear is present an intracranial section of the glossopharyngeal

nerve is necessary. On the other hand, when pain is confined to the distribution of the pharyngeal branches, division of the nerve high in the neck is adequate. But intracranial section appears to be the operation of choice.

FACIAL HEMIATROPHY

Synonym.—Parry-Romberg syndrome.

Definition.—A peculiar malady confined to some part of the distribution of the trigeminal nerve, or rarely extending from thence on to the area of sensory distribution of the upper four cervical nerves. It is characterised by a progressive atrophy of all the tissues, skin, subcutaneous tissue, muscle and bone, without sensory loss or paralysis. It comes to an arrest after a few years. No pathological condition has been discovered to account for the atrophy.

The disease may commence in childhood even as early as the second year, but it is most commonly started in early adult life. Females are much more often affected than males.

Symptoms.—The atrophy may be distributed over the whole area of the supply of the trigeminal nerve, or, as is more usual, may be confined to one or more of its branches. In general atrophy, a gradual diminution in the bulk of the whole side of the face is the first indication of the disease. When the disease is confined to one of the three great divisions of the nerve, the atrophy usually commences in one spot, commonly on the cheek just below the malar bone, where the skin becomes thin and pale from loss of pigment, and the down falls out. The submalar fat disappears, leaving an unsightly hollow. The atrophy spreads to the side of the nose, where the cartilages and bones become gradually smaller. The jaws gradually decrease in size upon the affected side, until they are too small to hold the teeth, which are actually pushed out by the decreasing size of the tooth sockets. The half of the tongue upon the affected side decreases in size, and thereby is rendered sickle-shaped. Even the eye may be remarkably lessened in size. The upper part of the first division of the fifth does not seem so liable to involvement, for it is rare to see any diminution of the size of the forehead, or dropping out of the hair of the scalp. The atrophy not uncommonly affects the ear. The skin in the end becomes very thin and parchment-like.

Treatment.—The only treatment is cosmetic, to improve somewhat the appearance of the face, by the injection of semisolid paraffin, to replace the fat and fill the unsightly submalar hollow.

THE SIGNS OF LOCAL LESIONS WITHIN THE SKULL AND BRAIN

Owing to the complete inaccessibility of the central nervous system to direct examination by any method comparable with those in use in the case of the viscera, the clinical localisation of disease within that system must necessarily depend upon the study and interpretation of disorders of function in tissues innervated by the nervous system. Many bodily functions have a localised representation in the brain and in the spinal cord. It must be remembered, however, that in clinical diagnosis we are concerned not directly with the localisation of functions within the nervous system, but with some-

thing rather different, namely, the localisation of symptoms of lesions. A simple example will serve to make this distinction clear. In a case of unilateral ataxy of movement, our object is to locate the lesion which by damaging some part of the nervous mechanism has allowed ataxy to develop. We may decide from our examination that this lesion is within the cerebellum, and we conclude that a destructive lesion of this organ is followed by ataxy. This ataxy is clearly produced by the activity of the intact remaining parts of the brain, working without the co-operation of the cerebellum. In this instance we have not localised any "function" of the cerebellum; we have simply localised the symptom following a lesion of this organ. Nor can we conclude that one of the functions of the cerebellum is to prevent ataxy, the fact being that the functions of this organ are still very imperfectly understood. Nevertheless, the localisation of symptoms of cerebellar lesions can be performed with reasonable accuracy.

This brings us to a brief consideration of the ways in which lesions within the nervous system may disturb its functions. The functions of a region of the brain that is directly involved in a disease-process may be deranged in either of two ways. They may be stimulated to overaction, or they may be impaired or destroyed. We may thus speak of "*irritative*" or *excitatory symptoms* on the one hand, and of *paralytic symptoms* on the other. A Jacksonian fit is an example of the first; a hemiplegia of the second. Further, although there is some measure of localisation of topographical and of functional representation within the brain, normally this organ works as a whole and derangement of the functions of one region may derange the functions of the whole, as we have already seen illustrated in the case of cerebellar ataxy. There is another way in which such general disturbance may follow a local lesion, and that is by what is known as diaschisis or shock. We see this mode of disorder in the coma which accompanies a cerebral hæmorrhage. In this state the cerebral hemispheres are for the time being out of action, even those parts that are not actually damaged by the lesion. Such *shock symptoms* are necessarily transient. A final group of symptoms are those we speak of as "*release symptoms*." When the coma of the hemiplegic subject has passed off, he is left with paralytic symptoms, namely, the hemiplegia. In a few weeks the paralysed limbs become spastic, their tendon jerks increase, and clonus makes its appearance. These symptoms of persistent overaction of nervous mechanisms freed by the lesion from the normal control of higher mechanisms, are what we refer to when we speak of release symptoms. Such symptoms may persist for years and may in some instances entirely dominate the clinical picture. But the practical task of localising lesions is sometimes even more complicated than this analysis of disorders indicates. The degree of disturbance of function produced by any lesion depends also on temporal factors. A suddenly arising lesion, such as an arterial occlusion or a hæmorrhage, or a direct injury is apt to produce a much more severe and widespread disorder of brain function than a slowly developing lesion. Thus, the intracranial cavity may come to accommodate a large new growth which compresses and markedly deforms the brain without giving rise to any subjective discomforts or disabilities, or to any abnormal physical signs discoverable on examination. Again, it is known that a chronic cerebral or cerebellar abscess is commonly present for some weeks before it reveals its presence by signs or symptoms. This is its period of clinical latency. Finally,

a tumour within the brain, while it may give rise to symptoms of a general rise of intracranial tension, such as headache, papilloedema and sickness, may yield on examination no localising signs, and this not necessarily because it is in what is known as a "silent area" of the brain. Or, it may cause but a minimal disturbance of local function even when large regions of known and specific function are directly involved. Finally, we have to take into consideration that space-occupying lesion within the skull or brain may come ultimately to produce indications of local disorder of function in parts of the brain remote from the lesion. These may be spoken of as false localising signs.

From what has been said it will be apparent that at least two factors determine the symptoms associated with disease within the brain, namely, (i) the localisation of the lesion, and (ii) the nature of the lesion. The latter determines its rate of development, its stimulating or paralysing effects upon the nervous tissue, and its capacity for producing remote effects.

Hence it is that the localisation of a lesion within the brain (the topographical diagnosis) and the determination of its nature (the pathological diagnosis) are frequently something more than a simple essay in applied anatomy and physiology, and that complete diagnosis calls also for a knowledge of the natural history of the different disease-processes, that is, for clinical experience. In this chapter we must be content with a brief consideration of the signs upon which we depend for the localisation of symptoms. We may take first the various regions of the brain, and secondly, since we have to deal not only with lesions within the brain, but also with all lesions within the skull, that may be outside the brain, we will consider the symptomatology peculiar to lesions in the three cranial fossæ.

THE CEREBRAL HEMISPHERES

General lateralising signs.—A lesion within or involving one hemisphere may reveal by the signs it produces whether it is right- or left-sided without affording further localising information. Such signs are unilateral diminution or absence of the abdominal reflexes, a unilateral extensor type of plantar response, and a just perceptible unilateral paresis of movements of the lower part of the face.

The frontal lobes.—These include that part of the hemisphere anterior to the ascending frontal convolution. The lesions to be met with in this region include tumour, abscess and thrombosis of the anterior cerebral artery, the last named being comparatively rare.

The syndrome of the anterior cerebral artery consists of spastic weakness of the crossed lower limb with the appropriate changes in the reflexes, sometimes a slight degree of weakness of the crossed face and arm, sometimes forced grasping and groping in the arm of one or both sides, and apraxia of the left arm. There may also be some mental obfuscation.

The syndromes of frontal lobe tumour vary according to the rapidity of development of the tumour and under other factors not fully understood. As a rule an early, if not the initial, symptom is a change in the patient's mental state. He becomes apathetic and lacking in initiative. The association and flow of ideas tends to fail. He sits about idly. He is apt to permit the unhindered passage of urine and even of feces, and to be totally indifferent to and unaware of the social embarrassments such conduct involves. This

form of "incontinence" is in fact a diagnostic symptom of great value in frontal lobe lesions. Rarely, the patient develops an abnormal facetiousness and euphoria—the so-called "Witzelsucht." Movement is disordered by the development of apraxia, and sometimes by that of forced grasping and groping, another useful sign of frontal lobe involvement, though it must be admitted one occasionally seen in lesions elsewhere in the cerebral hemispheres, as in a case under the care of the present writer in which bilateral grasping and groping and forced sucking were prominent features, the lesion being found at necropsy to be a bilateral degeneration of the thalamus. Nevertheless, this sign is an important one. It has been analysed by Walshe and Robertson into two components: (1) Volitional grasping movements made by the conscious patient when some object is felt by him in his palm or is seen by him to approach his hand. These movements wane and cease when consciousness is failing, or when attention is defective. (2) A true tonic reflex grasp of any object held in the hand, if this object be so pulled away by the observer (or by the patient with his other hand) so as to put the flexors of the fingers on the stretch. The flexors tighten as the pull is maintained and may become of great force, so strong indeed that sometimes the patient can be pulled out of the bed by this involuntary grasp which he is unable voluntarily to relax. This reflex may persist even though consciousness be lost. If the orbital lobule be involved, there may be bilateral anösmia and even direct pressure upon one optic nerve. These two symptoms will be further considered in connection with the syndrome of the anterior fossa of the skull. The tumour being an expanding and space-occupying lesion may when in this situation lead to the appearance of slight crossed hemiparesis, and when left-sided may be accompanied by motor or "expressive" aphasia. When, in the case of tumour, the corpus callosum is involved, the patient becomes completely apathetic and silent and immobile, displaying no initiative of any kind.

Syndromes of the central region (region of the "motor cortex.")—Hemiplegia is the characteristic paralytic manifestation of a lesion, Jacksonian fits of an "irritative" lesion. Such a fit may be followed by transient hemiparesis, and in the case of tumour by a slowly progressive and permanent hemiplegia. Local involvement of some part of this region will affect face, arm or leg predominantly according to its situation.

Conjugate deviation of the head and eyes may be met with, away from the side of the lesion, if this be irritative, as at the commencement of an apoplexy, or of a local fit, or towards the side of the lesion if the lesion be paralyzing. The two forms of conjugate deviation often follow the one after the other, from the same lesion which is at first exciting and afterwards paralyzing. Conjugate deviation of the head and eyes is seen with acute lesions rather than with those of slow development. The more deeply the lesion extends into the sublying white matter, the more does it tend to produce an extensive hemiplegia, since the pyramidal fibres converge from the cortex towards the capsule. Loss of localisation to sensory stimuli is not infrequent from simultaneous involvement of the neighbouring post-central convolutions.

Parietal lobe.—A characteristic series of sensory disorders may mark the presence of a lesion in this region. These include defective localisation of tactile stimuli, defective appreciation of two simultaneous contacts (Weber's compass test), defective appreciation of three dimensional space

(i.e. of size and form). There is, in addition, defective power of differentiating varying intensities of stimulus (painful or thermal), and a ready fatigue of sensory functions. The simple recognition of painful and thermal stimuli may be relatively intact. It will be seen that the defects in spatial discrimination which result from these modes of sensory loss lead to that inability to recognise and identify objects held in the hand, or to describe their size, shape or texture, which is known by the name of astereognosis. The appreciation of active movement and passive position is apt to be faulty, and some ataxy may result therefrom. Trophic changes may be observed in the periphery of the limbs, and lesions in this situation seem to be responsible for the arrest of growth which is seen in cases of infantile hemiplegia. Jacksonian attacks, consisting of a peripheral sensory aura, sometimes followed by convulsions, occur. These localising signs are confined to the opposite side of the body.

Occipital lobes.—Lesions of the cuneus and region of the calcarine fissure on the mesial aspect of the occipital lobe result in hemianopia of the opposite field, but central vision escapes. Gordon Holmes has found that if the lesion is limited above the calcarine fissure a quadrantic hemianopia of the lower field results, and if the lesion is below the calcarine fissure the quadrantic hemianopia resulting is of the upper field. Since central vision is represented at the posterior pole of the hemisphere, a lesion of the posterior pole causes central hemianopic scotoma, vision in the periphery of the field remaining intact. Consequently a bilateral lesion of both posterior poles will result in bilateral central scotoma, and a bilateral lesion of the calcarine region will produce blindness of both peripheral fields, central vision remaining intact. If the lesion extends deeply into the occipital lobe so as completely to sever the optic radiation to the occipital cortex, complete hemianopia, affecting both the central and peripheral part of the visual field, will occur. The hemianopias resulting from lesion of the occipital lobe are distinguished from those due to lesion of the optic tract by the fact that in the former the pupil reacts to light thrown on to the blind part of the field (Wernicke's hemianopic pupil phenomenon). On the outer surface of this lobe, a lesion extending deeply on the left side may sever the connection of the visual centres with the speech centres, and so produce word-blindness. Such a lesion is usually situated at the junction of the left occipital and temporal lobes. Jacksonian attacks are often of great value in occipital localisation, and take the form of visual hallucinations, often accompanied by transient hemianopia.

Temporal lobe.—The uncinate and hippocampal regions of this lobe are the cortical seats for taste and smell, and the localising symptoms which are rarely absent when lesions in this region exist are Jacksonian attacks in the form of hallucinations of taste and smell, nearly always of an unpleasant nature. The hallucination is often immediately followed by a "dreamy stare," during which smacking movements of the lips, or clamping movements of the jaw, or spitting may occur. Experience seems to prove that all highly organised hallucinations, whether auditory, visual or psychic, when occurring from organic lesion of the brain, point to a lesion in the uncinate region. The senses of taste and smell are not lost from a unilateral lesion of this region, since they are bilaterally represented in the cerebral hemispheres. The outer surface of the temporal lobe is concerned with hearing, but from

the complete semi-decussation of the auditory path, unilateral lesions never produce detectable deafness. On the left side, however, the temporal lobe is concerned with speech, and destruction results in serious disorder of speech functions. Inasmuch as lesions of this region are situated far forward toward the insula, they result in "verbal aphasia," or towards the centre of the convexity of the temporal lobe, in amnesia, or lack of recall of words and "word-deafness," while if towards the posterior limits of the lobe they produce "word-blindness" from severance of the visual path to the speech region. Deeply seated lesions isolating the speech region from the incoming auditory path produce jargon aphasia. Jacksonian attacks, consisting of auditory hallucinations, which may or may not be followed by aphasia or by convulsions, may occur. Extensive lesions of the left temporal lobe cause much mental impairment. On account of the wide excursion which the optic radiation makes into the deep part of the temporal lobe in its course from the thalamus to the cuneus, homonymous hemianopia, especially of the upper quadrants, is very common in deep-seated lesions of the temporal lobes. The occurrence of incontinence of sphincters of a mental type is occasionally seen in some cases. When lesions extend deeply there may be a paresis of the opposite face for emotional movements, out of all proportion to the loss of volitional movements.

Internal capsule.—In this region, the chief motor tract is condensed into a small space, and is situated immediately in front of a narrowly localised sensory tract, while not much farther, posteriorly, the visual path enters the thalamus. Lesions of this region therefore produce severe and widely-spread hemiplegia of the opposite side, often associated with hemianæsthesia and not infrequently with hemianopia of the opposite side. From the proximity of the thalamus and corpus striatum, there is often involvement of these structures in a capsular lesion, with appearance of the characteristic, spontaneous involuntary movements and sensory loss.

BASAL GANGLIA

Optic thalamus.—A very characteristic clinical picture results from destruction by thrombosis of this structure which is termed the "thalamic syndrome" of Dejerine and Roussy; there is hemiparesis with spontaneous involuntary movements of the opposite side, which may be of the nature of tremor, intention-tremor, choreic, athetotic, dancing or irregular movements. Most post-hemiplegic involuntary movements are due to a lesion of the thalamus. In addition, there is hemianæsthesia, often with a characteristic hyper-sensitivity to aggressive stimuli, such as tickling, cold water, etc., which may produce agonising distress. Sometimes spontaneous, constant and unrelievable pain occurs on the opposite side. Emotional movement of the opposite face may be impaired much more than is volitional movement. The thalamic syndrome is not invariably, or even commonly, seen when the lesion is a tumour. In this case, as Smyth and Stern have pointed out, the symptom-complex varies according to whether the growth primarily arises in this structure or invades it from its lateral aspect. In the former case they arise in the sub-ependymal glia and spread laterally. They are characterised by early mental deterioration, with conjugate ocular palsies. Sensory changes are absent or only terminal in appearance. In

the case of tumours secondarily invading the thalamus from its lateral side, sensory changes of the order described under the "thalamic syndrome" of Dejerine and Roussy, Head and Holmes, are seen.

Corpus striatum.—Little is certainly known of the symptomatology of focal lesions of this structure, and the various syndromes (tremor-rigidity, athetosis) which have been described are associated with diffuse lesions involving other parts of the cerebral hemispheres in addition to the corpus striatum. However, a local lesion (thrombotic) in a neighbouring mass of grey matter, the corpus subthalamicum, or corpus Luysii, is followed by violent unilateral choreiform movements, the so-called *apoplectic chorea*.

Region of the falx cerebri.—Lesions of this structure are likely to affect both hemispheres equally. Tumours opposite the paracentral lobules cause bilateral crural monoplegia, and those in the posterior region of the falx, bilateral hemianopia. Thrombosis of the superior longitudinal sinus produces widely spread bilateral softening of the hemispheres, with double hemiplegia.

Corpora quadrigemina.—The oculo-motor nuclei lie on either side of the aqueduct of Sylvius, and lower down on either side of the middle line, in the floor of the upper part of the fourth ventricle, and lesions of this region cause nuclear ophthalmoplegia—that is, paralysis of both eyes in terms of the conjugate movements upwards, downwards or laterally. From before backwards, lesions of this column of oculo-motor nuclei will produce reflex iridoplegia, paralysis of accommodation, paralysis of upward, downward and lateral movements respectively. Immediately ventral to the oculo-motor nucleus and decussating beneath it, lie the superior peduncles of the cerebellum, involvement of which causes bilateral ataxy of limbs and trunk. Lesion of the dorsal part of the quadrigeminal layer produces a characteristic syndrome of nuclear ophthalmoplegia with bilateral ataxy, which is termed Nothnagel's syndrome. The pyramidal fibres for the face leave the pyramidal tract in this region of the tegmentum and may here be involved alone, causing bilateral spastic paralysis of the face. In the ventral portion of this region of the brain stem are the crura cerebri with the third nerve, perforating each crus to emerge upon its inner side, and the optic tract running round the crus from the geniculate bodies to the optic chiasma. A lesion of one crus will cause hemiplegia of the opposite side, and paralysis of the third nerve on the same side. This pathognomonic localising combination is known as Weber's syndrome. Situated a little more dorsally, a lesion of the crus will produce ophthalmoplegia of one eye with tremors and inco-ordination of the opposite limbs. This is known as Benedikt's syndrome. Extension of a lesion outwards from the crus will cause tract hemianopia, in which the half-fields are completely involved, with no light reaction from the blind fields. Interference with the fillet may cause hemianæsthesia.

PONS AND MEDULLA

In these regions the motor and sensory tracts, the cerebellar peduncles, the cranial nerve nuclei, and the outgoing cranial nerves are closely packed together, and the signs resulting from destruction of these will be varying combinations of spastic paralysis, ataxy and sensory loss—from interference with the long conducting tracts—in the body and limbs, with

nuclear and peripheral nerve palsies and anæsthesia in the region of the face. If the lesion is unilateral the body and the face will be affected on opposite sides, causing the "crossed paralyses" or "alternate paralyses" of lesions of the brain stem, of which facial palsy with contralateral hemiplegia, trigeminal palsy and sensory loss, and vagal palsy with contralateral hemiplegia are usual varieties. From the smallness of the brain stem lesions most often involve both lateral halves of this structure, and bilateral symptoms result. Lesions of the brain stem below the oculo-motor nuclei, cause small pupils (pontine myosis) from cutting off those nuclei from the spinal cord, whence the tonic dilator of the pupil—the cervical sympathetic system—emerges. Glycosuria may occur from interference with the vasomotor centre, and involvement of the respiratory centre is frequent. The common lesion involving the medulla is softening of the lateral region following thrombotic occlusion of the posterior inferior cerebellar artery, the so-called cerebellar apoplexy (see p. 1602).

CEREBELLUM

When lesions of this structure develop suddenly they are apt to produce more striking disturbances of movement than when they develop gradually, a point which it is important to remember when the presence of an abscess or a tumour within the cerebellum is suspected. Further, it is well to regard the several different components of cerebellar ataxy not so much as special disorders of different cerebellar functions, but as expressions of a single disorder, which owe their varying appearance to the varying nature of the clinical tests employed. The current nomenclature of cerebellar symptoms is redundant and complicates description, but it is universally employed and so is here adopted, but with no conviction of its scientific value.

Nystagmus.—In unilateral lesions there is a coarse nystagmus on deviation of the eyes to the side of the lesion, with a finer and more rapid movement on deviation away from the side of the lesion. In extensive lesions, there may even be considerable difficulty in deviation to the side of the lesion, and in this condition a true nystagmus may be present only on looking to the opposite side. In bilateral lesions the nystagmus may be symmetrical, but it may be entirely absent. Rarely—usually after acute lesions—such as gunshot wound or operative interference—the phenomenon of "skew deviation" may appear temporarily; the eye on the side of the lesion being displaced down and in, the opposite eye upwards and outwards.

Hypotonia.—This is usually seen in acute lesions rather than in chronic ones, and consists of a marked flaccidity and extensibility of the limb muscles, and leading to the "pendular" form of knee jerk.

Dysdiadochokinesis.—This is a clumsiness and slowness in the performance of rapidly alternating movements (pronation-supination), although the single movement can be normally performed. In carrying out this test, it is common to see adventitious movements of the limb occur, the whole limb being in movement.

Dysmetria.—When the patient is asked to extend the arm to pick up some object, such as a glass, the limb is shot forwards with undue haste and force and may overshoot the mark.

Tremor.—This is not a resting tremor, but an unsteadiness which develops

during movement, and in purposive movements tends to increase in range and severity as the climax of the movement is reached. It is an "intention tremor." Similarly, if the arms be horizontally extended, they may show a tendency to droop, which is corrected by a series of jerks which thus gives the form of a tremor.

Excessive rebound.—This diagnostically valuable sign may be present when more striking components of the cerebellar syndrome are absent. If the arms be horizontally extended by the patient, and the observer smartly strikes them downwards somewhere in the region of the hand, the arm on the normal side is quickly brought to rest in its original position with a minimum of recoil. On the side of the lesion, however, the hand and arm "bounce" freely, and may swing two or three times before being brought to rest.

Gait.—In bilateral lesion the gait has a reeling, tottering character, and in strictly unilateral lesions there may be a tendency to sway and deviate to the side of the lesion. The disorder may vary in severity from a slight unsteadiness to a complete inability to stand or walk unassisted. There is a tendency to walk with the legs abnormally separated, to raise the legs unduly, and to stamp them down heavily (manifestations of dysmetria).

Numerous other defects may be elicited by special tests, but one only calls for special mention, that is the defective movement of the articulatory musculature in speech. The defect is known as "scanning" or *staccato* speech. It consists in a slowness of articulation, and a tendency to say each syllable of a word as though it were a separate word. In polysyllabic words the syllables are not normally run together.

The cerebellum forms part of the non-sensory afferent nervous system. It is not a sensory organ and there is no disturbance of any form of sensibility in cerebellar lesions (Holmes).

THE ANTERIOR FOSSA OF THE SKULL

The lesion commonly found in this region is meningioma arising from the olfactory groove. The signs are unilateral anosmia from pressure upon the olfactory bulb and tract; primary optic atrophy and visual loss on the side on which the tumour develops; and crossed papilloedema as an expression of the general rise of intracranial tension. Aneurysm of the anterior cerebral artery may give rise to a similar syndrome, papilloedema in the crossed eye being absent.

THE MIDDLE FOSSA OF THE SKULL

A rich variety of lesions may arise in or invade this fossa, and the syndromes also vary according to the situation, mesial or lateral, of the lesion.

The lesions in the midline include pituitary adenomata, tumours of the pituitary stalk, and meningioma of the sellar diaphragm. In the lateral parts of the fossa, passing from the mesial to the lateral extremity, we have to consider cerebral aneurysm, meningioma arising from the sphenoidal ridge, and growths invading the base of the skull and either occluding its foramina and thus producing cranial nerve palsies, or actually invading the

cranial cavity. Epithelioma of the naso-pharynx is the common lesion of the last-named type.

(i) *Region of the optic chiasma and the pituitary body.*—The most common lesion in this region is pituitary tumour, which involves the optic chiasma, at first in the middle line posteriorly, and subsequently advancing forwards. Three sets of symptoms are likely to arise: (1) Those due to dyspituitarism, such as acromegaly or gigantism if there is hyperpituitarism, or Fröhlich's dystrophia adiposo-genitalis, or Lorain infantilism if there is hypopituitarism. Adenomata of the pituitary body produce hyperpituitarism if they contain eosinophil cells, and hypopituitarism if such cells are absent. (2) Those due to the pressure upon the optic chiasma, which commence as bitemporal paracentral scotomata, which enlarge as the compression extends until a complete bitemporal hemianopia results. It cannot be too strongly impressed upon the reader that the pattern of the visual field defect is determined by the position of local pressure upon the visual paths, and that any variety of defective field may occur. While bitemporal loss is the most usual, yet when the pressure is far forward, unocular hemianopia, blindness of one eye, and central scotoma are all of common occurrence, and when the pressure is farther back than usual homonymous hemianopia is frequently seen. And (3) those due to the general effect of the tumour, namely, headache and vomiting. Optic atrophy is the rule, as the result of the direct pressure, and not papilloedema. It is to be remembered, that all pituitary cases are prone to headaches and subject to fits. We have seen also (p. 1593) that cerebral aneurysm may be productive of this syndrome, or at least of the chiasmal component thereof.

(ii) *Lateral region of the middle fossa; syndromes of the sphenoidal ridge.*—The dural sinus which runs along the sphenoidal ridge (sinus sphenoparietalis) is one of the sites of election of the development of meningioma. From the point of view of localising diagnosis this ridge may be divided into three parts, namely, outer, middle and inner (or clinoidal).

A meningioma arising from the *outer end* of the ridge may produce as its localising syndrome unilateral exophthalmos without squint, some fullness of the temporal fossa with local tenderness on pressure, together with the general signs of raised intracranial tension.

Meningioma of the *middle part of the ridge* may remain for long without clear localising indications, and radiography (including ventriculography) may be necessary. Meningioma of the *inner or clinoidal part of the ridge* commonly gives rise to a striking and characteristic syndrome which in addition to the general signs of raised intracranial tension, unilateral failure of vision due to primary optic atrophy, unilateral exophthalmos, crossed papilloedema, more or less complete ophthalmoplegia on the side of the lesion and finally symptoms referable to pressure on the temporo-sphenoidal lobe (uncinate fits, hemiparesis, personality changes). It will be remembered that internal carotid aneurysms also arise in this neighbourhood and may produce a similar symptom-complex, papilloedema, however, being commonly absent. The syndrome of naso-pharyngeal tumour when the skull is invaded also closely resembles this. In other words, this syndrome, in partial or complete form, should arouse in the observer's mind the possibility of one or other of these three varieties of lesion—meningioma of the inner end of the ridge, cerebral aneurysm, and naso-pharyngeal tumour invading the skull.

THE POSTERIOR FOSSA OF THE SKULL: SYNDROME OF THE LATERAL RECESS

Lateral recess.—The angle formed by the posterior surface of the petrous bone and the tentorium is a common situation for neurofibromata which grow usually from the eighth nerve, but occasionally from the seventh and from the fifth nerve, and press into the lateral lobe of the cerebellum. A highly characteristic clinical picture results, of slowly oncoming nerve deafness, unilateral signs of cerebellar involvement and some peripheral facial spasm, to which are sometimes added facial weakness and tinnitus. Such tumours are not of great size, and therefore headache and papillœdema are often absent or occur late.

INTRACRANIAL TUMOURS

Under this heading are grouped all new formations which encroach upon the intracranial space, and which produce the familiar pressure symptoms and local symptoms of tumour, though some of these are not, strictly speaking, neoplasms.

Ætiology.—The brain is one of the commonest seats of new growth in the body. Further, new growth is one of the commonest forms of structural disease of the brain—vascular lesions naturally not being included under this heading. Thus, out of a total of 1309 patients admitted to the National Hospital in 1928, there were 163 cases of intracranial tumour, 132 of disseminated sclerosis, and 113 of neurosyphilis.

Age.—Cerebral tumour may occur at any age, but it is relatively uncommon in the very young and in the very old. It seems to be somewhat more common in the female sex. The relation between head injury and the first appearance of symptoms of cerebral tumour, is one which occurs much too often to be ignored, though it is likely, in some of the cases in which this relation exists, that the blow on the head has simply served to bring a pre-existing tumour into symptomatic prominence, either by causing œdema or hæmorrhage in its substance, or vicinity. It must be remembered in this connection that a cerebral tumour may exist for long periods without definite symptoms.

Pathology.—The pathological classification of intracranial tumours has a practical importance, for when the nature of a new growth can be determined clinically, some idea of its future behaviour can be formed, and the surgeon can make his plans to meet the special problems which each variety of tumour presents.

The chief varieties of intracranial tumour are as follows:

Tumour of the brain substance—Glioma.

Tumour arising in the meninges— $\left\{ \begin{array}{l} \text{Meningioma.} \\ \text{Auditory nerve tumour.} \end{array} \right.$

Tumour of the pituitary body and stalk.

Secondary carcinoma.

Infective granuloma—Tuberculoma, Syphiloma.

Blood vessel tumours.

Parasitic cysts.

It is not possible to indicate the relative incidence of all these different types of tumour, but it is possible to state that glioma constitutes 40 per cent. of all intracranial tumours, and meningiomas and pituitary tumours together from 20 to 30 per cent. Formerly, the incidence of secondary carcinoma was said to be about 6 per cent., but a recent estimate by Elkington places the figure at 20 per cent., and there can be no doubt that as greater precision in diagnosis is reached the frequency of this complication of visceral carcinoma will be more fully recognized.

As its name implies the *glioma* is a tumour arising in the glial or supporting tissue of the brain, but within the limits of this term are included growths of varied cytological type and modes of growth. Some are richly cellular, highly vascular, rapidly growing and fairly circumscribed tumours. To these the name *glioblastoma* is given. Others are diffuse infiltrating tumours, invisible to the naked eye except where degenerative processes have occurred in them, very extensive and often bilateral. To this type the name of *astrocytoma* is given. They are prone to cyst formation and grow more slowly than the glioblastoma. In childhood a variety of glioma known as the *medulloblastoma* is common. It is a richly cellular, highly malignant growth of the fourth ventricle.

Very many other types of glioma have been described, but these classifications are ephemeral and largely artificial, for each type may be represented within a single tumour, and changes from one type to another—from astrocytoma to glioblastoma—may occur in a given growth. Scherer has shown that none of these types is capable of extirpation, the astrocytoma because it is diffuse and largely invisible, the glioblastoma because death follows attempt to remove it. Thus, there is no such thing as a benign glioma. The best that can be said is that the astrocytoma recurs in some cases relatively slowly. Recurrence is invariable in all who survive partial extirpations. The glioma is not always a single tumour, contrary to what has been believed in the past.

Meningioma and auditory nerve tumours.—These tumours of the meningeal sheaths which insulate the ectodermal nervous system occur next in order of frequency to the gliomas. The meningioma, or endothelioma as it is sometimes called, grows from the endothelial cells of the arachnoid villi where these penetrate the walls of the dural venous sinuses. It is therefore found in the neighbourhood of the various sinuses, especially the superior longitudinal, the spheno-parietal and the petrosal sinuses. The meningioma does not invade the brain, but compresses and displaces it, and may become imbedded in it. It may also develop outwards and invade the skull, appearing externally as a rounded boss on the top of the head. The auditory nerve tumour may be single, or may appear as part of a generalised neurofibromatosis, in which case it may be bilateral on the eighth nerve. It grows in the lateral recess, where it compresses and stretches the fifth, seventh and eighth nerves, and also compresses and displaces the cerebellum and gives rise to internal hydrocephalus.

Secondary carcinoma is probably more common than is generally realised. It is a frequent event in pulmonary cancer, and the presence of a secondary involvement of the brain may first bring the patient to notice. Indeed, in all adult cases presenting the signs and symptoms of intracranial tumour the possibility of carcinomatous metastasis should be explored, especially

in a patient who is losing weight. Secondary carcinoma gives rise to numerous deposits in the brain, of various sizes, and may also produce a fine infiltration of the pia-arachnoid, with or without macroscopic masses in the brain. When this "meningitis carcinomatosa" occurs alone its diagnosis may be difficult. Secondary growths may follow primary carcinoma of the breast, stomach, uterus, or prostate, as well as of the lung.

Cholesteatomata.—Sometimes called "mother of pearl" tumours, on account of their glistening appearance, are found in connection with the basal meninges. Their origin is uncertain. They are either of slow growth, or run a symptomless course. They consist of a greasy, greyish, friable and more or less laminated mass, made up of layers of a closely packed mosaic of flat polygonal cells. The tissue is necrotic, and contains no blood.

Among the rarer tumours of the brain may be mentioned dermoid tumours, teratomata, chordomata, which arise from rests of the anterior end of the primitive notochord and are found below the base of the brain, lipomata, fibromata, neuromata, neuroblastomata, consisting actually of undifferentiated nerve cells, enchondromata, angiomas and psammomata.

Cysts.—Cysts of the following nature may occur—(1) Congenital interpeduncular or pituitary cysts, which arise from a pharyngeal rest in connection with the development of the pituitary gland; the resulting signs are those of pituitary insufficiency, together with those of pressure upon the optic chiasma. (2) Simple serous cysts, which are presumably the remains of soft tumours, which have become completely degenerated. (3) Tumours containing cysts, presumably on the way to the formation of the above. (4) Blood cysts, the rare results of hæmorrhage which has become arrested. (5) Cysts which result from softening after embolism and thrombosis.. When occurring in the young, these cysts may lose every trace of their original origin, and form thin-walled cavities, containing colourless fluid, often extending from the ependyma to the pia mater, and involving the whole thickness of the pallium. They are termed "porencephaly." (6) Cystic distension of the ventricles from obstruction, which forms local or general hydrocephaly. These are met with in connection with tumours in any situation and result from adhesive meningitis, particularly syphilitic meningitis. (7) Dermoid cysts. (8) Parasitic cysts, of which the more common is the bladder worm of the tapeworm, *Tænia solium*, which is called, on account of the thickness of its wall, *cysticercus cellulosa*. They are usually multiple, and choose the region of the fourth ventricle as their site of predilection. They may be multiple in the basal meninges, and constitute a "cysticercus meningitis." It is usual for these cysts to shrink and to become calcified and obsolete in from 3 to 6 years. Less commonly, the hydatid of *Tænia echinococcus* is found. It is usually single, may reach a large size and present the signs of a slowly growing tumour with eosinophilia.

Infectious granulomata.—Tuberculomata are more common in the young; but they may occur at any age. They vary in size from that of a millet seed to that of a hen's egg, and are more often found in the posterior fossa of the skull than above the tentorium. When large, coagulation necrosis and caseation occur in the centre, and on section the tumour presents a dry yellowish crumbling or even diffuent centre, with a greyish-red peripheral

growing zone, where are located living tubercle bacilli and actively growing tubercles. The tuberculoma has an important aspect in connection with operation for extirpation. They are often situated favourably for extirpation, yet in every case where this operation has been performed the patient has succumbed to tuberculous meningitis, often after recovery from an apparently completely successful operation. Such a tumour recognised on decompression should be left severely alone.

Syphiloma.—This is not a common intracranial tumour. It grows most commonly from the meninges, and is therefore a surface lesion, though it may burrow deeply in the brain tissue. It is most commonly found above the tentorium. It is occasionally very hard in consistency, and tends in many cases to scar and become obsolete. It is sometimes impossible to distinguish this tumour from a tuberculoma without the aid of the microscope and the serum reaction.

Actinomycomata and tumours from streptothrix infection occur in very rare cases.

Symptoms.—The rates of growth of the different kinds of tumour vary widely. Some cases run their course from onset of symptoms to fatal termination within a few weeks, while in others there is evidence of gradual growth over a period of years. In the latter group it may be only in the final stage that the true nature of the illness becomes apparent, and only in retrospect that earlier symptoms assume their real significance. This perhaps is especially so in the case of those tumours which for months or years have manifested their presence only by generalised epileptiform fits. In yet other cases, an intracranial tumour may remain latent during life, being revealed unexpectedly at post-mortem examination.

Between these two extremes a great variety of symptom-complexes may be presented by an intracranial tumour. Thus, it may first show itself by producing signs of raised intracranial tension alone—that is, by general signs, or by signs of a gradually progressive local lesion alone—that is, by focal signs. Whichever of these two elements is initially lacking will probably appear later. A third manner in which a tumour may first signal its existence is—as has been mentioned—by the occurrence of generalised epileptiform fits in the absence of any other symptoms and signs. In this instance, also, general and focal signs will probably ultimately make their appearance. Again, a sudden onset of symptoms from hæmorrhage into a glioma, or from œdema of surrounding brain, may usher in the clinical course of a tumour within the skull.

The age of the patient is not without influence in determining the symptomatology and clinical course of a tumour. Thus, in childhood the early appearance of greatly raised intracranial tension—that is, of general symptoms, is the rule. This is mainly due to the fact that at this age the tumour is commonly in the fourth ventricle, and is thus favourably placed to produce internal hydrocephalus. In elderly persons, on the other hand, the picture of tumour is apt to be blurred, general signs are late in development, and focal signs are indistinct. Possibly the presence of a background of cerebral arterial degeneration and its associated cerebral change are responsible for this blurring of clinical outline. It may be supposed that the tumour does not write its mark upon a clean slate where there is arterial and cerebral degeneration already present.

GENERAL MANIFESTATIONS.—These symptoms are the result of raising of the intracranial pressure, and accordingly fail when there is no considerable raising. Therefore they tend to be absent in all tumours of the brain stem, in infiltrating tumours of the centrum ovale, and also in advanced age and in the subjects of marked arterial disease. They consist in the following signs: Papilloedema, headache, vomiting, loss of vivacity and mental drowsiness, nasal irritation, giddiness, alteration of pulse-rate, of blood pressure, respiration, and general convulsion.

Papilloedema.—This is by far the most constantly present of all the general manifestations. Papilloedema appears to be a stasis œdema of the nerve-head owing to the increased intracranial pressure forcing the cerebro-spinal fluid into the meningeal sheath which invests the optic nerve, and into the perivascular spaces which accompany the central vessels of the nerve. The nerve sheath becomes distended, and venous stasis occurs. On ophthalmoscopic examination the earliest changes are increased redness of the disk, distension of the veins, loss of distinctness of the nasal margin of the disk, with disappearance of the physiological pit. As the process increases the whole margin of the disk becomes lost. It enlarges in area, and becomes visibly swollen and presents the appearance of a mole-hill as seen from above. The point of emergence of the vessels, at the centre of the disk, becomes buried by white exudation, which occurs also all over the disk, and taking a form determined by the radiating nerve fibrils, gives the disk the appearance of being striated in a radial fashion, like a chrysanthemum. A similar exudate may rupture the membrana limitans interna in little droplets at the macula, and coagulating as it comes in contact with the vitreous humour, produce the characteristic radially arranged macular figure of "macular fan," exactly similar to that seen in renal disease. The venous congestion of the retina leads to multiple hæmorrhages, which infiltrate along the radially arranged nerve fibres, and for this reason are flame-shaped. With the outpouring of much exudation, the disk becomes white. In the course of time the hæmorrhages become white flame-shaped scars, the whole disk contracts, the swelling disappears, and the disk becomes white, flat and atrophic, and distinguished only from that of primary optic atrophy by the scarred remains of the exudate at its edge, producing a fluffy outline like that of torn cotton-wool, along the vessels and at the centre. In the early stages of papilloedema, even though there be considerable swelling of the disk, vision may be little impaired. As the process increases however, in proportion to the degree of the swelling to the amount of the exudate, and to the length of time the papilloedema has lasted in a severe condition, vision becomes impaired, and blindness results. Peripheral constriction of the visual fields, large pupil and dimness of vision, are the signs that, if the papilloedema be not speedily relieved, blindness will certainly result. Perfect vision may be retained for a time, even with a high degree of papilloedema. So important is papilloedema in the diagnosis of tumour of the brain, that it is necessary to bear constantly in mind all other causes which may give rise to it.

Papilloedema may occur in certain general intracranial conditions other than tumour. In meningitis it occurs as a late sign, and rarely before the tenth day, and as so many cases of meningitis do not survive so long, it is chiefly met with in the more chronic forms, such as tuberculous meningitis,

and untreated cases of meningococcal meningitis. Abscess may also cause papilloedema ; but it is by no means common in this condition.

Apart from intracranial disease papilloedema occurs in the following conditions : (1) Local conditions of the retina and optic nerves. In connection with tuberculoma of the retina in the neighbourhood of the disk, the most intense papilloedema may be found. Retrobulbar neuritis occurring close behind the disk may cause a similar condition, especially if it be of a syphilitic nature. In these conditions the papilloedema is often unilateral, but in any of them it may be bilateral. (2) Renal disease may give a retinal picture of intense papilloedema, macular figure and hæmorrhages, sometimes quite indistinguishable from that due to tumour. This is often seen in the small white kidney of young subjects, and sometimes in small red kidney, but there is no form of renal disease, even including tuberculous, amyloid and lardaceous kidney, in which papilloedema has not been observed. (3) Anæmic states of every kind sometimes give rise to papilloedema. As regards groups (2) and (3), it is essential to emphasise the facts that papilloedema, headache and vomiting may occur as a symptom-complex, both in renal disease and in anæmic states. (4) Septicæmic conditions, and especially those producing arthritis. Of these infective endocarditis is the most common ; but it has occurred with every form of septicæmic arthritis, and even in cases of acute rheumatism. (5) Further, papilloedema has been noted in connection with tumours, and with compressions and fracture-dislocations of the cervical cord, and also with acute myelitis.

The retinal changes in diabetes are always, and those in renal disease often, distinguishable from papilloedema resulting from increased intracranial pressure. In diabetes the change is essentially a hæmorrhagic retinitis from degeneration of vessels, sometimes with waxy-looking exudation in circinate patches ; and in renal disease it is often a general œdema of papilla and retina, with hæmorrhages and white patches far away from the disk. The papilloedema resulting from increased intracranial pressure is always bilateral, though it may appear in one eye before the other, unless there be local pressure upon one optic nerve, which always delays or prevents papilloedema appearing in that eye. Otherwise, an earlier commencement upon one side is of no localising value whatever.

Headache.—Although this symptom is a characteristic concomitant of raised intracranial pressure, it cannot be directly attributed to this, since a lumbar puncture which lowers this pressure may lead to increase of headache. It is probable that the pain arises from stimulation of sensory nerves in the walls of the cerebral arteries by changes in tension. The dura mater is probably not the sensitive structure responsible for headache in cases of intracranial tumour. After destruction of the fifth nerve by Gasserectomy, headache never again occurs upon that side. The sensation may vary from a mere feeling of fullness of the head to the most agonising pain. It is more often remittent than continuous, and may be absent for long periods together. It is rarely localised to any definite region, except when the growth actually involves the bone, or when pressure has caused local thinning of the bone, when local pain and tenderness on pressure may occur. Usually it is referred indefinitely to the frontal or to the occipital or to the vertical region. When occipital it may be associated with pain and stiffness of the neck, and head retraction. This is due to a general pressure effect, and does not indicate any

localisation. Headache may be entirely absent, even in the presence of severe papilloedema. It may precede the development of papilloedema, but more often it is later in its appearance.

Vomiting.—Only two-thirds of all cases of intracranial tumour present vomiting as a symptom. It rarely occurs in the absence of the two chief signs of increased intracranial pressure, papilloedema and headache. When the headaches are severe, it may be associated with much nausea, and the attacks are often referred to by the patient as "bilious attacks." Usually a result of increased pressure, it may be directly produced by lesions of the cerebellum, irritation of the vestibular nerve, and by the visual disorientation resulting from diplopia. As a symptom of intracranial tumour it hardly deserves the cardinal importance which has been assigned to it in most descriptions of this disease.

Loss of vivacity and mental drowsiness.—Even when intellectual capacity shows not the slightest impairment, there is from the first onset of symptoms a loss of vivacity, a slight heaviness and an absence of restlessness which is of value in diagnosis. It is almost unheard of for a tumour patient to suffer from insomnia. As the symptoms increase, so do heaviness and drowsiness, though a perfect but slow cerebration may persist until the latest stages of the disease.

Giddiness.—Though this is an inconstant sign, it is often met with, and it may be due to vestibular irritation, when it amounts to an actual vertigo, or it may be a sense of general unsteadiness. It is met with most often in sub-tentorial tumours, but may be quite a general symptom when very high pressure exists.

Convulsions.—As has been mentioned, generalised epileptiform fits, indistinguishable from those of idiopathic epilepsy, may usher in the clinical course of tumour, and may be present for long periods as the sole indication of tumour. The onset of such fits in an apparently healthy middle-aged individual, never before subject to them, should always give rise to the suspicion of tumour of the brain. When later on in the course of the illness general and focal signs of tumour appear, the convulsions may not increase in frequency, and it is not possible to regard them as a sign of raised intracranial tension. They more probably arise from local circulatory disturbances. Beyond saying that they are most frequently found in tumours of the cerebral hemispheres, no localising value can be attributed to them.

Blood-pressure, pulse-rate and respiration.—There is a slight compensatory increase of the blood-pressure for every raising of the intracranial pressure, so that the cerebral circulation may be kept going. The failure of such compensation is often the cause of the sudden death which occurs in tumour cases. The pulse-rate is in the inverse ratio of the blood pressure, and, therefore, of the intracranial pressure, and the pulse is slower than normal, where pressure is above normal. Respiration tends to be slow, and when the physiological condition of the intracranial contents is much disturbed, it tends to become irregular, grouped, and may show the wax and wane of movements which bears the name of Cheyne-Stokes respiration.

FOCAL SIGNS.—These have been fully described in the section upon the localisation of lesions of the brain. In connection with localisation, however, it is important to recognise certain possible sources of fallacy in making a diagnosis. Blindness from papilloedema prevents any localisation by means

of the visual functions. Papilloedema usually causes at one stage great peripheral constriction of the visual fields which might be attributed to a bilateral lesion of the cuneus, and it may cause altitudinal hemianopia, *i.e.* blindness of the upper half of both fields, by sagging of the exudation into the lower part of the retina. Jacksonian epilepsy may occur in long-standing cases without any relation to the position of the tumour.

Paralyses of cranial nerves may be serious pitfalls. They are of value in localisation when occurring early, and in association with alternate hemiplegias, and paralyses of the eighth, ninth, tenth, eleventh and twelfth are always of sure localising value. Paralysis of the sixth cranial nerve, perhaps, should always be disregarded as a localising sign for the following reasons: When the intracranial pressure increases from the presence of a growth, the first effect is that any superfluous cerebro-spinal fluid, of which there is normally very little, is expelled from the skull. Later, with further increasing pressure, since the only escape from the rigid skull is by its only opening, the foramen magnum, the medulla and cerebellum are pushed backwards towards the foramen magnum and come to fill up this aperture as with a cork. In all long-standing cases of increased pressure, the cerebellum will be found on autopsy, and especially when hardened *in situ*, to be deeply marked by the edge of the foramen, part of the cerebellum and medulla actually occupying the spinal canal. Corking up of the foramen magnum in this way offers a marked impediment to the flow of cerebro-spinal fluid, and is a most important factor in the production of hydrocephalus, secondary to tumour. Bearing this in mind the immediately fatal effects which have followed lumbar puncture in long standing cases of high intracranial pressure will be at once understood and for ever avoided. This shifting backwards of the medulla and cerebellum will cause stretching of those cranial nerves attached to the medulla, in proportion as they are directed antero-posteriorly, and take a straight course between their attachments to the dura mater and their origin from the medulla, and of these the sixth nerves will be most affected, and afterwards the third, seventh and fifth in that order. These nerves will not only be stretched, but are subject to the increased pressure also, and they may accordingly cease function simply as the result of the increased pressure. Special mention should be made of tumours of the pituitary body and stalk. In the previous section the localising signs of lesions in the region of the optic chiasma have been enumerated, but since the different varieties of tumour in this locality have their own typical symptom-complexes, the following table may be useful in differentiating them.

Diagnosis.—The differential diagnosis of intracranial tumour has to be made—(1) from other conditions causing papilloedema, (2) from other conditions causing headache, and (3) from other local lesions causing local signs within the brain. Renal disease, conditions of severe anæmia, encephalitis and meningitis may on occasion give rise to a combination of all three of these, very easily confused with the papilloedema, headache and vomiting of cerebral tumour.

Hydrocephalus is only distinguishable from intracranial tumour by the enlargement of the head which takes place in young subjects, but when the skull is rigidly closed, the symptoms are identical with those of a non-localisable tumour.

DIFFERENTIAL DIAGNOSIS OF TUMOURS IN THE PITUITARY REGION
(WALSHE)

	ADENOMA.			PITUITARY STALK TUMOUR.	MENINGIOMA.	GLIOMA OF OPTIC CHIASMA. (rare)
	Chromophobe.	Chromophile.	Mixed Cell.			
Age Incidence.	From adolescence onwards.			From 10 years to early adult life.	From 30 years onwards.	Usually in childhood.
Fundus Oculi.	Primary optic atrophy.			Papilloedema in children; usually primary optic atrophy in adults.	Primary optic atrophy.	Primary optic atrophy.
Visual Fields.	Bitemporal hemianopia.			Bitemporal hemianopia.	Bitemporal hemianopia.	Bitemporal hemianopia, proceeding to early blindness.
	(—Occasionally homonymous hemianopia—)					
Pressure Symptoms.	Absent, or late.			Early and severe, except in adults.	Absent, or late.	Absent, or late.
Glandular Symptoms.	Hypopituitarism.	Hypertrophic or Hypopituit.	Mixed.	Hypopituitarism.	Nil.	Nil.
Situation.	Sellar.			Suprasellar.	Suprasellar.	Suprasellar.
Radiological.	General enlargement and deepening of sella.			Shadows above and in sella. Sella shallow, and with uneven floor.	Commonly no change.	Enlargement of sella forwards beneath ant. clinoid processes.

Intracranial abscess is not often confused with tumour when it has an obvious cause in the vicinity of the brain, from bone disease, or an embolic cause at a distance, such as ulceration of the lung. It is an acute disease and rarely develops an increasing papilloedema. Accuracy in the early diagnosis of tumour cases depends upon the pertinacity with which every case of headache, every case of "fits" and indeed every case which shows any nervous symptom whatsoever, is systematically examined for signs of organic disease, and importantly upon that skill and practice with the ophthalmoscope which is so easily acquired with patience and a little determination. The presence of a tumour having been determined, the necessity is to localise it.

Above the tentorium tumours may be difficult or impossible to localise. So far as decompression is concerned the least indication, however slight, should determine the position of decompression. The external surface of the head should be carefully examined, and especially after it has been shaved, for now and then important indications of the position of a tumour may be afforded, for tumours may grow from the bone, or when internal may cause local absorption of the bone, and bulging of the skull. X-Ray examination should not be omitted, though it does not often afford important information. Tapping of the lateral ventricles, with analysis of their content as to protein concentration, and the introduction of air into the ventricles, with subsequent radiogram, and especially the injection of a thorium salt into the internal carotid artery in the neck, with immediate radio-instantogram, which shows the cerebral arteries and points out any region evascularised by local pressure, are all methods of value. Ventriculography is dangerous, and should only be performed when immediate decompression is practicable if found to be necessary.

The determination of the nature of a growth may be difficult or impossible, and length of clinical history may be a fallacious guide, since a slow-growing tumour may be long latent, and sudden in its production of symptoms. It may be recalled, however, that in childhood medulloblastoma is the most frequently occurring of all intracranial tumours, and that it has a very characteristic picture: headache, vomiting, papilloedema, bilateral external rectus palsy and unsteadiness of gait. Again, a well-marked picture of a progressive frontal lobe lesion generally indicates the presence of a glioma, and the same may be said of the symptom-complex of a temporo-sphenoidal lobe lesion. But there can rarely be any certainty as to the pathological nature of a tumour, even when it is a secondary carcinoma, since the primary growth may be latent and unsuspected.

It is important to remember that the finding of a positive Wassermann reaction in the serum of a patient presenting signs of intracranial tumour does not necessarily—or probably—indicate that the growth is a gumma. Both syphilis and intracranial tumour are common diseases, and their occasional association is less rare than cerebral gumma.

Course and Prognosis.—An intracranial tumour usually causes increasing symptoms, which progress with exacerbations and remissions, until papilloedema ends in blindness, and until the pathological intracranial condition becomes incompatible with even vegetative existence. At any time death may occur from vascular lesions, acute cedema or sudden raising of pressure. Tumours occasionally become obsolete—thus a tuberculoma may become scarred and calcified, and a glioma may become calcified or cystic; but this result is too rare for consideration within the grounds of practical perspective. The average duration rarely exceeds a year after the diagnosis has become possible.

Treatment.—The natural termination of a case of intracranial tumour is death, and the ideal of treatment must be the successful removal of the growth. Failing this, and it is frequently impossible, all that can be hoped for is the relief of headache and sickness, and delaying of blindness.

In respect of the radical, surgical treatment of tumours, it will be remembered that probably more than half (if we include glioma and secondary carcinoma) are in the brain substance, and can be extirpated only by

mutilating operations, which may leave in their wake grave physical and mental disabilities. The success of such an extirpation cannot, therefore, be adequately expressed in terms of "survival period"—as it is not infrequently assessed—but rather in terms of the kind of existence which is prolonged. This may be purely vegetative, and distressing to the patient and his relatives alike. We may say, then, that the treatment of the gliomas is, and must of necessity always remain, the forlorn hope of surgery. On the other hand, signal successes have been obtained in the case of the meningioma, the auditory nerve tumour, the pituitary adenoma, and a few cystic astrocytomas (particularly of the cerebellum).

It will be seen, therefore, how important it is to be able with some precision to determine the type of tumour present in any given case. When this is not possible, an exploratory operation is often justified. But it would be a mistake to suppose that surgical intervention is a matter of routine in every case in which intracranial tumour is diagnosed. Each case must be considered on its merits.

Failing the possibility of a successful removal, the palliative operation of decompression may be needed to relieve the symptoms caused by raised intracranial tension. This consists in the free removal of bone, and the incising of the dura mater, over the region of the tumour when this is known, or, failing localisation, in the right subtemporal region. For brain-stem tumours, decompression is not only useless, but also dangerous.

Relief of pressure by dehydration.—There are circumstances in which it may be desirable and necessary to reduce the brain volume and the intracranial pressure; for example, to relieve pressure headache, to avert impending coma or death, to render the patient capable of co-operating in his examination and thus facilitating a localising diagnosis, and finally to make surgical procedures more easy. Weed and M'Kibben have shown that the foregoing may be done by administering hypertonic solutions. In the ordinary case, the rectal injection of from 2 to 3 ounces of magnesium sulphate dissolved in 8 ounces of water may be tried. But for a very rapid effect, intravenous injection of from 50 to 75 c.c. of a 50 per cent. solution of dextrose, or of a 15 per cent. solution of sodium chloride, is effective. Pain and vomiting may be relieved with the various analgesics of the coal-tar series. When intracranial pressure becomes so high as to cause agonising pain, pulselessness and impending death, morphine in full doses will always relieve, and it is not dangerous. Convulsions should be combated with administration of bromides.

HYDROCEPHALUS

Definition.—The term "hydrocephalus" denotes a uniform distension of the ventricular system of the brain by the accumulation of cerebro-spinal fluid within it; and this distension is associated, sooner or later, with an expansion of the cranial bones and enlargement of the skull.

Hydrocephalus was formerly divided into acute and chronic, acute being applied to the condition of tuberculous meningitis. But since any marked degree of ventricular distension is unusual in that affection, and enlargement of the head very rarely occurs, this term has fallen out of use. In the majority of cases in which general atrophy of the cerebral tissues

occurs, fluid accumulates both in the ventricles and in the sub-arachnoid space; but such compensatory enlargement is not to be regarded as, in any sense, of the same nature as true hydrocephalus. Such accumulation of fluid is found in cases of cerebral diplegia and general paralysis of the insane in children, and it also occurs in the brains of old people. It is merely the result of wasting and shrinkage of the brain-tissue, and the accumulation of fluid takes place in order to fill up the space which is vacated within the rigid skull.

The enlargement of the head, which is not uncommonly found in rickets, has no connection with hydrocephalus. It is probably the result of mal-nutrition of cranial bones, which grow irregularly, and, being unduly soft, yield somewhat to the intracranial pressure. In rare cases of moderate degree, ventricular distension has been met with, but the enlargement of the head is never progressive, and the symptoms of hydrocephalus are absent.

According to their clinical aspect, cases of hydrocephalus may be placed in one of three groups—(1) congenital hydrocephalus, in which the enlargement of the head is present at the time of birth; (2) acquired primary hydrocephalus, which may appear at any period of life; and (3) secondary hydrocephalus. Under the name of secondary hydrocephalus may be grouped together all cases in which there is obstruction in the usual path by which the cerebro-spinal fluid leaves the ventricular cavities, or to the venous outflow from the choroid plexuses. But it is by no means clear that such obstruction is the sole or even the important agent in producing the ventricular distension.

Ætiology.—Hereditary influences are of importance in the causation of congenital hydrocephalus. This disease frequently affects several children of the same parents, and it may even appear as a striking family disease, affecting members of several generations of the same stock. Spina bifida, meningocele and hydromyelia are of frequent occurrence in association with this disease, and arrested and irregular development of the brain stem and cerebellum are the rule. Among other bodily deformities not infrequently associated with congenital hydrocephalus, may be mentioned harelip, cleft palate, talipes, rectal and testicular ectopia and imperforate anus. In a few cases definitely syphilitic lesions of the ependyma in the region of the brain stem have been found. The causation of primary hydrocephalus occurring after the time of birth is often obscure. The majority of the cases occur in childhood, yet no period of life seems to be exempt. In children, acute infective diseases, and especially gastro-intestinal infections, may occur as antecedents of hydrocephalus. In adults, syphilis stands in important relation in certain cases, some of which have been examined pathologically.

The causes of secondary hydrocephalus are, first, the sclerosing forms of meningitis, especially posterior basic and epidemic meningitis, very rarely tubercular; secondly, intracranial neoplasms encroaching upon the ventricular system, especially tumours of the brain stem and subtentorial region; thirdly, adhesive phlebitis of the cerebral blood-sinuses.

Pathology.—Hydrocephalus is directly due to an excess of cerebro-spinal fluid present within the ventricular system of the brain. The fluid is normally secreted by the choroid plexuses of the ventricles, which overflow into the subarachnoid space through the foramina of Luschka and Majendie in the roof of the fourth ventricle. It then fills the basal cisterns

and passing forwards between the tentorium cerebelli and the brain-stem flows up over the cerebral hemispheres. It leaves the subarachnoid space through the arachnoid villi, which pierce the walls of the dural venous sinuses and discharge their contents into the venous blood. Some of the fluid passes downwards into the spinal subarachnoid space, but its absorption is probably wholly within the skull by the channels mentioned. Obstruction to the absorption of the cerebrospinal fluid into the blood stream is the essential factor. This obstruction may arise at various points. There may be congenital atresia of the aqueduct of Sylvius which traverses the brain stem and joins the third and fourth ventricles. It may arise from occlusion of the foramina of Luschka and Majendie by adhesions resulting from an old acute lepto-meningitis. It will be apparent from these considerations that congenital hydrocephalus must be due either to atresia of the aqueduct, or to adhesions blocking the exit of the fluid from the ventricular system, these adhesions being the result of some pre-natal inflammatory process. Acquired hydrocephalus may follow an acute lepto-meningitis which has been productive of adhesions at either, or both, of the situations named above. It may also follow some other mode of blocking of the exits from the ventricular system. Such blocking is commonly produced by an intracranial tumour. The distension of the ventricles which necessarily ensues upon defective absorption of cerebro-spinal fluid from any of these causes is naturally maximal in the infant or child in whom the state of the skull vault allows of expansion of the intracranial space and of an enormous degree of stretching of the cerebral walls of the ventricles, especially of the lateral ventricles.

The quantity of fluid which is found in the ventricles after death varies greatly, a usual quantity being from 15 to 20 ounces. In long-standing cases with great cranial enlargement, very large quantities have been met with. The characters of the fluid do not differ greatly from those of normal cerebro-spinal fluid. Its density varies from 1008 to 1010. It is clear, colourless or slightly yellowish, and the reaction is alkaline. It contains a very small quantity of albumin and a comparatively large quantity of alkaline chlorides.

The dilatation of the lateral ventricles is always more extensive than that of the third ventricle, and is usually symmetrical upon the two sides, and it affects the body of the ventricle more than the cornua, so that the central cortex is the most thinned. The foramina of Monro are greatly enlarged and the anterior pillars wasted.

The convolutions are flattened, and the sulci indistinct. The thickness of the cerebral substance is much reduced. In advanced cases, the cerebral hemispheres have the appearance of a thin-walled sac, which collapses entirely when the contained fluid is allowed to escape. In a few cases, the aqueduct has been found closed, as if by antecedent adhesive ependymitis.

Symptoms.—The clinical manifestations of hydrocephalus fall into two groups, which result, respectively, from the effects of the abnormal intracranial pressure, first upon the brain-case, and secondly upon the nervous structures. In the congenital form, the enlargement of the head is the first noticeable feature; and this is true also of some cases of acquired hydrocephalus in young children. In most cases of acquired hydrocephalus, on the other hand, the nervous symptoms are first in evidence—namely, per-

sistent headache, vomiting, mental impairment, convulsions and sometimes papilloedema. The evidence of cranial enlargement may succeed these symptoms, and the older the subject, and consequently the more resistant the cranial walls, the more severe are the nervous symptoms, and the later is the cranial enlargement in appearing. In some cases of congenital hydrocephalus, enlargement of the head takes place during intra-uterine life, and it may be so great as to make delivery impossible without destruction of the head. More frequently, the cranial enlargement, not noted at the time of birth, becomes evident during the first few weeks of life.

Enlargement of the head is the most striking feature of hydrocephalus in children. The increase usually affects all the diameters of the cranial cavity, and is most marked on the vertex and least at the base. Trousseau compared the opening out of the cranial bones, which occurs as the head enlarges, to falling back of the petals of an opening flower. The forehead is large, rounded, and projects forwards; the temporal fossæ are obliterated, and the parietal eminences carried backwards. The vertex is often somewhat flattened, as also may be the occipital region. The direction of the external auditory meatus alters with the increasing size of the head; normally directed obliquely forwards, it comes to look directly inwards, or even obliquely backwards in severe cases. The head is frequently asymmetrical. In young children the sutures may be widely open, and then there is marked bulging along those lines and at the fontanelles. The skull may attain enormous dimensions, and many examples are recorded in which the circumference has been from 60 to 90 cm. The face is characteristically triangular, contrasting markedly with the forehead. Wasting of the facial subcutaneous tissues and retarded development of the maxilla and mandible often render this contrast still more striking. Bulging of the orbital plates of the frontal bone presses down the eyeballs, so that the pupils become more or less covered by the lower lids, and a band of the sclerotic may be visible between the iris and the upper lid. The hydrocephalic child often uses his hands to depress the cheeks, and so draw down the lower lids out of the position in which they impair the line of vision. The hair of the head becomes scanty, the subcutaneous veins of the scalp are often greatly developed and distended, and sometimes a vortex of distended veins radiates from the region of the anterior fontanelle. The general nutrition is poor, and bodily development retarded, in proportion to the severity of the effect of the intracranial pressure upon the nervous system. Auscultation may reveal a cephalic bruit, but this is neither a characteristic nor a constant sign in hydrocephalus, for it is frequently met with in rickety children, and may be present in a normal subject.

The nervous disorders which appear during the course of hydrocephalus are both variable and inconstant, and acute symptoms are of rare occurrence if the disease appears at an age at which the skull is still yielding. On the other hand, if the ventricular distension commences when the growth and ossification of the skull are complete, the nervous symptoms which arise are very severe, and resemble closely the general effects of intracranial growths. In secondary hydrocephalus, the symptoms due to this condition emerge from those of the preceding meningitis or sinus thrombosis, or are blended with those of the coexisting intracranial growth.

In children, the nervous symptoms of hydrocephalus, whether it be congenital or acquired, may be summed up in the following list, the symptoms

being frequent in the order in which they are enumerated; convulsion, mental failure, spastic paralysis of the limbs, headache, optic atrophy, nystagmus, vomiting, papilloedema. There is no constancy in the occurrence of these symptoms. Convulsion may not occur at all, and mental acuity may be unimpaired. Spastic weakness occurs in less than one-half of the cases, whilst optic atrophy is met with still more rarely, and papilloedema is distinctly unusual.

Convulsion.—While it is to be borne in mind that the whole course of hydrocephalus in children may run without the occurrence of convulsion, yet in the majority of cases this symptom is conspicuous. In some of the post-natal cases the symptoms of cerebral disorder are ushered in by convulsion, and it is probable that such convulsions are the immediate expressions of the morbid process, of which the primary hydrocephalus is the final result. The convulsions which recur at intervals throughout the course of the majority of cases of hydrocephalus result from a condition of functional instability of the cerebral cortex, which long-continued increased intracranial pressure brings about. The convulsions are usually general, with loss of consciousness.

All degrees of mental reduction occur, from the least noticeable to complete idiocy. The more severe forms of mental impairment are met with in congenital cases, and especially when cerebral agenesis, porencephaly and teratological defects are associated. The psychical reduction is less prominent the greater the age at which the symptoms commence, and, as a rule, the intelligence is far greater than the severity of symptoms (cranial enlargement, paresis, etc.) might lead one to expect. Cerebration is usually slow and the disposition placid, and periods of somnolence are of common occurrence.

The effect of long-continued ventricular distension in many cases is to cause degeneration of the pyramidal system, and, according to its degree, the latter entails bilateral spastic paralysis with contracture. The first signs of the onset of this event are exaggeration of the deep reflexes, and the change in type of the plantar reflexes from the flexor to the extensor response. The lower extremities are affected earlier and to a greater extent than are the upper, and at one period of the disease a case may present the picture of cerebral paraplegic rigidity comparable with that of Little's disease. The upper extremities are affected later. The paresis of the limbs is almost always symmetrical and equal upon the two sides. Sensibility is generally normal.

Vision is interfered with in a considerable proportion of the cases. The enlargement of the infundibular portion of the third ventricle, by pressure upon the inner borders of the converging optic tracts, may cause bitemporal hemianopia with atrophy of the nasal portions of both optic disks, this condition subsequently progressing to complete blindness and complete optic atrophy. More often the increased intracranial pressure causes atrophy of the optic tracts and secondary atrophy of the optic disks.

In other cases, optic atrophy is the result of papilloedema. In late childhood and in adult life papilloedema is the rule, and optic atrophy seems always to be consecutive to this. Strabismus is commonly present in congenital cases, and it is most frequently convergent. Nystagmus is met with in the subjects of hydrocephalus who are blind from optic atrophy, and it is of

frequent occurrence in long-standing cases in which spastic paresis is well-marked.

Headache is often complained of, and especially during the early days of illness in acquired cases, but this symptom never dominates the clinical picture in children, and is never so severe and persistent as that arising from the presence of an intracranial growth. Cerebral vomiting is of comparatively rare occurrence.

When one considers the profound anatomical alterations which take place in the advanced stages of the disease, the occurrence in some cases of unusual symptoms indicative of interference with the functions of the cerebellum, brain stem and cranial nerves is easily explicable. Unilateral or bilateral ataxy, vertigo, deafness, anosmia and paralysis of cranial nerves, are the most important of such unusual symptoms.

The signs of failure of the nervous system as a whole usher in the fatal result in severe cases. For some days or perhaps weeks before death, hebetude may become profound; spastic paresis gives place to flaccid paralysis with muscular wasting, the deep reflexes disappear, and the sphincter mechanism loses its control and subsequently its tone.

Hydrocephalus which commences in late childhood or in adult life presents an aspect widely different from that just described. At these periods of life, the bones of the skull are firm and resistant, and the sutures resist for a long time before yielding to the increased intracranial pressure. The general symptoms are acute, and the course of the disease is often rapid to a fatal termination. There is usually no enlargement of the head to aid the diagnosis, and the symptoms—headache, vomiting and papilloedema—resemble those of a non-localisable intracranial growth.

The headache is severe and usually paroxysmal, and it may be so intense as to cause sudden death, while, not infrequently, the sufferer loses all control during the paroxysms. Speaking generally, the headache is of much greater severity in adult hydrocephalus than in intracranial growth. Similarly, vomiting is apt to be more severe and persistent than that associated with cerebral growth. In many of the cases, a fatal result occurs before enlargement of the head, and before cerebral degeneration has produced further signs of spastic paresis than an increase of the deep reflexes, foot clonus and the change of the plantar reflexes to the extensor type. General convulsions and attacks of coma are not rare.

Diagnosis.—Where enlargement of the head is manifest the diagnosis of the disease presents no difficulties. The large head of rickets is easily distinguishable from hydrocephalus by its different conformation, by the association of the other signs of rickets, by the absence of nervous symptoms, by its non-progressive nature, and by the results of anti-rachitic treatment. The distinction between the primary and the secondary forms of hydrocephalus in children should present no difficulty, if a correct history of the early symptoms can be obtained. The initial manifestations in the primary form are slight, and cannot be confused with those of meningitis or of sinus thrombosis. Intracranial growths which cause early and marked hydrocephalus are situated in some part of the brain-stem from the third ventricle to the medulla, and growth in such a position must of necessity produce such early pathognomonic localising signs as to leave no excuse for erroneous diagnosis, save imperfect observation.

In adults the absence of cranial enlargement in most of the cases make it impossible to separate the malady with certainty from intracranial growths. It must be borne in mind, however, that headache, vomiting and papilloedema of rapid progress are not necessarily signs of intracranial growth, but may be the symptoms of primary hydrocephalus.

Prognosis.—This depends upon the cause of the hydrocephalus, upon the degree of severity of the symptoms, and upon whether it is progressive or not. In all severe and progressive cases the prognosis is hopeless, and the same is true of hydrocephalus secondary to inoperable neoplasm. In some of the slighter cases, both of the congenital and of the acquired form, the process becomes arrested, and the patient may attain to adult life with the possession of all his faculties. In cases in which the disease becomes stationary, the prognosis as regards mental capacity and the continuance of recurring convulsion has to be considered. If the mental capacity at the time of arrest is fair, it is not likely to deteriorate further, unless epilepsy is established. When mental reduction is marked at the time of arrest, a great degree of improvement cannot be reasonably expected.

Treatment.—While some cases of hydrocephalus cease to progress, and the symptoms disappear permanently under medical treatment, a like result has occurred in cases in which no treatment has been applied.

The importance of syphilis in the ætiology of hydrocephalus suggests the employment of anti-syphilitic treatment.

The results of surgical interference for the relief of pressure and to attempt the re-establishment of a way out for the cerebro-spinal fluid have been, up to the present, so unfavourable, that many writers and authorities consider such measures unjustifiable. It must be borne in mind, however, that in severe and progressive cases one is dealing with a necessarily fatal malady, and a few encouraging results have been published, which appear to justify further investigation. Paracentesis of the ventricle is both useless and dangerous, for when relief follows the operation it is only temporary, and where cerebral tension is very high an immediately fatal result may supervene. Repeated lumbar puncture is advisable in the earlier days; but this is only possible in cases in which the theca is in free communication with the ventricular space.

ENCEPHALITIS

Acute inflammation of the brain occurs under widely different clinical associations. It may occur as a primary disease or as a complication of known infective processes, affecting the system locally, generally, or as an associated event in diseases of the meninges. As a primary condition it is met with in the form of lethargic encephalitis. It is the constant result of trauma to the skull, if this be sufficiently severe. It is found as the result of infection of the brain with pyogenic organisms, either from local sources in the neighbourhood of the brain (septic bone disease), or from pyæmia, and may be then either suppurative (brain abscess) or non-suppurative. Infections by many of the specific fevers may cause it, and especially measles and scarlet fever. Acute encephalitis may occur in rare cases as the sole manifestation of cerebral syphilis. In all forms of meningitis there is some degree of extension of the inflammation into the brain tissue, and this

assumes an important degree in tuberculous meningitis, and sometimes in epidemic meningitis. The symptoms common to all forms of encephalitis are the general symptoms of severe intracranial disease—headache, somnolence, coma, irritability, convulsions, delirium and vomiting; and, in addition, local symptoms of irritation and paralysis, which are determined by the position and extent of the lesions.

1. SUPPURATIVE ENCEPHALITIS

Synonym.—Intracranial abscess.

Ætiology.—Suppuration within the brain substance is never primary, but the result of extension of infection from neighbouring tissues or by the blood stream from foci of infection in distant organs. In rare cases, the focus of original infection is undiscoverable.

The following are the important causal factors :

1. *Trauma.*—In the case of penetrating wounds the missile may be the source of the infection. Lacerated wound with fracture may allow of infection from the surface or from the middle ear, nose or pharynx. In these cases, meningitis often occurs in addition to abscess. Though not traumatic in a strict sense, any local lesion of the brain may become a locus resistentiæ minoris for the settling down of suppurative organisms derived from the blood stream, and in this way abscess has followed upon vascular lesions and the lesions of primary encephalitis.

2. *Extension from infected regions* in the immediate vicinity. The important cause of infection is any form of infective disease in the bones or soft tissues of the skull, calvarium and surrounding regions. Caries of the petrous bone from middle ear disease is the most common cause, while septic conditions of the nasal cavities and their accessory sinuses, or of any of the bones of the skull, suppuration of the scalp, orbital cellulitis and carbuncle of the neck are other causes. The exact manner of advent of the infection into the brain substance may differ in different cases. It may be by a septic thrombosis of a vein communicating between the infected region and the brain, or by extension along lymphatics similarly communicating, or by direct extension, as when the temporal lobe becomes adherent to the tegmen tympani, or it may be trans-meningeal by the direct transference of organisms across the meningeal space, without general meningitis occurring. That this latter mode of infection is a common one is suggested by the facts, that when the primary disease affects the upper surface of the petrous bone, the abscess is in the temporal lobe, and when the posterior aspect of the temporal bone is affected the abscess is in the cerebellum; and, most importantly, in all these cases of abscess from extension, the cerebro-spinal fluid shows the presence of polymorphonuclear leucocytes, thus showing that the meninges have been infected, although no symptoms of meningitis arise.

3. *Pyæmic states.*—Abscess of the brain does not often occur in symptomatic pyæmia. It may occur in infective endocarditis, and then multiple abscesses may be found. Sometimes in this condition multiple small spots of encephalitis, containing many polymorphonuclear cells but not definite abscesses, are met with. Much more commonly, abscess results from a single septic embolus from chronic pulmonary infection, such as bronchiectasis,

empyema and lung abscess. In rare cases metastatic abscess may arise from bone disease, liver abscess and in the course of specific fevers. The micro-organisms responsible for the infection are usually streptococcus, pneumococcus and staphylococcus, and often the infection is mixed. *B. coli* is sometimes found, and in rare cases streptothrix and oidium albicans.

Pathology.—The abscesses which result from local disease of the skull bones and surrounding tissues may be extradural, subdural or encephalic: in the first two cases they are invariably situated in the immediate vicinity of the antecedent seat of infection. The extradural abscess may reach a very considerable size and may burst externally, or into the meninges or into a blood sinus. The subdural abscess is confined in meningeal adhesions between the dura mater and pia mater. The contiguous surface of the brain is generally softened and has often disappeared, the abscess cavity extending deeply into the brain substance. This variety rarely has any capsule on the cerebral side. Encephalic abscess commences generally in the subsulcine white matter of the temporal lobe, and lateral lobe of the cerebellum. In one-half of all cases the abscess is in the temporal lobe, and in one-third in the lateral lobe of the cerebellum. The remainder are divided between the parietal lobe, the pons Varolii and the frontal lobe, in order of diminishing frequency. The size of the abscess varies up to that of a hen's egg. A recently formed abscess is irregular in shape with ill-defined limits, but in about 7 days it shows a definite capsule which may rapidly become of considerable thickness. The interior of the abscess cavity is usually of a greyish-green colour, and the pus is greenish and often fetid. The surrounding brain tissue is always cedematous and often softened. Rupture occurs in about one-sixth of all cases that are not afforded operative interference, and the rupture takes place most commonly into the ventricle and less frequently into the arachnoid space.

Symptoms.—An encephalic abscess has its origin in inflammation, and constitutes, when developed, a foreign body within the skull. Death may result from the effects of continually increasing intracranial pressure and wide interference with cerebral function, or from spread of the infection from the abscess. The symptoms may be grouped in four classes—(1) those of local suppuration; (2) those due to increased intracranial pressure; (3) localising signs dependent upon the position of the abscess; and (4) those of terminal extension of the infective process.

In extradural and subdural cases, the symptoms are generally acute and the course is rapid; the signs of pressure are severe, whilst localising signs are rare and a state of latency is not observed. In the majority of encephalic abscesses, on the contrary, the signs of initial suppuration are slight and are apt to be swamped by the symptoms of the preceding disease, otitis media, empyema, infective endocarditis, etc., and for this reason may be easily overlooked. A latent period in which symptoms are insignificant or completely absent may follow, and last for weeks or months. In the end, the latent period is broken, either by an acute outburst of symptoms, the result of extending infection, or the signs of progressive intracranial tumour arise.

The general symptoms which are likely to appear when a brain abscess is developing or emerging from a latent condition are pyrexia, which may be associated with rigor, headache, vomiting, irritability, vertigo, drowsiness deepening into coma, slowing of the pulse, respiratory and cardiac irregu-

larity, convulsions rarely, and papilloedema as a late sign. In addition, there is a leucocytosis of the polymorphonuclear variety in the blood, and a small number of polymorphonuclear leucocytes in the cerebro-spinal fluid in those cases arising by extension from disease of the cranial bones, but not in metastatic abscesses. The general symptoms vary much in their intensity and in the individual incidence of each of them, and in metastatic abscesses they may be almost absent, the local signs alone giving the indication that a cerebral lesion is present. Headache is rarely absent, and may be intense with spreading abscess. Vomiting is also an almost constant sign. Drowsiness is one of the most valuable of all the indications when any cause for the occurrence of cerebral abscess is present. Slowing of the pulse is also an important indication of a rising intracranial pressure. Papilloedema occurs late, and is often not present in acute abscesses at the time when diagnosis is all-important from a surgical point of view. It rarely occurs until an abscess has been present for a week, and is generally of low grade. With half-latent chronic abscesses, and with metastatic abscesses which attain a large size, it may be intense.

Local signs.—Generally speaking, the more recent and acute the abscess is the less definite are the local signs. In more chronic abscess, and in metastatic abscess, the local signs are usually more distinct. When there is a local cause for the abscess this constitutes an important localising sign, since abscess forms almost always in the immediate vicinity of site of infection. Thus rhinogenic abscesses are situated in the frontal region, and otitic abscesses are almost invariably situated either in the temporal lobe or in the lateral lobe of the cerebellum of the same side as the ear disease.

Metastatic brain abscesses may be situated anywhere, but they are more common in the region of distribution of the Sylvian artery, and in my experience have been more common in the posterior part of this supply—that is, in the parietal and occipital lobes. Metastatic abscess is sometimes preceded by definite indications of the embolism which gives rise to the abscess, such as local convulsion, local transient weakness or loss of consciousness, and such an event may precede the signs of abscess by many weeks. The local signs of lesion in the various regions of the brain are described in the section under that heading. Local diagnosis is often difficult owing to the condition of somnolence preventing the possibility of accurate examination. In these circumstances such slight signs as the absence of the abdominal reflex on one side, the presence of an extensor response in the plantar reflex of one side, or any aphasic signs, are important indications of temporal lesions; and unilateral hypotonia and nystagmus and attitudes, of cerebellar lesions. The initial signs and symptoms of encephalic abscess may lessen and disappear, and the abscess is said to become latent. The latency may be complete, or it may be broken by occasional headaches and transitory symptoms indicative of intracranial mischief. Much more commonly the abscess grows, and death occurs invariably in the absence of surgical interference, either from increasing intracranial pressure or from the rupture of the abscess, either into the ventricle, or on to the surface, with the production of general meningitis.

Diagnosis.—In those cases where there is no local cause for the formation of an intracranial abscess by direct extension, and no distant cause known or discoverable for the formation of a metastatic abscess, diagnosis is difficult,

and the distinction of an abscess from a tumour can hardly be made with certainty. The presence of pyrexia and of a polymorphonuclear leucocytosis in the blood may suggest the diagnosis in some cases. Where, however, the common antecedent causes of abscess exist in the form of ear disease, etc., or suppurative chronic lung disease, the diagnosis is relatively simple. For example, the advent of local or general intracranial signs in a case of chronic bronchiectasis from the first leaves no alternative diagnosis. When ear disease or local septic conditions of the region of the skull are present, local and general intracranial signs are due either to meningitis, abscess, sinus thrombosis, osteomyelitis of the base of the skull, or rarely to acute otitis.

Meningitis can be at once distinguished not only on account of the more irritative and rapidly oncoming symptoms, which differ somewhat from those of abscess, such as head retraction and rigidity of the neck, Kernig's sign, delirium and tremors, but by the lumbar puncture which gives the turbid cerebro-spinal fluid, containing polymorphonuclear cells and organisms in quantity. It must not be lost sight of that an abscess at any stage of its formation may be complicated by the development of general suppurative meningitis. Sinus thrombosis is usually accompanied by much oscillation of temperature, and by repeated rigors and oedema, and tenderness in the region of the emissary veins of the blocked sinus may be present. The diagnosis of the latter condition is not of vital importance, and its consideration should cause no delay in summoning the aid of the surgeon. The presence of any symptoms of intracranial disturbance, where tympanic septic disease exists, calls for immediate surgical interference, and the surgeon, after cleaning out the diseased tympanum, completes the diagnosis by examining the lateral sinus, both the temporal lobes and the lateral cerebellar hemisphere, and proceeds to those measures which the results of his exploration indicate. Acute otitis media may give rise to severe intracranial symptoms like those of meningitis, convulsions even occurring which may subside dramatically after perforation of the tympanic membrane, but it must be remembered that the chronic and not the acute forms of otitis give rise to septic extension to the brain. Osteomyelitis of the base of the skull extends from chronic bone disease in the region of the ear or nose. There is much pain in the base of the skull, and sometimes many cranial nerves are implicated at their foramina of exit. Skiagraphy will indicate the loss of bony structure. The malady is a chronic one, and usually ends in a terminal meningitis.

Prognosis.—Cerebral abscesses with very thick walls and inspissated or even calcified contents have been found post mortem, many years after the presumed time of formation of the abscess. Spontaneous evacuation of an abscess through the diseased ear, or through a sinus in the area of the local disease causing the abscess, has been followed by recovery. It is probable that no abscess becomes permanently quiescent after it has given rise to severe symptoms. The prognosis in cases of cerebral abscess, therefore, is that a fatal result will occur, unless successful surgical interference is possible. If the abscess is reached and drained, recovery often occurs rapidly, but this is never certain, for extensive perifocal softening, meningitis and sinus thrombosis may occur. Moreover, a general suppurative encephalitis may extend, in spite of draining the abscess.

Treatment.—The most rigorous prophylaxis should be employed that all patients suffering from septic nasal and ear diseases, and infective disease of the scalp and cranial bones, shall not pass out of observation until such disease is beyond all doubt cured. The only treatment for developed abscess is exploration and drainage. The liability to the occurrence of septic meningitis may perhaps be lessened by the administration of hexamine. The usual measures for the relief of pain should be employed.

2. LETHARGIC ENCEPHALITIS (see p. 1576)

MENINGITIS

Definition.—The inflammatory processes to which we apply the name of meningitis are infective in origin, and usually have their seat in the leptomeninges—the pia-arachnoid. A true inflammatory lesion of the dura mater, that is, pachymeningitis, is much less common, and is usually a localised process due to the direct spread of infection from adjacent bone.

Acute leptomeningitis, on the other hand, is usually generalised, and even when it arises from a local focus of infection it spreads rapidly throughout the subarachnoid space, this spread being facilitated by the cerebro-spinal fluid and also by the negligible bactericidal potency of this fluid. Further, the inflammation not only produces its characteristic changes in the pia-arachnoid, but also greatly changes the composition of the cerebro-spinal fluid. These changes may be said to reflect with considerable accuracy the nature and cause of the meningitis, and thus it is that the examination of this fluid has so great a diagnostic value. Acute leptomeningitis may be a primary infection, or may be secondary to some infective lesion elsewhere in the body.

Pachymeningitis may be cranial or spinal, and is usually secondary to either syphilis, tuberculous disease of bone, or middle-ear suppuration. The condition formerly known as “pachymeningitis interna hæmorrhagica” is now regarded as traumatic and not inflammatory in origin, and is described under the heading of chronic subdural hæmatoma (cf. p. 1593).

The fine infiltration of the pia-arachnoid by the cells of secondary carcinoma, of glioma, or sarcoma has been spoken of as a meningitis, but although such an infiltration may give rise to symptoms resembling those of a true meningitis, the term is not strictly accurate, though it is well to bear in mind that this form of new growth does occur and give a picture of meningeal irritation.

EXAMINATION OF CEREbro-SPINAL FLUID

The normal cerebro-spinal fluid is clear and colourless. As obtained by lumbar puncture, it is found to be under a pressure of from 60 to 150 mms. of water and to contain from 0 to 5 cells (lymphocytes) per c.cm. Its chemical composition is as follows:

Protein (mainly

albumin)	.	0.01 per cent.	(10 to 20 mgrms. per 100 c.c.)
Glucose	.	0.05 to 0.08	„ (50 to 80 mgrms. per 100 c.c.)
Chlorides	.	0.72 to 0.75	„ (725 to 750 mgrms. per 100 c.c.)

The lumbar puncture is made in the first interspinous space above a line joining the highest points of the iliac crests, which is the space between the third and fourth lumbar spines, or it may be made with equal rectitude in the space between the second and third lumbar spines. The needle should be introduced exactly in the middle line and at right angles to the surface, close to the upper spinous process of the interval used. Normally the fluid escapes drop by drop. If it runs rapidly or spurts out, this is an index of the increase of the cerebro-spinal pressure. Such an increase is met with in all meningeal inflammation, congestion and hæmorrhage, and in increased intracranial pressure. But it is also produced by coughing or straining, and its accurate measurement by a manometer is now part of the routine examination of the fluid.

Queckenstedt's phenomenon.—If, with a manometer in attachment to a lumbar puncture needle, the jugular veins be compressed, an immediate rise in cerebro-spinal fluid pressure in the normal person will be noted, the pressure rising as high as 300 or 400 mms. of fluid. It falls as rapidly when compression is released. If there be a block in the spinal subarachnoid space, or in the exits from the ventricular system, there will be no rise (complete block), or a slight rise with a delayed fall (incomplete block). Again, if the removal of a few c.cms. (4 to 8) of cerebro-spinal fluid is followed by a persistent fall in pressure of about 50 per cent., there is probably a block in the spinal subarachnoid space. These two tests afford valuable information in cases of suspected "obstructive" hydrocephalus, or of spinal block from tumour, or other local disease.

Increase of Protein Content.—This is of high importance, and occurs in all conditions of meningitis, and especially when the thecal space is obstructed by tumour, pressure from without or meningeal adhesions. In pathological conditions the protein content may reach 0.8 per cent. or more. A high protein content sometimes associated with xanthochromia, in the absence of cellular elements, is highly characteristic of thecal obstruction and is known as "Froin's syndrome." Lethargic encephalitis does not give any increase of protein.

Xanthochromia is a yellow colour of the cerebro-spinal fluid, and it is met with when the cerebro-spinal space is obstructed by tumours of the cord or meninges, or by external pressure, and in some forms of polyneuritis and meningitis. The yellow colour may result from an extravasation of blood either into the arachnoid or into the central nervous system.

Spontaneous coagulation of the fluid is met with in some cases of meningitis, when there is spinal obstruction, and in some varieties of acute polyneuritis.

Blood may occur from every condition of hæmorrhage, injury and encephalitis, and sometimes in meningitis. Blood that has been long shed into the cerebro-spinal fluid tends to become brownish and later yellowish. Leucocytosis is indicative of meningitis and often occurs in encephalitis, and in the neurotropic virus infections and in mumps. A lymphocytosis is characteristic of tuberculous and syphilitic meningitis, poliomyelitis, lethargic encephalitis, sinus thrombosis and mumps. A polymorphonuclear cytolysis occurs in meningococcal meningitis and all the suppurative forms of meningitis. In tuberculous meningitis there is often a mixed cytolysis at first, in which the polymorphonuclear cells may form 60 per cent. of the total. In

poliomyelitis, the lymphocytosis disappears after the end of a week. In lethargic encephalitis there is frequently no lymphocytosis.

Pus may be present in quantity in all the septic forms of meningitis, and especially in pneumococcal and epidemic meningitis.

Decrease in Glucose Content.—All conditions of meningitis cause decrease in the glucose.

Alteration of Chloride Content.—Diminution in chlorides is highly characteristic of tuberculous meningitis, and a reduction below 0.65 per cent. is pathognomonic of that condition. Increase in chlorides occurs in uræmia and other conditions of salt retention.

Lange's Colloidal Gold Reaction.—In neurosyphilis and in some cases of disseminated sclerosis, the globulin fraction of the total protein of the cerebro-spinal fluid increases and may almost equal the albumin fraction. The high globulin content gives the fluid a power of precipitating colloids from suspension. The estimation of this power in the case of colloidal gold is the basis of Lange's test. To ten dilutions of cerebro-spinal fluid (from 1 in 10 to 1 in 10,000) constant amounts of colloidal gold are added, and the mixtures allowed to stand for 24 hours. The form of the precipitation curves has a differentiating value. Thus in general paralysis the first six dilutions are precipitated (paretic curve), in tabes dorsalis, the third and fourth dilutions show the maximal precipitation (luetie curve); in meningitis, the sixth to eighth dilutions are precipitated (meningitic curve). In disseminated sclerosis the combination of negative Wassermann reactions in blood and fluid and a paretic curve in the fluid is frequently found.

The nature of the organismal content is determined (1) by the direct examination of films made from the centrifugalised fluid, (2) by cultures from the fluid, and (3) by the inoculation of animals from the fluid.

NAME.	ORGANISM.	CEREBRO-SPINAL FLUID.
Tuberculous meningitis	Tubercle bacillus.	(Clear or turbid. Lymphocytes, either alone or in greater numbers than polymorphs. Tubercle bacilli Chlorides diminished.
Pneumococcal meningitis	Pneumococcus.	Turbid. Polymorpho-nuclear leucocytes. Pneumococci.
Meningococcal meningitis . Sporadic or posterior-basal. Epidemic or " Spotted Fever."	Still's diplococcus. Weichselbaum's diplococcus.	{ Clear or turbid. Polymorpho-nuclear leucocytes. Intracellular diplococci.
Pyogenic meningitis .	{ Staphylococcus. Streptococcus. B. influenza. Gonococcus. Streptothrix.	Turbid. Polymorpho-nuclear leucocytes. Organisms.
Syphilitic meningitis .	<i>Spiracheta pallida.</i>	Clear. Lymphocytes only. Wassermann reaction +.
Other forms .	{ <i>B. typhosus.</i> <i>B. enteritidis.</i> }	Turbid. Polymorphs.
Rheumatic meningitis	<i>Diplococcus rheumaticus.</i>	
Serous meningitis		Clear. Few cells. Sterile.
Traumatic meningitis		

The Wassermann reaction in the fluid is positive in all conditions of recent syphilitic disease impinging upon the meninges, and always in general paralysis. Though often positive in tabes, it may be found negative. Lumbar puncture is dangerous in cases of long-standing increased intracranial pressure, and if performed in such cases a minimum of fluid should be withdrawn. It may in some cases cause severe headache of long duration. It may be difficult or impossible to perform when there is bone disease of the lumbar vertebrae.

The most useful classification of the varieties of meningitis is according to the nature of the micro-organism producing the inflammation.

1. TUBERCULOUS MENINGITIS

This disease results from the general invasion of the cerebro-spinal leptomeninges by the tubercle bacillus, and this organism invariably arrives in the meninges by the blood stream from some previously existing focus of tuberculous infection within the system, and most commonly from caseous tracheo-bronchial glands and tuberculous bone disease. Occurring at all ages, it is the form by far the most frequently met with in the second and third years of life. The characteristic features of the cerebro-spinal fluid are, that it is usually under considerable pressure, it is clear or only slightly turbid, has no visible deposit before centrifugalisation, but it often forms a fine flocculent clot. It contains an excess of albumin. The normal sugar is generally absent. There is a pleocytosis with a high proportion of lymphocytes, 70 to 80 per cent. being of this nature, and the rest being polymorphonuclears. Careful examination will almost always reveal the presence of the tubercle bacillus.

Ætiology.—The inheritance of a lowered resistance to the invasion of the tubercle bacillus is an important factor, especially when such a tendency exists in both parents. The sexes are equally affected. Tuberculous meningitis is rare during the first year of life, and especially during the first 6 months of life, when posterior basal meningitis is most common. Its greatest incidence is during the second and third years. It is common throughout childhood and early adult life, after which it becomes increasingly rare. The primary focus from which the organisms are spread to the meninges is most commonly a tuberculous mesenteric or bronchial gland.

Sometimes the source of infection is tuberculous disease of the lungs, of the abdomen, of the ear, of the joints, or of bone. Operations upon the sites of tuberculous disease may directly cause the dissemination of the tubercle bacilli, and especially surgical procedures upon tuberculous intracranial tumours, upon spinal caries, and upon tuberculous disease of bones and joints. The acute specific fevers, and especially measles, are sometimes the exciting causes of the disease. Injury to the head sometimes determines the attack.

Pathology.—In tuberculous meningitis three kinds of lesions of the meninges may be met with—(1) grey tubercles unassociated with inflammatory deposit; (2) tuberculous meningitis characterised by the presence of tuberculous granulations associated with a fibrinous and purulent exudation—the superficial tissue of the nervous system underlying the meninges is in this case always involved; (3) tuberculous tumours of any size up to that

of a pigeon's egg. It is not uncommon to find such a tumour to be the focus of widely spread meningitis. The three kinds of lesions may coexist in the same case.

The flattening of the convolutions and the dry sticky feel of the surface of the brain are highly characteristic. The disease affects the pia-arachnoid and its processes, the small vessels entering the surface of the brain and the superficial tissues of the latter. Occasionally a few tubercles are found upon the inner surface of the dura mater. Generally the convexity of the brain escapes, or is little affected. In the intercerebral space, around the optic chiasma, covering the tips of the temporal lobes, along the commencement of the Sylvian fissures and around the brain stem, there is an inflammatory exudation of tough consistency and of a pale yellowish-green colour. Spreading from the edge of this in decreasing numbers, grey tubercles are seen in the pia-arachnoid, particularly along the Sylvian fissures. They may be found wherever the pia-arachnoid extends (the convexity, as a rule, excepted), but except at the base of the brain they are not, as a rule, accompanied by the characteristic tough exudation.

The brain as a whole is soft, and local softening of the walls of the ventricles, of the velum interpositum, and of the fornix is often present. This softening is caused by spreading of the tuberculous process from the pia-arachnoid to the small vessels of the surface of the brain, on the walls of which tubercles develop, sometimes in such numbers that a small entering vessel, when observed under low magnification, after the brain tissue has been removed by careful washing, may resemble a bunch of grapes, each grape being a tuberculous nodule. Thrombosis is a common event in the vessels so involved, and softening follows. Some degree of thrombosis in the superior longitudinal sinus and in the veins of Galen is commonly present. It is probably owing to the softening of the nervous tissues that occlusion of the foramina by which the cerebro-spinal fluid leaves the ventricles does not often take place, and that, therefore, a condition of hydrocephalus does not occur in this form of meningitis. The cranial nerve palsies which are so frequently met with in this disease are the result of implication by adhesions and local interference with the blood supply of the nerves at the base of the brain, by the newly formed adhesive tissue. In the majority of cases the membranes of the spinal cord are affected, and the most common situation of the tubercles is upon the inner surface of the theca, and in the pia covering the lumbar enlargement.

Symptoms.—The onset is usually gradual, with signs of vague and slight illness. In children, general apathy and neglect of amusements and play, headache, loss of appetite, dullness, fretfulness, restlessness at night with grinding of the teeth during sleep, headache, vomiting and pyrexia are common symptoms. In older subjects, lassitude, depression, mental alteration, perversity and hysterical¹ manifestations are common. Constipation is usually present, and the breath has a peculiar fetor. The facial expression is one of illness and frowning discomfort, and there is disinclination to talk. Young children may be speechless for days together. As a rule, in this stage of the disease young children complain of nothing, and delirium is rare; but as age advances, delirium increases in frequency, and headache, usually frontal is increasingly complained of. These slight and vague symptoms may last from a few days to several weeks, and constitute what has been called

the prodromal stage of the malady. An early disappearance of the knee and ankle jerks, and the occurrence of retention or urine are often early signs and should be looked for in suspected cases. In those cases which are said to being acutely, careful inquiry will generally reveal that some symptoms such as the above have preceded the acute onset. The further development of the disease is marked by the appearance of a lethargy, which soon deepens into a stupor, from which it is difficult or impossible to arouse the patient. Vomiting is of frequent occurrence, and headache may be severe. The child lies upon its side in the "cramped" position, resenting any disturbance. The expression becomes vacant, with wide-open eyes and dilated pupils, as if fixed upon some distant object. There is often some retraction of the angles of the mouth, and there is frequently a bright malar flush. In the later stages the limbs are generally extended and rigid. Stiffness of the neck is the rule, and head retraction may occur, but this is never so marked as in posterior basal meningitis. The abdomen is always markedly retracted and a *tâche cérébrale* is often conspicuous. A single sharp cry, apparently causeless, called the hydrocephalic cry, and which is common in all forms of meningitis and also in other infantile intracranial affections, is sometimes heard.

Ocular phenomena make their appearance towards the end of the first week of the developed disease. All varieties of varying and persistent strabismus and ptosis are met with, paralysis of the external rectus being the most common. Rolling movements and independent movements of the eyeballs may occur. None of these signs is constantly present. The pupils may be contracted at first, and may show varying inequality, but in the later stages they are dilated. Papillœdema is often present towards the end of the second week, if the patient survives so long. It is of moderate intensity, the height of the swelling rarely exceeding two dioptries. Choroidal tubercles sometimes occur.

Convulsions are common in every stage of the disease in children, but rare in adult cases. They may be the first symptom of the onset, but are more often met with in the later stages of the disease. They may be local or general. Repeated rhythmic movements are frequent, and are specially noticeable in connection with the mouth, where sucking and champing movements and grinding of the teeth are common. Rhythmic movements of the limbs may also occur. Coarse tremor upon movement of the limbs is the rule, and spasmodic twitching of the muscles is frequent. In rare cases, movements exactly like those of chorea occur. Kernig's sign is usually present.

The temperature is usually raised one or two degrees, but it presents no characteristic features, some cases being apyrexial throughout. Irregularity of the pulse is the rule, and is of considerable diagnostic importance. Rapid in the early stages, it tends to become unduly slow in the stage of coma, and again rapid as death nears. Cheyne-Stokes breathing and grouped breathing are common. Constipation is usually a marked and persistent feature.

Course.—The course of tuberculous meningitis is progressive to an invariably fatal termination in from a few days to 3 weeks after the appearance of definite symptoms, and no case of recovery is known to me in which the diagnosis has been unquestionably proved by the recovery of the tubercle bacillus from the cerebro-spinal fluid.

Diagnosis.—The early symptoms of the disease may give rise to difficulty in diagnosis, but the latter is relatively simple when the disease is advanced. The diseases liable to be confused with tuberculous meningitis at its commencement are gastro-intestinal catarrh, the exanthemata—especially enteric fever—and pneumonia. It must be borne in mind that in children convulsion, strabismus, head retraction and stiffness of the neck, with pyrexia, may be symptomatic of many maladies apart from meningitis, especially of apical pneumonia. In enteric fever the temperature is higher and the headache more severe, and irritability and resentment of interference are not present; the decubitus is usually dorsal. Widal's test is of importance in this connection. When distinctive signs of intracranial disease have appeared the diagnosis has to be made from other forms of meningitis, sinus thrombosis, tumour, abscess and middle-ear disease. Careful examination of the retina and of the tympanic membranes is then necessary. In all cases the diagnosis must be made certain by the examination of the cerebro-spinal fluid, which will be found to contain lymphocytes in excess and tubercle bacilli. These organisms are sometimes difficult to isolate from the fluid, but their presence can be readily demonstrated by injecting the fluid into the subcutaneous tissue of guinea-pigs, when the characteristic lesion of tubercle results. It must be remembered that in some cases the polymorphonuclear leucocytes may be in excess, but these cases are at once distinguished from other forms of meningitis by the presence of numerous lymphocytes, by the absence of the meningococcus and of the other germs producing suppuration, and by the presence of the tubercle bacillus. Pirquet's skin reaction is often absent in tuberculous meningitis.

Treatment.—From the unvarying fatal issue of the malady, treatment can only be directed towards the relief of symptoms. Temporary improvement and relief of headache may be brought about by lumbar puncture, which may for this purpose be repeated several times. Bromides, chloral, aspirin and other analgesic drugs may be used to relieve the headache, check the convulsion and diminish the restlessness. Hexamine in large doses, inunction of mercury and administration of tuberculin have been largely used, but without any success. General treatment must be that which will secure such comfort as is possible for the patient. Where swallowing is difficult nasal feeding should be adopted.

2. PNEUMOCOCCAL MENINGITIS

Pneumococcal infection of the meninges most commonly follows upon a similar infection elsewhere in the body, empyema and pneumococcal otitis being the commonest lesions, while pneumonia, abdominal infection, abscess and joint infection are less common. In one-third of the cases, however, the meningeal infection is primary. The characteristics of the cerebro-spinal fluid are that it is purulent and sometimes so thick that it will not flow through the needle. It is greenish-yellow in colour, contains a large amount of albumin, and multitudinous polymorphonuclear cells, among which the characteristic pneumococcus is found. In fulminant rapidly fatal cases the fluid may be turbid from the presence of pneumococci alone, no reaction in the form of pleocytosis being present.

Ætiology.—The disease may occur at any age. It is sometimes a terminal

event of a pneumococcal infection elsewhere, and passes almost unnoticed, or is discovered only at the autopsy. Meningitis which follows operations upon the nose and disease of the nasal bones is usually of the pneumococcal variety.

Pathology.—The surface of the brain and spinal cord is highly characteristic. Usually the whole surface of the vertex and of the base is covered with a thick, tenacious, greenish-yellow pus, which is contained in the meshes of the arachnoid, and between this and the dura. The ventricles often contain pus. A similar exudation is found upon the spinal cord, more especially upon the dorsal aspect, and in the cervical and lumbo-sacral regions. The major affection of the vertex of the brain is the peculiarity of this disease, and only in the rarest cases is the base alone affected. The exudation is characterised by the greater amount of fibrin than in other forms of meningitis.

Symptoms.—The symptoms are those which are common to all forms of meningitis. Some of the cases are indistinguishable symptomatically from cases of tuberculous meningitis. Others run a very rapid course and present few features other than headache, vomiting and pyrexia, with rapidly oncoming and quickly fatal coma. In others again, the meningeal symptoms are concealed in the terminal asthenia of a previously existing pneumococcal infection elsewhere, such as empyema, purulent pericarditis or peritonitis.

Diagnosis.—This rests upon the presence of signs of meningitis or the existence of coma alone, together with a cerebro-spinal fluid which is purulent from the presence of polymorphonuclear leucocytes, containing the pneumococcus.

No case of recovery from this form of meningitis has hitherto been recorded. Lumbar puncture and intrathecal injections of anti-pneumococcal serum may be performed, but on account of the thick nature of the exudate, little relief must be expected from the former, while the latter cannot possibly avail except in primary cases.

3. MENINGOCOCCAL MENINGITIS (see p. 36)

4. PYOGENIC MENINGITIS

Apart from meningococcal and pneumococcal infections, suppurative meningitis may result from the invasion of the meninges by staphylococci, streptococci, gonococci, *B. influenza*, *B. anthracis* and streptothrix.

Staphylococcal and streptococcal infections are by far the most common. They may result in young children from septic conditions of the umbilicus and from infections of the skin. Usually they are due to extension of an infection of adjacent structures to the meninges, and follow disease of cranial and spinal bones, especially caries of the middle ear, erysipelas and other infections of the scalp, wounds of the meninges, especially bullet wounds, rupture of intracranial abscess, and they may occur in the course of a general septicæmia.

Pathology.—The pathology of these conditions does not materially differ from that of pneumococcal meningitis. In all cases the exudation

is purulent, and in the meningitis due to *B. anthracis* it is of a red colour, due to concomitant blood effusion. The cerebro-spinal fluid contains large numbers of polymorphonuclear leucocytes, together with the micro-organism responsible for each variety. Suppurative meningitis resulting from bone disease and from wounds of the meninges may be localised by the formation of meningeal adhesions, and an intrameningeal abscess may result. Such an abscess situated upon the upper surface of the temporal bone is not an uncommon result of caries of the middle ear.

The clinical aspect is that common to all forms of acute meningitis, high pyrexia, rigors and delirium being conspicuous. The course is rapid to an almost invariably fatal termination. In the localised form where drainage can be ensured and extension of the infection prevented, recovery should take place. Several cases of recovery from gonococcal meningitis have been reported. Influenzal meningitis is invariably fatal.

Diagnosis.—This depends upon the presence of the clinical signs of meningitis and of a cerebro-spinal fluid containing polymorphonuclear leucocytes in large quantities, and upon the recognition in this fluid of the several micro-organisms responsible, by microscopic and cultivation methods. The recognition of *B. influenzae* requires that cultures should be made upon some blood medium, for otherwise the organism may be easily overlooked and the fluid reported as sterile. Further, the presence of some well-known cause for suppurative meningitis, such as ear disease, staphylococcal infection, etc., suggests the diagnosis.

Acute otitis media may give rise to symptoms closely resembling those of meningitis, such as headache, pyrexia, vomiting, head retraction and delirium. In such cases examination of the ear, which should be made a routine in all cases where meningitis is suspected, will reveal tympanic distension, the relief of which is followed by a speedy disappearance of the symptoms. In this connection it must be borne in mind that meningitis and intracranial abscess never follow directly upon acute otitis, but they are the sequelæ of chronic otitis, which has resulted in caries of the temporal bone. When evidences of caries of the middle ear are present in a case presenting cerebral symptoms, distinction has to be made between meningitis and abscess of the brain or cerebellum. Here the presence of localising symptoms, either temporal or cerebellar, and the presence of papilloedema and any tendency to a temporary abatement of the symptoms point to the existence of an abscess, and further lumbar puncture will in all but the rarest cases settle the point. In cases of abscess in which cells and organisms are found in the cerebro-spinal fluid, these exist in small numbers only, as compared with the copious cells and organisms present in the fluid of suppurative meningitis.

Treatment.—In cases of meningitis secondary to temporal caries, the source of infection should be at once cleared out by surgical procedure. Repeated lumbar puncture may relieve symptoms, and injection of an anti-serum to the organism present may be tried. Vaccines may also be used.

The *treatment and prognosis* of all the foregoing forms of meningitis, except tuberculous meningitis, have undergone a great change since the introduction of sulphanilamide and its derivations. The mode of employment of these drugs is dealt with on p. 49 (cerebro-spinal fever).

5. BENIGN ASEPTIC MENINGITIS

Synonyms.—Epidemic Serous Meningitis; Benign Lymphocytic Meningitis.

Ætiology.—This is unknown. No organisms have been found in the cerebro-spinal fluid. The malady so named appears of wide distribution.

Pathology.—Since recovery is the rule, nothing much is known of this, but lymphocytic infiltration of the lepto-meninges has been found in one fatal case.

Symptoms.—Children are mostly affected, but no age appears exempt. The onset is abrupt, with headache, sickness and fever. The typical signs of meningeal irritation are present, neck and spine rigidity, Kernig's sign, irritability and restlessness and sometimes delirium. Somnolence is unusual. In young children convulsions may occur. The fever mounts to 102 or 103 and fluctuates. It may disappear in 2 or 3 days, or persist for 3 weeks. Lumbar puncture yields a cerebro-spinal fluid under pressure, usually clear but sometimes opalescent. The cell count ranges from 50 to 1500 per c.mm. After the first two or three days these cells are almost wholly lymphocytes. The sugar and chloride contents remain at normal height, thus differing from the findings in other forms of acute lepto-meningitis, and resembling the findings in acute poliomyelitis.

Diagnosis.—This depends upon the cerebro-spinal fluid findings and upon the benign course of the illness. For a few days differentiation from poliomyelitis may be impossible.

Prognosis.—Recovery is the rule.

Treatment.—Repeated lumbar puncture reduces the intracranial tension. Beyond this, only general nursing care is needed.

6. SYPHILITIC MENINGITIS

Meningitis due to infection by the *Spirochæta pallida* is one of the characteristic lesions met with in practically all cases of syphilitic disease of the central nervous system, and plays its part in the production of the symptom complexes of these maladies, from acute cerebral syphilis and acute myelitis to general paralysis and locomotor ataxy. It may occur at any period after infection, but one-half of the cases occur during the first four years. In a few cases the symptoms have been noticed coincidently with the syphilitic roseola.

Pathology.—The morbid process consists essentially in an infiltration of the meninges with lymphocytes and plasma cells, spreading from the perivascular lymphatics where the spirochætes multiply freely, and it may lead to scarring and opacity of the membranes, with consequent strangling of the nerves and vessels and occlusion of the arachnoid space, or to massive gummatous formation in the meninges. It is essentially a chronic form of meningitis though it may result in the production of acute symptoms. A marked feature is that the meningeal changes may be found actively progressive in one spot, and equally regressive in another. The disease may be local or diffuse, and it may attack the dura (pachymeningitis) and involve the overlying bone, or it may spread from the pia-arachnoid into the sublying nervous tissue (meningo-encephalitis).

The cerebro-spinal fluid is characteristic. It is usually under increased

pressure, is clear and colourless, and contains lymphocytes and no other cell forms. The number of the lymphocytes present is in direct proportion to the activity of the meningeal syphilis. The spirochæte has rarely been found in the fluid, yet inoculation of apes with the fluid has proved successful.

Symptoms.—Apart from those conditions of nervous syphilis in which meningitis is associated with arterial disease, the formation of massive gummata and neuronie degeneration, syphilitic meningitis may be described as giving rise clinically to the following conditions :

1. *Headache.*

2. *Hydrocephalus.*—In those acute cases of cerebral syphilis characterised by rapidly oncoming headache, vomiting and papilloedema, mental reduction and somnolence without localising symptoms, and which respond readily to treatment, it seems certain that ventricular distension, consequent upon adhesive meningitis and ependymitis, is responsible. A more slowly oncoming ventricular occlusion may give rise to symptoms which cannot be distinguished from those caused by a non-localisable intracranial tumour. Syphilitic meningeal occlusion may give rise to typical hydrocephalus, and a considerable proportion of the cases of infantile hydrocephalus are of this nature and are due to congenital syphilis. A few cases are recorded in which chronic hydrocephalus of this nature has occurred in adult life.

3. *Infantile syphilitic meningitis.*—This is a chronic malady which commences insidiously during the first few months of life, with signs of general nervous deterioration. The appearance of the brain is very characteristic. The membrane over the vertex is opaque and thickened and adherent to the cortex. The gyri are shrunken, the sulci wide and the surface of the brain has in parts the appearance of wash-leather. The child does not get on, and takes an ever-decreasing notice of its surroundings. Power of movement lessens, the limbs become rigid and the clinical aspect comes to resemble exactly that of a severe cerebral diplegia. Convulsions are of frequent occurrence. The diagnosis is not difficult, for the signs of meningitis are obvious and those of congenital syphilis may be present. There is an excess of lymphocytes in the cerebro-spinal fluid, both in which and in the blood there is a positive Wassermann reaction. The prognosis in any case where the symptoms have become marked is most unfavourable.

4. *Adult syphilitic meningitis,* with a symptom-complex closely resembling that of tuberculous meningitis, has been reported on many occasions. In some of the cases the onset coincided with the appearance of the syphilitic roseola. The diagnosis depends upon the presence of signs of active syphilis, upon the cerebro-spinal lymphocytosis and upon the existence of a positive Wassermann reaction. The prognosis under appropriate treatment is good.

5. *Paralysis of cranial nerves.*—This common and often isolated symptom of nervous syphilis may result from sclerosing basal meningitis or from the presence of a gumma in the course of the nerve. Several of the nerves may be involved together in one patch of meningitis. Any of the cranial nerves may be affected from the olfactory to the hypoglossal, but the third or oculomotor nerve is by far the most frequently attacked.

Treatment.—The treatment of the above conditions is that appropriate for nervous syphilis in general (p. 1636). The combined administration of mercury by inunction and of arsenic compounds by intravenous injection

gives the best results. Iodide of potassium is not nearly so useful as when massive gummata are present, and, moreover, it seems to increase the scarring process. Its use should be avoided until the patient has been under the influence of mercury for some time.

7. OTHER FORMS OF MENINGITIS

Meningitis due to the typhoid bacillus is a rare malady. It may occur as a primary disease, but is usually a complication in the course of enteric fever. It is to be remembered that while many cases of enteric fever present cerebral symptoms, in very few can meningitis be proved to exist. The meningeal exudation, generally serous, is sometimes purulent. The cerebro-spinal fluid contains lymphocytes, and Eberth's bacillus is present. The symptoms resemble those of acute meningitis in general. The diagnosis depends upon the presence of enteric fever, of Widal's reaction and the discovery of Eberth's bacillus in the cerebro-spinal fluid. The malady is generally fatal, but a considerable number of recoveries have occurred, especially in children. In rare cases symptoms of meningitis occur in the course of rheumatic infection, and Poynton and Paine have brought forward evidence that such symptoms are the result of infection of the meninges with the *Diplococcus rheumaticus*. The term "serous meningitis" is applied to those cases of meningitis in which the cerebro-spinal fluid is clear and sterile. In such cases recovery is the rule, and the symptoms are not rarely rapidly relieved by lumbar puncture. The term "meningism" is used for a group of cases which present symptoms of meningitis and in which no pathological change can be found either in the cerebro-spinal fluid, or, if death occur, in the meninges or cerebral tissue. It is met with in children in association with acute febrile diseases, and is presumably due to the toxin present. Recovery is usually rapid and complete.

VIRUS DISEASES OF THE NERVOUS SYSTEM

Certain viruses have a selective affinity for the nervous system and are therefore spoken of as "neurotropic." They act upon the nerve cell, and to a less degree upon glia cells, but not upon the white matter. They are capable of multiplication and of exerting their pathogenic action only within the nerve cell, where their life and activity are short-lived. Thus, the infections to which they give rise are known as self-limited.

The essential lesion resulting from their presence is an acute necrosis of the nerve cell, leading to the death and destruction or to the damage of the cell. A secondary glial and vascular reaction ensues as a result of which lymphocytes pass into the cerebro-spinal fluid from the perivascular spaces in the affected regions of the nervous system.

Such virus infections are primary infections of the nerve cell and not invasions of the central nervous system secondary to general infection. The portal of entry in all cases, except that of rabies, is probably the nasopharynx by the mechanism of "droplet infection." The virus is carried thence via the olfactory filaments into the olfactory bulbs and tracts. Gaining direct access

to the brain in this way it seems certain that the virus is thereupon transported within the system through axis cylinders. This at least appears to be the mechanism in the most closely studied of the virus infections of the nervous system, acute poliomyelitis.

The so-called post-infective encephalitis that may follow the acute exanthemata is not a true virus infection of the nervous system, since the lesion is a demyelination and not an attack upon the nerve cell. This form of encephalitis, therefore, is probably to be regarded as an intoxication of the nervous system associated with a systemic virus infection.

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ACUTE ANTERIOR POLIOMYELITIS

Synonyms.—Infantile Paralysis ; Heine-Medin Disease.

Definition.—An infection of the nervous system by a neurotropic virus which acts primarily upon the nerve cell, the interstitial tissues of the nervous system and the leptomeninges being secondarily involved. Contrary to what was formerly believed, there is no initial phase of general systemic infection ; from the outset, poliomyelitis is a disease of the nerve cell. The virus is an obligatory intracellular parasite, and has a special affinity for the ventral horn cells of the spinal cord, within which it multiplies during its brief period of activity.

Ætiology.—A constant feature of the disease is its incidence in young children. They appear to be immune during the first year of life, susceptibility being greatest in the second, third and fourth years, and thereafter declining. Cases during adolescence and early adult life are not uncommon, but the disease is rare after middle life. It is possible that a very large proportion of the community has at some time been infected by poliomyelitis, only a very small proportion (less than 1 per cent.) having developed symptoms of infection. From 50 to 80 per cent. of the adult population possess serum containing protective antibodies, and this fact is taken as presumptive (though not certain) evidence of previous infection. That such clinically latent infection is possible may be seen from the occurrence of the many "abortive" cases of poliomyelitis in all epidemic outbreaks. The disease is much more prevalent during the hotter months of the summer, usually the months of August and September in the Northern hemisphere, and the months of March and April in the Southern. It is spread by human carriage by infected persons in the initial stage of their illness, and probably also by healthy carriers. The nasopharyngeal secretions are infective during the first ten days of the illness, and spread probably takes place by what is known as "droplet infection." It has been said that case-to-case infection does not occur, but the long-continued case-incidence in certain small, enclosed communities strongly suggests that, however difficult it may be to trace, such infection does in fact occur, and must be allowed for in dealing with outbreaks of the disease. There is some evidence that milk may also act as a channel of conveyance of the virus.

THE PATH OF INFECTION.—It is exceptional for the virus to be found in the blood or cerebro-spinal fluid, and it is not by either of these channels that it reaches the central nervous system. The portal of entry into the body is

the nasopharynx, whence the virus enters the free ends of the olfactory nerve filaments, and travels by the axis cylinders directly into the olfactory bulb, and thence into the brain. The subsequent passage of the virus to the ventral nerve cells is believed to be entirely axonal.

It is believed that *all* the symptoms of the declared disease, from the first onset to the appearance of paralysis, are due to the effects of the virus acting upon nerve cells in its passage from the site of entry into the brain, until it reaches its site of election in the ventral horn cells of the lumbar region of the cord.

Pathology.—The virus of poliomyelitis is an obligatory intracellular parasite, and its action takes place entirely within the nerve cell. The changes seen in the nervous system vary with the virulence of the infection. In very severe infections the nerve cells undergo acute necrosis. If the experimentally infected animal be destroyed at this initial stage no lesions other than these cell changes are found, and the rapidly ensuing cellular exudation and meningeal infiltration seen in fatal human cases are not present. But the necrosis of nerve cells in the surviving patient is naturally shortly followed by phagocytic processes, and amœboid (microglial) cells and polymorphonuclear leucocytes rapidly invade the affected areas and clear away the dead nerve cells. It is essential to bear in mind, however, that these processes are secondary and not, as used to be thought, the primary and essential lesion of poliomyelitis. In less severe infections, less acute forms of nerve cell changes are seen, and with these the cellular exudation is almost wholly of amœboid microglia cells. These phagocytic cells fill the perivascular spaces in the affected parts of the cord. Together with leucocytes, these cells finally overflow into the cerebro-spinal fluid. They appear here even before the development of paralysis; and it is this early indication of meningeal infiltration that led to the view formerly held that a meningitis preceded the involvement of the nervous system. In the affected regions of the grey matter of the ventral horns, some cells always remain unaffected by the virus.

Lesions in the viscera have been described: namely, hyperplasia of the lymphoid tissue and splenic enlargement—but these are not constant, and their presence at the final stage of the malady is of uncertain significance. They are no longer held to indicate the occurrence of systemic infection in poliomyelitis.

Cerebro-spinal fluid.—The fluid is clear, colourless, or faintly yellow, and under high pressure, and the titre of chlorides and sugar is normal. The protein content is increased. There is usually a pleocytosis from a moderate to a very large number of cells, which disappears rapidly with convalescence and is usually gone in a fortnight. It is usually described as polymorphonuclear at first and rapidly becoming lymphocytic, but in our experience this is certainly not the rule. We have seen the fluid normal throughout in indubitable cases; we have seen a pure lymphocytosis throughout and from the preparalytic stage; and we have seen high polymorph pleocytosis, both early and late, and also with a relapse on the tenth day. The nature and number of the cells seem not to afford any prognostic indications.

Blood.—In the early stages of the malady, there is a constant and very marked polymorpho-nuclear pleocytosis, which may reach as high as 30,000, with lymphocytic leucopenia. This leucocytosis disappears when the fever abates.

Symptoms.—It has been the practice to divide the clinical course of poliomyelitis into an initial stage of general infection; a second stage of meningeal involvement; and a final stage of paralysis from invasion of the nervous tissues. Cases in which recovery ensued after a brief febrile illness and without paralytic manifestations were spoken of as “abortive,” it being supposed that the infection had died out before the virus had invaded the spinal cord. This classification, based as it was upon an erroneous pathology, may now be abandoned in favour of a division into preparalytic and paralytic stages; it being understood that in both stages, and therefore also in abortive cases, the virus has in fact invaded the nervous system before any symptoms whatever have appeared, and that even the symptoms of the preparalytic stage are almost certainly the expression of the action of the virus upon the nervous system. In short, there is no clinical stage of poliomyelitis in which the virus has not already gained access to nerve cells, and begun to exert its pathogenic effects. The failure of serum therapy in the past is probably to be explained by this fact.

The preparalytic stage.—This may last from 1 to 4 or 5 days. It is almost invariably febrile, the temperature rising to 103 or 104 degrees. The pyrexia lasts for from 1 to 3 days and then declines, sometimes finally, sometimes recurring for a day or two as paralysis develops.

To those who have studied the disease in the course of a large epidemic there can be no doubt that the clinical picture of the opening phase is pathognomonic, and clearly to be distinguished from that of other febrile illnesses of childhood. Draper has given a very vivid account of this stage, from which the following statement is taken. The child is commonly flushed and miserable, and may be drowsy, but it presents a typical appearance of mingled apprehension and restlessness, and may be very irritable. In severe infections the child breathes rapidly, appears pre-occupied and in a state of tenseness. An ataxic tremor and involuntary muscular jerkings may be present. Extreme fearfulness, and confused and alarming dreams are common. The child is hypersensitive to even the lightest touch and resents being moved. Vomiting, probably of central origin, may also be present. Headache, pain in the neck and back, stiffness of the spine and pain in the back on active or passive flexion, diminution of tendon jerks, and some diffuse weakness all appear in sequence, and finally paralysis suddenly appears in one or more groups of muscles. On the hypothesis that the sequence of symptoms in poliomyelitis from the opening of the clinical picture until the appearance of paralysis accurately represents the passage of the virus through the nervous system from olfactory bulb to ventral horn cell, Faber has drawn up the following table of symptoms.

<i>Progress of Virus.</i>	<i>Characteristic Symptoms.</i>
Olfactory bulb to hypothalamus and brain stem.	<div style="display: flex; align-items: center;"> <div style="font-size: 3em; margin-right: 10px;">{</div> <div> Fever, drowsiness, alternating with restlessness, sweating, general hyperæsthesia, apprehension, fear, irritability, heightened sensibility to pain, headache and vomiting. </div> </div>
Posterior columns of cord. Posterior root ganglia.	
	<div style="display: flex; align-items: center;"> <div style="font-size: 3em; margin-right: 10px;">{</div> <div> Localised pains and tenderness, disturbances of pain sensibility, pain on flexion of spine, Kernig's sign, transiently increased tendon jerks. Temperature may fall now. </div> </div>

*Progress of Virus.**Characteristic Symptoms.*

Anterior horn cells.	{ Twitchings, tremor, unsteady movements.
No further spread :	{ Flaccid paralysis, loss of tendon jerks.
Infection dies out.	{ Diminution of extent of paralysis, disappearance of pain and tenderness.

It would probably be premature to say that every stage in this progress of the virus has been finally and conclusively proved, but it is of course quite reasonable to take the view that all the initial symptoms of poliomyelitis are capable of being produced by an infection wholly confined to the nervous system; in other words, the assumption that there is an initial stage of general infection is superfluous, and without supporting evidence.

The paralytic stage.—It was formerly the practice to differentiate a number of types of poliomyelitis according to the localisation of the paralytic symptoms. Thus there were cerebral, cerebellar, brain-stem, spinal and neuritic types. As Weston Hurst has pointed out, it is impossible to produce in the experimental monkey, no matter what the site of inoculation of the virus, any but a spinal type of paralysis, and it is extremely doubtful if in the human disease there are any other than brain-stem and spinal cases. The remaining cases are probably due to some other cause than the virus of poliomyelitis, and they will not be considered here.

1. SPINAL FORM.—In young children, the paralysis is often not apparent until the second or third day of the illness. In older children and in adults, the paralysis is usually present within 24 hours of the onset. The paralysis is always of the flaccid variety, with loss of the deep reflexes in the region of the paralysis, and subsequent atrophy of the muscles if it is lasting; it develops very rapidly in most of the cases, and seems to have its full limit of distribution at the moment of its appearance, which facts correspond exactly with the experimental pathology. In some cases, however, the paralysis spreads rapidly from its original site, either in ascending or, more rarely, in descending fashion. The ascending cases are very liable to be terminated with fatal bulbar involvement. In rare cases, relapse occurs, and the paralysis, after remaining stationary for several days, may spread suddenly to other regions. This event, which is due to a recrudescence of the infection, has also been observed in experimental poliomyelitis. It is often stated that relapsing cases are usually fatal, but in a very considerable experience of such cases we have never seen a fatal issue, which on theoretical grounds should be highly improbable, on account of the rapid development of immunity after infection.

The paralysis is generally much more widely spread at the onset than it is destined to be permanently. At first all four limbs may be completely helpless, and later there may be complete recovery in all but one limb. The widely spread temporary paralysis is due to a recoverable affection of the nerve cells, whereas the permanent palsy is the result of an actual destruction of the cells by a necrotic lesion. The paralysis may affect any muscles of the body, but those of the legs are by far the most commonly involved, while those supplied by the nuclei of the brain stem are never permanently paralysed. The trunk muscles may be affected alone, giving rise to spinal deformity, usually of a scoliotic or kypho-scoliotic type. Thus, poliomyelitis

comes to be one of the very common causes of spinal curvature in the young. The narrowing down of the initial paralysis begins to show itself after the end of the first week, and any muscle which will recover useful power will have done so before the end of the third month. The paralysed muscles undergo atrophy, which is more rapid and complete in those cases in which there will be no subsequent recovery; they give the reaction of degeneration. They are flaccid from the first, and in the course of time tend to develop a variable degree of contracture, and yet it is common to see a limb which remains permanently flail-like. Any muscle which shows a response to faradism 3 weeks after the onset will completely recover. When a limb is paralysed, there is usually a considerable degree of vasomotor paralysis, and there may be subsequent retardation of growth. Considerable deformities of the body and limbs may arise as the result of the loss of support, which results from the paralysis, from the action of unopposed muscles, and from the contractures. Such deformity may involve actual dislocation of joints, as in the shoulder joint, where the deltoid is paralysed and the pectorals escape.

The local lesion of the spinal cord is by no means confined to the grey matter, and may involve the contiguous white matter of the lateral column sufficiently to give rise to signs of lesion of the pyramidal tract, and in rare cases of lesion of other neighbouring tracts, such as the spino-thalamic tract with a result in a Brown-Séquard's syndrome of pyramidal deficiency upon the same side and loss of pain and temperature sense on the opposite side below the lesion. Paralysis of the cervical sympathetic is not rare when the lower part of the cervical enlargement is involved, with the usual signs of a small pupil and low-lying lid on the affected side. It is, however, generally a transient event.

Disturbances of sensibility of an objective kind are rare, and are almost always transient, and amount to blunting of pain and temperature sensibility, from involvement of the spino-thalamic tracts which are continuous to the ventral horns. Subjective disturbances are common, and consist of severe local pains in the limbs, back and neck. Tenderness of the muscles, and pain on moving the joints are sometimes very prominent, and may persist for many weeks. The dominance of the clinical picture by persistent pains in the periphery constitutes the so-called "neuritic" form of poliomyelitis. Sphincter paralysis is quite common in the early stages in cases in which the lumbo-sacral enlargement is affected, but it is always rapidly transient.

The reflexes, both superficial and deep, are at first lost in the affected region, and indeed are generally absent throughout the body in the early stages of a severe case, from the general effect of the virus upon the nerve elements. In the later stages they return, or remain permanently absent, according as the muscles recover or not. Any sign of a returning reflex, either deep or superficial, in the early days of the illness is a most useful prognostic indication that the muscles concerned with the reflex will entirely recover.

2. THE BRAIN-STEM FORM.—In this type, the incidence of the lesions is upon the grey matter of the brain stem from the medulla to the region of the red nucleus. The general symptoms of the onset are as in the spinal form. In place of the paralysis of trunk and limbs there is bulbar, facial, trigeminal or ocular paralysis, according to the situation of the lesions. An extensive lesion of the medulla itself proves very rapidly fatal. Lesions of

the upper brain stem are more commonly survived, and the resulting clinical pictures are, in order of frequency of occurrence, facial paralysis, spastic tremulousness from involvement of the upper part of the brain stem, and lastly ocular paralysis with nystagmus.

Course.—Most commonly within a few days of the onset of the paralysis a very considerable remission occurs, and the paralysis becomes much narrowed down in its limits; thus, with an initial paralysis of all four limbs and trunk, the limbs recovered rapidly, leaving a permanent partial paralysis of the trunk, and in a case where both legs were paralysed, the one recovered power within the first week, leaving the other permanently crippled. Sometimes, however, there is no rapid improvement or narrowing of the region of paralysis whatever.

The paralysis remaining after the rapid improvement is final, and admits of such improvement only as may occur from the recovery of a few cells which have escaped destruction upon the confines of the inflammatory lesions, and such recovery is very slow and never reaches more than a slight degree. A certain slow improvement in those paralysed muscles which retain some voluntary power is often observable, and is referable to hypertrophy of function in those elements which remain and to the acquisition of the aptitude which necessity produces. On the other hand, children afflicted with this disease during the period of active growth will often show what seems to be a progressive diminution of power in the weak muscles, and which is, in reality, a relative failure of these muscles under the strain of the increasing weight and length of the body and limbs.

Death is uncommon at any stage in the spinal form of poliomyelitis except during epidemics, when severe general symptoms are followed by widely spread paralysis, involving all the respiratory muscles, and in these cases it takes place on the first day of appearance of the paralysis. Weakness of the respiratory muscles and especially total intercostal palsy is not infrequently an indirect cause of death, even at long periods after the onset, if bronchitis or broncho-pneumonia occur.

Diagnosis.—During the stage of general pyrexial symptoms and before the paralytic manifestations appear, a definite diagnosis can hardly be made; but it may be suggested by the time of year, by the prevalence of an epidemic, and by the combination of a polymorpho-nuclear leucocytosis in the blood with a lymphocytosis in the cerebro-spinal fluid. When the paralysis first sets in, the diagnosis has to be made from acute rheumatism, in which the painful joints may cause an appearance of severe paralysis. In the same way, syphilitic pseudo-paralysis (acute syphilitic epiphysitis) may be diagnosed from poliomyelitis. From acute polyneuritis and Landry's paralysis, both of which maladies may have a pyrexial onset with similar general symptoms, poliomyelitis can generally be distinguished by the sudden onset of the paralysis and by the absence of any spreading tendency, and probably by the lymphocytosis in the cerebro-spinal fluid, and later on by the permanent atrophic paralysis. In the rare spreading types of poliomyelitis, the latter two points alone serve to make the diagnosis.

From almost all of the local lesions of the spinal cord, membranes and roots, whether these are of rapid onset, as for example hæmatomyelia and acute myelitis, or of slow onset, such as tumour, inflammation and pressure, poliomyelitis is at once distinguished by the absence of the conspicuous

sensory loss and sphincter trouble which accompany the former diseases. In the final stage of permanent muscular paralysis and atrophy, deformities and contractures, poliomyelitis presents little difficulty of diagnosis, but it should be borne in mind how frequently deformities of the trunk and especially lateral curvature of the spine have their origin in slight attacks of this malady where the lesions are confined to the dorsal region.

Poliomyelitis may simulate meningitis so closely as to be hardly distinguishable. The skin in the former malady may be suggestively flushed and pink. A sterile cerebro-spinal fluid with no micro-organisms and with a mixed lymphocytic and degenerating polymorph pleocytosis and with the chlorides and sugar content normal can hardly be from any other case than one of poliomyelitis.

Prognosis.—It is rare for complete recovery to occur in any case of spinal poliomyelitis in which paralysis has once set in. Though recovery may be nearly complete, yet there seems always to be some region in which permanent muscular atrophy persists, and in cases which otherwise clear up, this is frequently in the spinal muscles, giving rise to a lateral curvature. From this condition of nearly complete recovery to one in which there is not the slightest recovery from the initial paralysis, there is every gradation. The prognosis is not influenced by the severity or otherwise of the general symptoms, for the paralysis may be slight where the general symptoms are severe, and vice versa. Incomplete paralysis or the presence of reflex action, either superficial or deep, in any region at the end of the first week after the paralysis has set in, is a sure indication that useful recovery will occur in that region. Those regions which remain completely paralysed for several weeks after the onset are certain to remain permanently disabled. The prognosis as to the eventual usefulness of disabled limbs, or as to eventual power of walking, depends upon a consideration of the muscles which are permanently paralysed, as to whether they are essential muscles or not, and whether they can be assisted or supplanted by any mechanical apparatus which is light enough for the weak limbs to carry.

Second attacks of poliomyelitis are exceedingly rare, but two such cases have been recorded by Eshner and by Sanz. The occurrence of progressive muscular atrophy in subjects who have in early life been afflicted with poliomyelitis is not very rare, and it is usual for the progressive atrophy to commence in the region originally affected by the poliomyelitis. Potts has recorded a series of 28 such cases, and several others are to be found among the records of the National Hospital.

Treatment.—In the acute stage, the patient should be kept at rest upon a soft bed and fed upon a diet suitable to the febrile condition. Since the malady is an infectious, specific fever, and since the virus is known to exist upon the nasal, buccal and respiratory mucous membranes, and is presumably spread therefrom, bed and utensil isolation is necessary, with sterilisation of any contamination from the mucous membranes and mild daily disinfection of the mouth and nose. Salicylates, especially in the form of aspirin, will relieve the pain and fever, and seem to be decidedly beneficial. If pain be very severe there is no contra-indication to the use of morphine. If the respiratory muscles are seriously involved, belladonna or atropine is of great service both in stimulating the respiratory mechanism and in checking accumulation of bronchial secretions.

In cases in which the respiratory musculature is involved and in which a fatal issue may for this reason ensue, apparatus has been devised to effect an artificial respiration in the hope that the paralysis may recede and the respiratory musculature resume its function. It is particularly during epidemic outbreaks that such cases are seen, the paralysis progressively ascending from the lower limbs, or spreading by "jumps" at short intervals. There are two main types of apparatus, the Drinker respirator and its derivatives which consists of a closed compartment in which the patient lies recumbent, his head protruding through a rubber collar. A motor then produces alternating air pressures which passively move the chest. Another type, the Bragg-Paull respirator, consists of a rubber apparatus strapped round the chest which, again by the use of a motor, by alternate inflation and emptying, moves the chest. There may be a few cases in which a really good result can be achieved, namely, a useful measure of general muscular recovery ensues and the patient can take up a more or less active life. More numerous, however, are those in which although sufficient active thoracic movement to sustain life returns, the patient remains bedridden for life. Cases are on record also in which survival depends upon permanent retention in a Drinker respirator. It is probable, therefore, that such machines will find their greatest field of usefulness in other maladies than poliomyelitis, such, for example, as carbon monoxide poisoning and other essentially temporary causes of respiratory weakness. A modification of the Drinker machine is now provided in most hospitals throughout the country.

It has recently been claimed by Contat in Switzerland that heavy dosage with potassium chlorate during the pre-paralytic stage of the illness averts the development of paralysis. It is known that the virus of poliomyelitis is highly sensitive to oxidising agents. The patient is given nasal instillations of 2 per cent. solution of the substance, 5 drops four times daily. By mouth the dose ranges from 5 grains in the 24 hours in an infant to a total daily dosage of from 60 to 80 grains in an adult. This total is spread over frequent small doses during the 24-hour period. The administration is begun as early as possible and continued during the febrile period (2 to 3 days), and progressively diminished to cease on the fifth or sixth day. It is said that no albumin, red cells or casts are found in the urine during this medication. Contat also employed potassium chlorate as a prophylactic in contacts.

Serum therapy.—Since Netter first initiated this method of treatment some twenty-five years ago, the administration of the serum of individuals who had recovered from a known attack of poliomyelitis ("convalescent serum") has been a widely used method of treatment. Convalescent serum has been experimentally found to contain protective antibodies, and it has been thought that it may prevent the development of paralysis if administered during the preparalytic stage, and may limit the extent and severity of paralysis if given early in the paralytic stage. Recent re-assessment of the clinical evidence strongly suggests that these beliefs are illusory, and that serum therapy is unavailing at any stage of the malady. This result is indeed what the experimental evidence should have led us to expect. For Flexner found that the administration of convalescent serum to an inoculated monkey protected only if it were given at the same times as the dose of virus, or within 12 hours and *before* the appearance of symptoms. Administered after this moment, it was uniformly ineffective. But it is only after the

appearance of symptoms that it can be given in the human subject. Again, the theory adopted to rationalize serum therapy was that poliomyelitis was a general infection, with later involvement of the nervous system. Serum administered in the preparalytic stage was thought to prevent invasion of the nervous system. We know now that the nervous system has already been invaded before any symptoms whatever develop, and that this supposed prevention is from the nature of things quite impossible. Recent controlled observations in New York, in which 50 per cent. of the patients in an extensive epidemic were given serum and the remaining cases denied it, have indicated that serum therapy has no influence upon the course of the individual case.

Kleinschmidt, in his analysis of the 1938 epidemic in Cologne, adds further confirmation to this judgment.

Rest and posture.—It is all-important to secure as complete physiological rest as is possible for the weak or paralysed muscles for some time after the onset. Even in the slightest cases, the patient should be kept in bed for at least 3 weeks, during which time attempts at volitional movements should be discouraged. The posture of the paralysed region should be such as to secure the relaxation of the paralysed muscles; for if they are kept stretched by the action of opponent muscles which are not paralysed, recovery is greatly hindered. Appropriate postures can be secured by pillows, sandbags, splints and other devices. After a few weeks have elapsed, massage and passive movements should be regularly employed and re-educational exercises commenced, where there is sufficient power. Electrical treatment in any form is of very doubtful value. Re-education should be assisted by every appropriate mechanical device, but it must be carefully borne in mind that every mechanical apparatus which overweights the weak limb places a millstone around the neck of recovery. The lightest possible shoes should be worn, and if splints are indicated the excellent and almost weightless, moulded, celluloid splints should be employed, to the absolute exclusion of all heavier varieties. In the re-education of the legs for walking, a walking-machine on wheels is a necessity. Contractures and deformities, which hinder useful action, should be dealt with by passive movements, splinting, tenotomies and other surgical procedures.

There have recently been recorded several cases of acute bulbar poliomyelitis in children upon whom tonsillectomy and adenoidectomy had been performed during the course of a local outbreak of poliomyelitis. It appears that this operation opens a port of entry to the virus. The onset of the disease is at an interval of 10 to 20 days after operation, and the issue is commonly fatal. The performance of these operative procedures upon children is therefore strongly contra-indicated during outbreaks of poliomyelitis.

LETHARGIC ENCEPHALITIS

Synonym.—Epidemic Encephalitis.

Definition.—An acute febrile disease, occurring sporadically and epidemically, due to the infection of the nervous system, presumably from the nasal passages and by a purely axonic route, by a virus, which can be inoculated into the nervous system of monkeys, reproducing the disease. The

clinical aspect is that of a lasting, as opposed to an evanescent, infection, producing chiefly inflammatory reaction, and principally incident upon the upper parts of the nervous system, the cerebrum, basal ganglia and brain stem. Though very definite, it is remarkably polymorphic, and it is sometimes mono-symptomatic, and its type has changed greatly during the passage of an epidemic. The absence of evidence forthcoming of case-to-case infection has necessitated the assumption that infection is transferred by carriers, or by those in the pre-symptomatic stage of infection only.

History.—When we read of the influenza epidemic which swept over Europe in 1580 and which was accompanied by a malady so peculiar as to gain the title of “*schlafkrankheit*,” and afterwards of the epidemic described by Sydenham in 1675 as “*febris comatosa*,” the “sleeping sickness” of Tübingen in 1712 and Dubini’s epidemic of the fatal “electrical chorea” in Northern Italy in 1846, we cannot but agree with von Economo’s conclusion that these epidemics were epidemics of lethargic encephalitis. The subsequent epidemics of Mauthner’s “*Nona*” in Piedmont in 1891, and also Pfuhl-Leichtenstern’s “*hæmorrhagic encephalitis*” in 1905 have been proved identical with lethargic encephalitis, both clinically and pathologically. The malady became pandemic from 1917, reaching a maximum in 1920, since when it has gradually declined and it is now comparatively rare, if indeed any true cases occur. We have, however, seen a good many end-results of cases which had their commencement from 1910 onwards, showing that in England this malady was increasingly present, though unrecognised.

Ætiology.—During the period of its incidence, the disease occurred both sporadically and epidemically, with no centre of spread. It was more prevalent in the cold season of the year. No age is exempt from the malady, and cases have occurred in the seventh decade of life, but it is rare in young children and seems to be most incident in the first half of adult life. Infection presumably takes place, as in poliomyelitis, from human vectors alone, and by droplet infection. When once the virus has gained access to the nervous system by a peripheral axonic route, it is “*virus en cage*,” to use Economo’s term. It is imprisoned within the nervous system and cannot get out, but it may there survive for very long periods, giving rise to second and third attacks after apparent recovery, or to exacerbations of symptoms after long intervals of remission, or to insidious and progressive severe abrogation of nervous function long years in train of slight trivial and evanescent symptoms which marked the epoch of infection. Whether the infection thus pent up in the nervous system does on occasion manage to escape from the peripheral nerve terminals, as it does regularly into the saliva in the case of rabies and often into the skin in the case of herpes, and so get free, has not been determined.

The height of the epidemic incidence of lethargic encephalitis has many times coincided with a severe epidemic of influenza, but no further connection between the two conditions is known. Claimed at one time as an aberrant form of poliomyelitis infection, von Economo’s disease has proved quite distinct, both in its age incidence, seasonal prevalence, morbid anatomy and symptomatology. Economo first succeeded in transferring the disease to the monkey by intracerebral inoculation in 1916, and Loewe and Straus first proved that the infective agent was filtrable.

Pathology.—The pressure and quantity of the cerebro-spinal fluid are

always increased, and in a few of the cases blood or the products of hæmorrhage are present. In a third of our cases the cell count has been normal. In the rest there has been a moderate lymphocytic pleocytosis, with little or no protein increase, the titre of the sugar tending to a high normal and that of the chlorides being normal. No prognostic indications can be derived from the nature of the fluid. The vessels of the brain are markedly congested and full of blood, and the colour shows a characteristic change from the normal throughout the whole of the grey matter, varying from a rosy flush to a deep salmon-pink, giving rise to the term "the rose-coloured brain." When hardened in formalin, this colour becomes a heavy purple grey. Both subdural and deeply seated hæmorrhages are occasionally found. Economo describes the anatomical picture as one of unvarying constancy. It is that of an oedematous and congested brain, with all the grey matter conspicuously reddened in contrast to the white matter, which is of normal colour. There is a non-purulent and, properly speaking, a non-hæmorrhagic inflammation of the whole grey matter exclusively, the white matter being uninvolved. There is most conspicuous perivascular lymphocytic cuffing remarkable for the absence of any polymorphs, with an intense cellular infiltration of the grey matter with elements of the microglia, while the neuroglia is unaltered and demyelination does not occur. Accompanying and succeeding these inflammatory changes is a certain measure of neuronophagia, with primary loss of the ganglion cells.

Symptoms.—In the acute forms of the malady the onset is often ushered in by general symptoms, such as shivering, malaise, headache, and fever and bodily pains, a characteristic thickly coated white tongue and constipation, and sometimes vomiting and persistent hiccough. This train of symptoms usually appears in the story as an attack of "influenza." The pyrexia does not usually last longer than a week. Countless such attacks of "influenza," distinguishable only by the occurrence of transient diplopia, or of slight somnolence, and often even without any such distinguishing features, have been completely recovered from at the time, but have been followed, after long intervals, by the slow onset of the Parkinsonism of lethargic encephalitis. Again, the epoch of infection may apparently give rise to no symptoms at all, and long afterwards an insidious onset of Parkinsonism ensues, as has happened nowadays in many of the examples of Parkinsonism in childhood.

So many and varied may be the clinical aspects of this disease that it is useful to consider the separation of clinical types which Economo has laid down :

A. Acute Types.

- (1) The somnolent and ophthalmoplegic type.
- (2) The hyperkinetic type. Spontaneous involuntary movements, sleeplessness, great mental unrest, delirium and mania are here characteristic.
- (3) The amyostatic and hyperkinetic type. In this type Parkinsonian tremor and rigidity, salivation and the greasy face are conspicuous.
- (4) The cerebellar type. The symptomatology is that of the cerebellum, and recovery usually occurs.
- (5) The bulbar type.
- (6) The ophthalmoplegic type.

- (7) The neuritic type, which simulates acute fibrositis.
- (8) The mono-symptomatic type :
 - (a) Characterised by persistent trismus.
 - (b) Characterised by persistent hiccough.

B. Chronic Types.

- (1) The progressive Parkinsonian type.
- (2) The mental type.

A combination of all the types is very common.

THE NERVOUS SIGNS.—*Mental symptoms.*—An increasing lethargy, which often becomes very deep, is present in many of the cases. In this condition the patient will lie for days without stirring a muscle, taking no heed of his surroundings and passing the dejecta under him unheeding. Yet when roused by command and vigorous bodily stirring, he will wake up and hold a very intelligent conversation, lapsing back at once when he is left alone, even though his mouth be half full of unswallowed food. In this condition, *flexibilitas cerea* may often be demonstrated in the limbs. The lethargy may last for three weeks or longer even in patients who completely recover. It passes away gradually. Unrousable coma is invariably a sign of impending dissolution. Subsequent memory of events during the early days of the lethargy may be remarkably retained. Insomnia may be a troublesome early symptom, and even when the patients are markedly lethargic they will complain that they cannot sleep. Lethargy, however, may be completely absent and the early mental state be that of vivacious excitement and talkativeness. Irritability and restlessness may be present. In some cases the first nervous sign may be delirium or mental aberration, which may rapidly develop into acute and violent mania ; such cases are rapidly fatal. In cases which recover after severe symptoms, considerable mental reduction and self-obvious mental change may persist, but we have not seen any case in which insanity has resulted. Indeed, it has been said that no sufferer from this disease ever regains his original mentality, and it is a common experience to find personality very seriously changed in the way of mental reduction. Complete incapacity for any sustained work, entire change of character, anti-social tendencies, moral perversion and depressed neurasthenic states are not uncommon sequels of the disease. (See also page 1833.)

Convulsions are very rare, but they may undoubtedly occur as in other forms of encephalitis. Indeed, the initial clinical picture may be dominated by convulsion, and closely resemble "status epilepticus" from other causes.

Ophthalmoplegia and other paralyses in the region of the cranial nerves are most often nuclear in type, but peripheral paralysis of any cranial nerve may be met with, most commonly unilateral paralysis of the facial nerve. The pupils may show every abnormality which a lesion of the nervous system can produce. Inequality, unroundness, eccentricity and loss of light reflex and ciliary paralysis may occur. The loss of light reflex may be unilateral. The external ophthalmoplegia, being nuclear in origin, involves both eyes in terms of their conjugate movements, and the upward and downward movements are as a rule more severely impaired than are the lateral movements. Bilateral ptosis is very usual, and is a most important and valuable early indication of the disease. The common error is to consider it part of the sleepy state. The nuclear ophthalmoplegia is often irregular, giving

rise to strabismus and diplopia. Either in addition to the above or existing alone, there may be peripheral paralysis of any of the oculo-motor nerve trunks. The degree of the ophthalmoplegia varies in different cases from slight diplopia with hardly noticeable strabismus to complete paralysis of both eyes. It may be rapidly transient or permanently severe. In severe cases which survive there is always some improvement in the degree of paralysis in the course of time.

Vision.—The diplopia and loss of accommodation cause much defect of vision, but many of the patients complain of a loss of vision in each eye, which is too great for any such explanation, the cause of which is not yet explicable. Papilloedema has been reported in a few cases, in one of which at least meningeal hæmorrhage had occurred. It is transient and never reaches a high degree.

Oculogyric crises.—This term is applied to recurring attacks of tonic conjugate deviation of the eyes, most commonly upwards, sometimes upwards and to one side, less commonly downwards, or downwards and to one side. We have observed one case of a child in which the oculogyric spasm always proceeded to a torsion spasm of neck, trunk and limbs so severe as to roll the patient out of bed on to the ground with each access. The spasm is met with in the chronic stage of the malady, and there is always some degree of Parkinsonism. The attacks may last from a few seconds to 4 hours or more, and may occur frequently or at intervals of several days, and as the eyes are commonly fixed in an upward direction, they are peculiarly incapacitating. They have not been reported in any other disease than lethargic encephalitis, and may constitute, with some slight facial Parkinsonism, the sole sequels of this malady. We have not found any treatment which influences the frequency or severity of the attacks.

Bilateral nuclear facial paralysis and bulbar paralysis are not uncommon. Paralysis of any individual cranial nerve may occur, and also of any individual spinal root. Such paralysees always completely recover in the course of time.

Symptoms indicative of lesion of the basal ganglia are among the most common features of the disease, and they are often the most persistent. These consist of weakness of movement, rigidity with slowness of movement, and spontaneous involuntary movements. The weakness, rigidity and slowness of movement give rise to a peculiar immobility of facial and bodily expression and movement. The face is mask-like, the neck stiff and the head moved little and slowly, the trunk bent forward and stiff, the arms held away from the trunk, the whole appearance of the patient closely resembling that of paralysis agitans. Rapid fluttering of the eyelids when gently closed is characteristic of this condition. The spontaneous involuntary movements may be of a rhythmic tremulous nature, as in paralysis agitans, or there may be slow rhythmic, choreiform, athetoid, myoclonic, irregular or highly complicated movements: these may be met with at any stage of the malady, but most commonly appear some little time after the acute stage has passed away. Fibrillation and fascicular twitching of the muscles is very common in the acute stage. In cases where bulbar symptoms, either of a spastic or flaccid kind, are present, hypersalivation of the nature of a true sialorrhœa is often a most troublesome, though transient, symptom.

In addition to the above common symptoms and signs, other indications of involvement of the cerebral hemispheres may occur. Bilateral spasticity

with signs of involvement of the pyramidal systems, increased jerks, lost abdominal reflexes and extensor plantar responses are common. Hemiplegia, aphasia and hemianopia may occur, presumably as the result of local sub-cortical hæmorrhages. Meningeal symptoms may be very marked in the early stages, such as suboccipital headache, painful stiffness of the neck, head retraction, vomiting and Kernig's sign. Indeed, we have seen rapidly fatal cases in which the clinical picture throughout was hardly distinguishable from that of acute meningitis, but none of these cases showed any leucocytosis in the cerebro-spinal fluid. A major incidence of the lesions upon the cerebellum gives rise to the picture of acute cerebellar ataxy following a lethargic onset, and the end-result may be a condition closely resembling a usual type of disseminate sclerosis. Such cases make a good recovery in the course of time.

Peripheral pains are sometimes very severe and are usually quite local. They may be the first signs of the illness, and several of our patients had been treated for trigeminal neuralgia, brachial neuritis or sciatica before any other sign of the malady appeared. These pains may persist for months after recovery. They are obviously due to the lesion around the nerve roots which has been already referred to.

Spinal symptoms.—Since the lesions have been found in the spinal cord, it is only to be expected that focal spinal lesions should be met with in rare cases. These are usually acute atrophic paralyses similar to those of poliomyelitis. Those that we have seen have completely recovered. It has been argued, however, that this atrophic palsy is due to a lesion of the spinal roots. More severe lesions may apparently give rise to a condition resembling acute transverse myelitis.

Sphincters.—The incontinence which is almost constantly present, even when the lethargy is far from deep, is the result of the lethargy. Transient conscious dysuria is however not infrequent in the early stages of the disease. The deep reflexes may be lost in severe cases during the acute stages, and they are usually absent in premortal conditions. Otherwise they tend to be exaggerated, especially if involvement of the pyramidal system be present. The condition of the abdominal and plantar reflexes depends upon the presence or absence of lesions affecting the pyramidal tracts. In the former case, the abdominal reflexes will be absent and the plantar reflexes of the extensor type.

Attention must be drawn to a group of cases in which the initial manifestations of the disease are so slight as not even to interfere with the daily work or to call for medical attention, and yet in the course of months, or it may be even years, the most serious and completely incapacitating paralysis appears. A patient of ours noticed that he saw double, and did not feel very well for a few weeks. He recovered, but, two years later, had to give up work, by reason of a slowly oncoming Parkinsonism, which became extreme. A similar result in the slow and late development of grievous symptoms may follow any attack of lethargic encephalitis and make the prognosis in this malady very difficult.

Sequelæ.—The disabilities which this malady may leave in its wake seem endless and ever increasing as clinical experience widens. The mental, paralytic and Parkinsonian end-results have already been referred to, but special mention must be made of involuntary spontaneous movements,

recurring rhythmic movements, spasms and altered respiratory rhythm. Ceaseless rhythmic pulsatile movements may occur in any muscle, movements like those of convulsive tic may incapacitate the patients. Hideous recurring spasms may appear, sometimes local, sometimes general. Torticollis may occur. An unduly rapid respiratory rhythm may be established. (See also p. 1705.)

Course.—The course of the disease is extremely variable. It may be a slight transient illness lasting but a few days, and leaving no sequelæ after a few weeks; or a most malignant disease, fatal in a few days. In others, symptoms indicative of fresh lesions may occur repeatedly weeks and even months after the onset.

Diagnosis.—A diagnosis of lethargic encephalitis is even at this date (1941) not rarely made, but must be received with the very greatest reserve. Under this title most neurologists have encountered a great variety of nervous disease, including intracranial tumour, cerebral abscess, subdural hæmatoma, tuberculous meningitis, and the like. In typical cases the diagnosis presents no difficulty, the rousable lethargy, incontinence, ophthalmoplegia and negative, lymphocytic, or blood-containing cerebro-spinal fluid being so characteristic as to preclude possibility of error. The less usual forms of the malady, and especially those with very gradual onset and slight symptoms, often present great difficulty and require much care and full knowledge of the possible symptomatology of the disease for their recognition. There is no specific laboratory test for the malady, and the diagnosis must be based upon clinical grounds. Where meningeal symptoms are prominent, distinction has to be made from other forms of meningitis and from poliomyelitis. Here, the cerebro-spinal fluid is of the highest importance, as no polymorpho-nuclear leucocytes occur in lethargic encephalitis. In cases commencing with peripheral pains, excitement, maniacal symptoms or convulsions, careful look-out should be kept for the advent of ptosis, ophthalmoplegia, or lethargy, the appearance of which, following such symptoms, should at once suggest the diagnosis. It must be borne in mind that the clinical picture of the disease may be dominated by a hemiplegic condition, and that an apoplexy may occur during the acute stage of the disease. Slight cases of the disease are frequently unrecognised, or are indeed unrecognisable in the early stages, but here the diagnosis can often be made with certainty from the end-results; the peculiar ophthalmoplegia, the spontaneous involuntary movements, and the paralysis agitans-like syndrome being almost pathognomonic of the malady.

Prognosis.—A rapid onset and quick development of severe symptoms, marked pyrexia, delirium and maniacal excitement are bad prognostic signs and indicate a rapidly fatal issue. After the third week of the disease, the probabilities are all in favour of survival. The prognosis, however, as to how much permanent damage to the nervous system will eventually remain is hardly possible, since slow improvement may go on for months and even years. Of the acute cases occurring at the height of an epidemic, 40 per cent. are quickly fatal, 30 per cent. are reduced to chronic invalidism, and 30 per cent. recover completely (Economo). The spontaneous movements, even when very marked, may clear up in from 3 months to a year. The weakness, rigidity and tremors, which form the paralysis agitans-like picture in many of the cases, persist indefinitely.

Treatment.—Nothing being known of the infectivity and mode of spread of the disease, isolation and disinfection are not usually employed. Each case must in England be immediately notified to the public health authorities. No treatment is known which has any specific influence upon the disease. Intravenous injection of collosol iodine solution (150 c.c. for a dose), repeated on the second and fourth days, has been advocated, and is certainly without harmful effects. Intravenous sodium salicylate, in 15-grain doses in normal saline daily, certainly seems to clear up the symptoms in some cases and may do permanent good. It remains therefore to use those measures which will help to keep the patient alive and those which relieve symptoms. Relief of the constipation is most important and is often followed by striking improvement in the symptoms. After the acute stage, treatment is concerned with combating the physical and mental listlessness and depression, removing the rigidity with massage, passive movements and exercise, and withal brightening the days of a convalescence which is often long, tedious and hard to bear.

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Revised by F. M. R. WALSH.

HERPES ZOSTER

Synonym.—Zoster; Shingles.

Definition.—An acute infection of the posterior root ganglion, probably by a neurotropic virus, leading to severe pain in the distribution of the corresponding posterior root, and to the appearance of a crop of vesicles in the cutaneous distribution of the root.

Ætiology.—The virus of zoster stands in some as yet undetermined relation to that of chicken-pox, and the appearance of the latter malady in a susceptible subject some 14 days after contact with a case of zoster has been too frequently recorded to be of the nature of coincidence.

The disease is seen at all ages, but according to Head is perhaps most common in adolescents. In elderly patients it is frequently a more serious as well as a more painful affection than in young persons. It may arise without discoverable cause and with a febrile reaction and considerable malaise. It may also occur apparently "symptomatically" during the course of arsenical medication, or during such illnesses as pneumonia, tabes dorsalis and tuberculosis.

Pathology.—The essential lesion is an acute inflammation of the dorsal root ganglion of the same histological character as the lesion of acute anterior poliomyelitis. There are degenerative nerve cell changes, with accompanying microglial reaction and perivascular infiltration with round cells. Later, degenerative changes occur in the fibres of the dorsal roots and of the peripheral sensory nerves. The Gasserian ganglion and the thoracic and upper two or three lumbar ganglia are most often affected. There is an increased protein and lymphocyte count in the cerebro-spinal fluid.

Symptoms.—There may be an onset with fever which persists for 2, 3 or even 4 days. There is from the first pain at the place at which later

the herpetic eruption is to appear. This occurs on the third or fourth day of the illness. At first the rash is a patchy erythema, upon which appear small vesicles filled with clear fluid. From the fifth to the tenth day the vesicles dry up and shrink progressively until a scab is formed. This finally drops off, sometimes leaving considerable scarring. These scars may be anæsthetic to touch, pinprick and temperature sense. The pain before and during the evolution of the cutaneous lesion may be intense. It is of a burning and itching quality, and in frail and elderly persons it may persist as a most intractable post-herpetic neuralgia for months or even years.

Zoster of the ophthalmic division of the fifth nerve is most commonly found in elderly persons. Corneal vesicles may form and burst, giving rise to ulcers, which may spread and end in residual scarring (nebulæ), which impairs vision.

Herpes of the geniculate ganglion occasionally occurs. The vesicles are found in the pinna, and there is pain in this region, over the mastoid, and sometimes in the fauces (see p. 1512).

Localised paralysis may accompany herpes. Thus, in ophthalmic herpes there is occasionally third-nerve palsy, with ptosis and squint. In geniculate herpes, facial palsy and loss of taste over the anterior two-thirds of the tongue is the rule. In herpes of the lower thoracic ganglia there may be paralysis of the oblique abdominal muscles on the affected side. The marked local bulging of the abdominal wall which ensues resembles at first sight the presence of an abdominal tumour. These paralysees do not invariably clear up, though the facial palsy of geniculate herpes does so more frequently than the paralysis of the abdominal muscles.

Treatment.—The course of the cutaneous lesions is not influenced by treatment, which is directed to keeping the vesicles dry and free from infection. For this purpose a dusting-powder of starch or zinc oxide, or a collodion dressing may be used.

During the acute stage, pain may be relieved by aspirin or phenacetin, but the post-herpetic neuralgia of some elderly and debilitated patients may prove intractable, and so severe as to render life scarcely tolerable. A neurosis may be grafted on this pain and render the situation extremely difficult for the doctor. Various local remedies have been recommended, including rays of all kinds, local heat, electrical currents, analgesic applications, and so on. But none can be relied upon to afford material relief, though the neurotic patient may obtain some comfort from any of them that commands his faith. It may be necessary to keep the patient under some such mixture as the following: tinct. gelsem. min. 10, phenazone grs. 10, phenobarbitone. sol. gr. $\frac{1}{2}$, sod. bromid. grs. 10. Aq. chlorof. ad fl. oz. 1 t. d. s.

A warning may be sounded against prolonged bromide medication for elderly subjects. It sometimes renders them confused and feeble, and these debilitating effects may take some weeks to pass off even after bromide is withdrawn from administration. For such patients, small and carefully adjusted doses of phenobarbitone are probably better.

RABIES

Synonyms.—Hydrophobia ; Lyssa.

Definition.—This is an infective disease due to a filtrable virus which is located in the salivary glands and central nervous system. It is transmitted to man and most warm-blooded animals through infective saliva of canines or blood-lapping bats. There is a long and variable incubation period, and a short pyrexial illness of sudden onset characterised by fever, nervous exaltation and violent muscular spasms involving the œsophagus and respiratory system. Once symptoms have supervened, the patient invariably succumbs.

Ætiology.—The disease is generally transmitted either by the licking of a freshly abraded surface of skin or the bite of an infected dog. In Eastern Europe and the Orient, wolves not uncommonly transmit the disease and, owing to extensive laceration of the tissues, a greater proportion of people bitten by them develop the disease than with either dogs or jackals. It has been estimated that wolf bites entail a mortality of 80 per cent. In Trinidad, in 1925 an epidemic of paralytic rabies in man was attributed to the bites of vampire bats, cattle being the original source of infection.

The virus of rabies.—This is now known to belong to the class of neurotropic viruses that have a special affinity for attacking the grey matter of the nervous system. To this class belong also the viruses of acute anterior poliomyelitis and of Borna disease.

Pasteur, in 1881, discovered that rabies could be transferred in series from animal to animal by subdural inoculation of emulsions of central nervous tissue derived from an infected dog. In rabbits, after some twenty passages, the virus became modified : firstly, the incubation period of ordinary street virus which varied from 8 to 60 days was reduced to 7 days ; and, secondly, it lost its capacity to reproduce the disease on subcutaneous inoculation. Such a virus is known as fixed virus or virus fixé. Street virus, on the other hand, is transmitted from the local wound via the peripheral nerves to the central nervous system, and if the sciatic nerve be inoculated the lumbar cord becomes infectious several days before the virus can be demonstrated in the brain (di Vestea and Zagari). This accounts for the fact that cases bitten about the face, head and neck have such a short incubation period. The salivary virus is filtrable through coarse Chamberland and Berkefeld filters—not so, emulsions of infective brain tissue. The virus is destroyed at 50° C. and is attenuated by drying—a fact made use of in the preparation of anti-rabic vaccine by the Pasteur method.

Pathology.—Excess of cerebro-spinal fluid, petechial hæmorrhages of the piaarachnoid and injection of its vessels may be found at autopsy. Histological examination reveals cellular infiltration of the perivascular lymph spaces as well as Negri bodies within the cytoplasm of the nerve cells and their processes. These bodies were described by Negri in 1903. They are globular or ovoid structures, of variable diameter (0·5–25·0 microns), and are especially common in the Purkinje cells of the cerebellum and the hippocampus. Though demonstrable in the brain of 97 per cent. of dogs infected with street virus, they never appear in the salivary glands—a fact which supports the view that they are not parasitic protozoa, as Negri suggested, but rather some kind of cell inclusion or degenerative structure.

Symptoms.—The period intervening between the bite and the clinical

manifestations varies from 1 to 2 months as a rule, the limits being 11 days to over a year. Face, head and neck bites have a shorter incubation period than those on the upper extremity, and arm bites a shorter incubation than those implicating the leg. The onset is generally sudden, but prodromal symptoms are sometimes noted for a day or two before a hydrophobic syndrome appears. For convenience, three stages are described.

1. *The invasion stage.*—This includes prodromal features such as pain in the scar, fever, headache, rapid pulse, anxiety, restlessless, insomnia, irregular and sighing respirations, and phases of rushed speaking.

2. *The stage of excitation.*—This supervenes in 24 to 48 hours. There is intense restlessness, mental excitement, hyperæsthesia and hydrophobia which consists of a sudden spasmodic spasm of the muscles of the mouth, pharynx and larynx and, to a greater or lesser degree, the whole respiratory musculature. A typical attack may be induced by offering the patient water. As the glass approaches the mouth, the head retracts in a series of spasmodic jerks associated with gasping respirations, while any water reaching the mouth is immediately ejected. The shoulders are elevated, the chest expanded, and the sterno-mastoid and platysma muscles contracted. Later, the synaptic resistance in the reflex arcs becomes so lowered that a variety of sensory stimuli such as a sudden sound, cold air, strong light, a strange smell, and even the suggestion of water may suffice to induce the attack. The voice is altered. Frothy saliva collects in the throat and mouth and is flung off the lips during the attacks which may be characterised by intense fury or the most profound terror. Later, opisthotonus and general respiratory spasm are superadded. In the interval the mind is clear, the patient remaining quietly at rest in bed. Examination of the central nervous system reveals, as a rule, nothing more than increased deep reflexes. Glycosuria is not uncommon, and vomiting, exhaustion and emaciation characterise the final stage of the illness. Death during the paroxysm may occur from dilatation of the right heart, though sometimes near the end the spasms ameliorate or cease altogether.

3. *Stage of paralysis.*—If the patient survives long enough, paralysis of various types, including Landry's ascending paralysis, paraplegia and hemiplegia, may supervene. The patient lies helpless and exhausted, and generally dies in coma. In man this stage is rarely seen in canine-transmitted rabies, but paralytic rabies is commonly encountered in the bat-transmitted variety in Trinidad.

In the Trinidad outbreak all the cases were of this variety, and all proved fatal. The onset is acute, with fever and headache. Numbness and burning sensations in one or both legs, paresis of the legs and retention of urine follow. After 2 or 3 days the paraplegia becomes more complete, and the plantar and tendon reflexes disappear. One limb is commonly affected before the other. In a few days the paralysis begins to ascend, involving the muscles of respiration, of articulation and deglutition. There is dyspnoea, restlessness and death. The sufferer remains conscious, but may be delirious. Sensory changes are of variable intensity. A final brief coma precedes the fatal issue. During this time the temperature swings round 103° F., and there is profuse sweating. Hydrophobic symptoms are exceptional, and when present slight. The cerebrospinal fluid yields an increased globulin content, but is otherwise normal. The duration of the illness is from 4 to 8 days.

Rabies in the dog.—These animals never show the hydrophobic syndrome

observed in man. The earliest manifestation appears to be a change in temperament, followed by irritation and exacerbations of vicious fury in which the animal runs amok, biting wildly anything in its path. Later, swallowing becomes difficult, the bark is altered, the jaw drops and general paralysis ensues. Death invariably follows some 2 to 5 days after the first symptoms appear. In dumb rabies the stage of excitation is absent.

Diagnosis.—As a rule, little difficulty is experienced in diagnosis, but occasionally tetanus, the cerebral type of typhus fever, bulbar paralysis from any cause, and datura and other poisonings encountered in Oriental countries may need differentiation. Lyssophobia or hysteroid counterfeiting of the disease generally manifests itself within the first 10 days, and is unaccompanied by fever or other serious features.

Prognosis.—By no means all patients bitten by rabid animals die, but once clinical manifestations appear the disease invariably ends fatally. Estimates varying from 5 to 33 per cent. have been made of the death-rate in untreated patients, but of those receiving early anti-rabic inoculations in Pasteur institutes, not more than 1 per cent. die. The mortality varies with the site of the bite, the interposition of clothing, the number of tooth-marks, the extent of tissue laceration and the rapidity with which efficient local treatment has been instituted. Head, face and neck bites are particularly dangerous, as well as bites from wolves and jackals.

Treatment.—This is entirely preventive, and in England the muzzling order and the strict quarantine of all imported dogs has led to the eradication of rabies. In endemic areas canine bites should be promptly treated, and the suspected dog chained up, muzzled, and kept under observation. Should the animal be alive at the end of 10 days it is proof that the bitten person has not been infected. This rule, universally followed in Pasteur institutes, is based (1) on the knowledge that the infected dog never survives longer than 6 days from the onset of its illness, and (2) that the saliva of a rabid dog is never infective for more than 4 days before the onset of symptoms. In suspicious cases, especially the head, face and neck bites, treatment should be commenced without delay and discontinued if the dog survives.

The virus of rabies differs from that of yellow fever in not passing through the intact skin, and where there is a history of being licked by an animal suspected of rabies prophylactic inoculation need not be advised unless fresh skin abrasions were present at the time.

Local treatment.—If seen within 30 minutes, bleeding should be encouraged by the application of a ligature just tight enough to obstruct the venous return and the parts bathed with permanganate solution. Subsequently, each tooth-mark should be probed separately and cauterised or treated with pure phenol. For 3 days the wound should not be sutured; this particularly applies in the case of face bites.

Anti-rabic vaccination.—Owing to the long incubation period, it is feasible to attempt immunising the patient either by the inoculation of attenuated, living, fixed virus, as in the Pasteur and Högyes methods, or by the injection of carbolised or etherised vaccines in which the fixed virus has been killed. The Pasteur treatment consists of a series of 18 injections of emulsions made from the spinal cord of rabbits which had been dried for periods of from 14 to 35 days. Semple introduced carbolised vaccine; the most potent preparation consists of a 5 per cent. carbolised suspension of sheep brain

infected with Paris virus. In mild cases the course consists of 2 c.c. injected subcutaneously each day for 7 days; in average cases of 5 c.c. for 14 days; and in severe cases, such as head, neck and face bites, in wolf bites, or in children bitten on the bare skin, 10 c.c. are injected daily for 14 days. Itchy swellings may appear at the site of the inoculations about the eleventh day, but other complications following inoculation are fortunately rare. Paralytic accidents, however, have been recorded with all methods; they include a mild facial neuritis, dorso-lumbar myelitis and an ascending paralysis of Landry's type which is fatal in about 30 per cent. of cases.

Treatment of the paroxysm.—No specific treatment is known. Measures directed to alleviate the suffering of the patient should be instituted. These include chloroform inhalations and morphine, hyosine, chloral and atropine in large doses. Curare and tetrado-toxin have both been employed for the relief of spasms.

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CEREBRAL VASCULAR LESIONS

ANEURYSM

Pathology.—Miliary aneurysms, which are small seed-like dilatations of the finer arteries in the substance of the brain, are not uncommonly present in cerebral arterial degeneration. They are usually multiple. The extensive researches of A. G. Ellis in 1909 and of Pick in 1910 have proved conclusively that miliary aneurysms are of little practical importance apart from the associated arterial degeneration.

True aneurysms of the larger cerebral arteries are not uncommon and have in the past been the subject of a very extensive literature. In a recent analysis of over a thousand cases McDonald and Korb have ascertained the following facts:

(i) The arteries involved in order of frequency are the middle cerebral, basilar, anterior communicating, internal carotid, vertebral, anterior cerebral, posterior communicating and posterior cerebral.

(ii) Aneurysms occur at all ages; about 11 per cent. were below 20 years, 35 per cent. from 20 to 40 years and 54 per cent. above 40 years of age.

(iii) The sex incidence is approximately equal.

(iv) 63 per cent. of the vessels examined showed pathological changes, the remainder being described as normal. The predominant lesion was atheroma, and less common in order of frequency were mycotic aneurysm and syphilitic changes, only 5 per cent. showing changes of the last-named order. At all ages pathological changes in the affected arteries predominated, mycotic aneurysms being most common in young persons, atheromatous changes in older subjects.

These aneurysms may give rise to no symptoms during life, being merely post-mortem findings, they may rupture, or they may be of sufficient size to evoke tumour symptoms. Of these possibilities, rupture appears to be the most common, the artery most frequently affected in this way being the anterior communicating.

The aneurysms found on normal arteries are usually regarded as due to developmental defects in the wall, commonly seen at junctions on the circle of Willis. They may be multiple, and from their shape are spoken of as "berry" aneurysms. They may rupture freely, leading to a rapidly fatal issue, or they may leak recurrently. The wall may contain calcified material which shows as a ring shadow in the radiogram. Mycotic aneurysms arise from septic infection of the arterial wall, usually in association with septic endocarditis.

Symptoms.—Clinically, cerebral aneurysms fall into two main groups : (1) Those which rupture during life with the extravasation of blood, in greater or less amount, into the subarachnoid space, and sometimes also into the cerebral substance and into the ventricles. These form the so-called *apoplectic* group. (2) Those which produce symptoms in virtue of the pressure they exert on neighbouring structures, thus giving rise to paralytic symptoms. These form the *paralytic* group. It has to be added that this second group may ultimately rupture. There are also cerebral aneurysms that during life are clinically latent, being merely necropsy findings.

The clinical picture of subarachnoid hæmorrhage, following upon the rupture of cerebral aneurysms is now widely recognised, but that of the paralytic group is only now being clinically differentiated. Recent studies by G. Jefferson has clarified their clinical picture considerably, and in most cases their presence should be diagnosed.

1. SUBARACHNOID HÆMORRHAGE

Synonym.—Spontaneous Subarachnoid Hæmorrhage.

We have seen that bleeding into the subarachnoid space may be an accompaniment of head injuries in which the meninges are torn. It may also follow primary intraventricular hæmorrhage, but the usual cause of uncomplicated, or as it is sometimes called, spontaneous subarachnoid hæmorrhage is rupture of a cerebral aneurysm on the circle of Willis or on one of its component arteries. The situations in which such aneurysms are commonly found have been already enumerated. What has been called here the "berry" aneurysm may rupture suddenly and freely with the production of fatal apoplexy, or there may be recurrent leaking of blood in small amounts from such an aneurysm, leading to a syndrome of meningeal irritation.

1. *The apoplectic syndrome.*—The subject may have been subject to frequent headaches, or the episode may be quite unheralded until a sudden intense headache, rapidly followed by sudden lapse into unconsciousness, signals the free rupture of the aneurysm. It may be thought that an ordinary cerebral hæmorrhage has occurred when the comatose patient is first seen, but careful examination will generally reveal the absence of signs of a gross lesion in one cerebral hemisphere (cf. p. 1527) when this has occurred, whereas in uncomplicated subarachnoid hæmorrhage no signs of hemiplegia are present. On the other hand, a bilateral Babinski plantar response will be obtained and there will be marked neck rigidity. At first both pupils may be small and sluggish, but in fatal cases the pupils ultimately dilate. Lumbar puncture produces a fluid that resembles pure blood.

Recovery from hæmorrhage of this severity is by no means unknown.

In fatal cases death commonly ensues within 24 to 36 hours, or at some time during the first fortnight from fresh bleeding. If this period be safely passed the prognosis as to recovery is good. Some patients enjoy years of normal health after such an episode, while others are subject to recurrences of greater or less severity.

2. *The meningitic syndrome.*—In this case, the hæmorrhage is less abundant and therefore consciousness may not be lost. There is violent headache, restlessness, delirium, rigidity of neck and spine, Kernig's sign, bilateral extensor plantar responses, and sometimes diplopia and squint. Within a few hours, or somewhat later, ophthalmoscopic examination may reveal the presence of flame-shaped hæmorrhages in the nerve fibre layer of the retina, or massive hæmorrhage in the subhyaloid space. The last-named are characteristic of subarachnoid hæmorrhage. Both forms of hæmorrhage arise from the passage forwards of the extravasated blood into the subarachnoid space surrounding the optic nerves. A low grade papilloedema is occasionally also observed.

In non-fatal cases of both types, the temperature may remain raised for 7 or 8 days, and the urine for the first 48 hours only may contain abundant albumin and some sugar. The addition of these features to the clinical picture may, if the possibility of their occurrence is overlooked, lead to an erroneous diagnosis of uræmia or diabetes.

In small leaking hæmorrhages the cerebro-spinal fluid is more or less heavily bloodstained, and may for two or more weeks be discoloured, yellow or brownish according to the amount of blood originally present. As with all other conditions of high and rising intracranial pressure, sudden and unexpected death is a common event in undrained cases. Subarachnoid hæmorrhage does not always reach the general subarachnoid space freely. When effused upon the vertex, it may spread out and clot at its edges and from without, while the bleeding is still going on at its centre. Thus, a pancake-like hæmatoma accumulates upon the surface of the brain, thin and clotted at its edges, which prevents further spread, and still liquid and ever accumulating at its centre. Not infrequently the pressure of the growing liquid centre bursts into the hemisphere causing the addition of a sudden hemiplegia to the syndrome, and this has been named by Froin the "meningo-cerebral hæmorrhage." Draining of the cerebro-spinal fluid gives no relief when this pancake hæmatoma is present.

3. *The lumbago-sciatica syndrome.*—This rare condition, first described by Professor Arthur Hall, commences with pain and stiffness in the lumbar region, followed by pains in the legs, and sometimes the leg jerks are absent. For a week or more there may be no indication that the cause is intracranial, but thereafter in undrained cases the symptoms spread upwards to the arms and neck, and head retraction, headache and vomiting are added. Pyrexia is the rule. The diagnosis depends upon the characteristic cerebro-spinal fluid of subarachnoid hæmorrhage. The explanation of this syndrome is not easy. It may be that deposit of fibrin upon the roots of the lower theca is the cause of the quite local meningeal irritation. All the reported cases that have been treated with regular drainage by lumbar puncture have made good recovery.

4. *The recurring coma syndrome.*—The description of a typical case will best illustrate this condition. A man of 28, during a period of four years,

on four occasions, and at long intervals without any prodromal symptoms, fell unconscious in the street, and on each occasion he was taken to the nearest hospital where, on account of the persistent coma, lumbar puncture was done, with the discovery of blood in fair quantity in the cerebro-spinal fluid. On each occasion, the coma disappeared somewhat suddenly after 24 hours, and the patient insisted on leaving hospital and returning to work within a week, as he felt quite well. This patient came under my observation for the prevention of further attacks. The only abnormality found was a yellow cerebro-spinal fluid resulting from long antecedent hæmorrhage. The presence of leaking aneurysm has been pathologically proved in several similar cases.

Differential Diagnosis.—The recognition of subarachnoid hæmorrhage is an easy matter in those cases in which the train of symptoms calls at once for the examination of the cerebro-spinal fluid and blood is found in that fluid. The distinction of the apoplectic forms from other varieties of cerebral hæmorrhage can only be made: (1) by the age of the patient, practically all hæmorrhagic apoplexy being in the first half of life the result of ruptured aneurysm; and (2) by preceding symptoms, such as headache, diplopia, ophthalmoplegia and migrainous phenomena. In those cases in which blood does not escape into the cerebro-spinal fluid, as in very many of the subdural hæmorrhages, the diagnosis is both difficult and uncertain. The insidious onset of irregular headaches with periods of mental confusion and drowsiness alternating with period of recovery, especially if following a fall or slight blow on the head, should, during the second half of life, always suggest the possibility of subdural hæmorrhage. Adie has suggested that all cases of migraine with transient ophthalmoplegia are due to aneurysms. It seems certain that many of the numerous cases of sudden death occurring in ophthalmoplegic migraine have been the result of terminal hæmorrhage due to the presence of aneurysm. On the other hand, the majority of the cases of migraine with ophthalmoplegia make perfect recovery.

Prognosis.—When the aneurysm ruptures frankly and widely and the bleeding can be free, the outlook is hopeless, and death occurs in from a few minutes to a few hours; nor does drainage avert the consequences of so large an opening into a main arterial trunk. If, as so commonly happens, there is a slower leakage which perhaps is intermittent, the outlook will depend: (1) upon the cessation of the bleeding and the healing of the leak by clotting; and (2) upon the possibility of the free escape of the effused blood into the subarachnoid space and its removal by repeated lumbar drainage. In many of the cases of subarachnoid hæmorrhage, the bleeding ceases and the pressure and the dangerous results therefrom can be well relieved by lumbar drainage, repeated whenever the symptoms demand it, and healing of the aneurysm, by clotting and calcification, occurs with complete recovery. In other cases there may be repeated attacks of leaking at intervals of weeks, months or even years, and again many of such patients make good recovery in the end. When the bleeding is wholly or mainly subdural and when a subarachnoid hæmorrhage clots at its edges upon the surface of the brain, drainage and the relief of symptoms is impossible, and the prognosis is serious in the extreme but for the possibility that the site of the bleeding may be located and the clot turned out and the hæmorrhage arrested by surgical procedures.

Treatment.—In the case of subarachnoid hæmorrhage the patient must be kept absolutely at rest, with the administration of sedatives. An immediate injection of morphia is indicated when the patient is not comatose and has the usual intense headache. It also may be necessary on the recovery of consciousness on account of headache and restlessness to take this step. It is doubtful whether repeated lumbar puncture is advisable, as it may lead to recurrence of hæmorrhage, but if there be signs of raised or of rising intracranial tension (and progressive slowing of the pulse is generally a reliable indication of this), then lumbar puncture may be expedient. When recovery sets in, the patient should still be kept in bed for at least 3 or 4 weeks. During the first fortnight recurrent hæmorrhage is more likely to occur than later during the course of convalescence.

2. UNRUPTURED ANEURYSM OF THE INTERNAL CAROTID ARTERY

Synonym.—The Paralytic Syndrome.

The aneurysms of this group, like the berry aneurysm, mostly occur at the bifurcation of an artery. In this case, on the internal carotid artery where the ophthalmic artery arises, or at the junction of the anterior cerebral and anterior communicating arteries. There seems no doubt that they are more often seen in women than in men, mostly in middle life.

Symptoms.—The onset may be sudden, in which event diagnosis is relatively easy; or the symptoms may develop so gradually as to make a diagnosis of intracranial new growth probable. The arrangement of the circle of Willis and its components is such that the visual pathway (optic nerves, chiasma and tracts) are very commonly affected by the development of an aneurysm in this situation. Similarly, when there is a carotid aneurysm the proximity of the carotid artery to the cavernous sinus commonly leads to ocular palsies.

ANATOMICAL CONSIDERATIONS.—The arrangement of the circle of Willis and its relations to the visual pathway (optic nerves, chiasma and tracts) are such that aneurysms in this situation usually exert pressure upon some part of this pathway and thus give rise to visual defects. Further, the proximity of the internal carotid artery to the cavernous sinus may lead in similar manner to ocular palsies and to disturbances in the field of the trigeminal nerve. The optic tracts pass forward above the posterior communicating arteries, the chiasma lies within the arterial circle, and the optic nerves pass forward below the anterior cerebral and anterior communicating arteries. In other words, the visual pathway in its passage forward passes through the circle Willis from behind and above, and leaves it anteriorly on its under side.

It will be seen, therefore, that there are supra-clinoid and sub-clinoid aneurysms of the carotid trunk. The latter arise in the cavernous sinus, do not commonly interfere with the visual pathway, but may compress the third nerve and the ophthalmic division of the trigeminal nerve. The supra-clinoid aneurysm arise from the carotid where the ophthalmic artery goes off or at the final bifurcation of the main trunk. In these circumstances the visual pathway is generally affected.

Symptoms.—The symptoms may evolve slowly, as though a new growth were in question, or may develop with dramatic suddenness. There may be

paralysis—partial or complete—of the third, fourth and sixth nerves with squint and ptosis, severe pain, and some sensory loss round the eye in the field of the first division of the trigeminal nerve. There may be slight proptosis of the eye, and rarely only some papillœdema. The visual field defects vary from case to case. There may be monocular blindness when the optic nerve alone is compressed, or monocular blindness with a temporal hemianopia in the eye on the side away from the lesion. Other recorded defects are bitemporal and homonymous hemianopia. Pain is a prominent symptom. Symptoms of pituitary dysfunction are usually not found. X-ray examination may reveal a ring calcification of the aneurysm, while angiography may show the shadow of the aneurysm.

Treatment.—Ligature of the carotid artery appears to be the only method of treatment of an unruptured aneurysm of this type.

CHRONIC SUBDURAL HÆMATOMA

Subdural hæmatoma stands in no ætiological relationship to subarachnoid hæmorrhage, but is invariably traumatic in origin, the hæmorrhage occurring from cerebral veins as these traverse the subdural space. Yet it is convenient to consider it at this juncture.

Ætiology.—The condition under consideration was formerly known as pachymeningitis hæmorrhagica, the name expressing the belief that an inflammatory process was in question. It was further supposed that the lesion was in some unexplained way peculiar to chronic alcoholic subjects and to sufferers from general paralysis.

W. Trotter was the first to point out that in reality the lesion was traumatic in origin, and that the frequent absence of a history of injury was what might be expected in the individuals specified above, who are both more than normally liable to falls and head injuries of a minor order and less than normally capable of recalling these injuries. In fact, subdural hæmatoma is found at all ages and is invariably traumatic. Falls on forehead or occiput, not at the time apparently productive of serious injury, may yet lead to a tearing of the cortical veins as these pass from the surface of the brain to enter the dural sinuses. The tear is commonly in the subdural space on one or both sides of the vertex (superior longitudinal surface). When after the shock of the fall the blood pressure rises again to normal and on subsequent occasions when there is a transient rise in this pressure blood leaks from the torn veins and collects in an ovoid mass on each side of the vertex.

Rarely such a subdural collection may be found at the base of the brain. The periphery of the blood clots thus formed tend to organise so that a fine capsule is built up round the hæmatoma. This may remain solid or its centre may liquefy with the formation of a cyst containing a thin brownish fluid. In time the dura over this cyst and the cyst walls become thickened and stained, and it is clear from the condition sometimes found at necropsy that a subdural hæmatoma may be compatible with many years of survival and may during life give rise to no clearly recognizable symptoms. Such hæmatomas may reach a large size, and a 10-ounce mass over each hemisphere has been observed.

Symptoms and Diagnosis.—The difficulty which still frequently sur-

rounds the diagnosis of subdural hæmatoma depends in large measure upon a general unawareness that it is a not uncommon lesion, and from a survival of the old and now obsolete notion that its occurrence is largely confined to sufferers from the two affections named above. It must be emphasised again, therefore, that subdural hæmatoma may follow an apparently trivial head injury in persons at all age periods ; that essentially its symptomatology is that of a space-occupying lesion, with a feature characteristic of hæmatoma : namely, a remarkable fluctuation in the course and severity of the symptoms ; that owing to the bilateral nature of the lesion the signs are apt to be difficult of localising interpretation ; and finally that in the presence of such a somewhat blurred picture of raised intracranial tension a history of head injury some days, weeks, or even months before the onset of symptoms should always give rise to the suspicion that a hæmatoma, and not a new growth, may be present.

There is almost invariably a clinically latent period in the development of a subdural hæmatoma. This may vary from a matter of days to one of weeks. On the whole, it may be said that in young persons, the latent period is apt to be shorter and the symptoms more severe and of more rapid evolution than is commonly the case in elderly subjects. In young subjects, too, there is usually no difficulty in obtaining a history of head injury, either a fall upon the head or a blow sustained at sport or in some other way. The initial symptom is usually headache, fluctuating in intensity, apt to be most severe on awaking in the morning or on physical exertion. With the passage of days or weeks this becomes more severe and soon other symptoms are added to it. The patient has days on which he is drowsy. He may pass rapidly into stupor or even coma, emerging again to become almost normal. Transient accesses of diplopia with squint may be noted. Examination during a period of maximal symptoms may reveal a papilloedema, sometimes severe in rapidly developing cases. The plantar responses may, on one or both sides, be of the extensor type. There may be an inequality of tendon jerks on the two sides, the abdominal reflexes may be diminished on one or both sides. They may even be absent. Periods of mental confusion may also occur.

This fluctuation in the severity of the symptoms, the fugitive character of the physical signs and the generally downhill tendency of the illness, despite the fluctuations are amongst the features which are characteristic of subdural hæmatoma and help to differentiate it from that of intracranial new growth. When the syndrome develops rapidly, it is more common to meet a marked slowing of the pulse than in new growth.

In most instances there is no trace of blood in the cerebro-spinal fluid.

In all cases there is a great liability to a rapid development of coma with a fatal issue. Yet, the occasional finding at necropsy of what is clearly a subdural hæmatoma of very long standing, unsuspected during life, shows that from time to time the sequence of events briefly reviewed above fails to develop. Though it has to be confessed that even in such cases it is highly probable that careful clinical examination and an awareness on the part of the examiner of the symptom-complex of hæmatoma might have made diagnosis possible during life.

Diagnosis and Treatment.—The features which should make clinical diagnosis possible have been described, but in certain doubtful cases certainty

can be obtained only by an exploratory operation. This may consist in bilateral trephine holes and tapping of the subdural space, or in the turning down of osteoplastic flaps and the evacuation of the cyst when found. It is clear that treatment is essentially surgical.

EMBOLISM

The majority of embolisms of the cerebral arteries occur in valvular heart disease, 89 per cent. (Saveliew). Embolism may also occur from detached portions of clot from an aneurysm, from thrombi in connection with atheroma or syphilis of the aorta, and from detached clots which may form in the region of the pulmonary veins and left heart where there is no cardiac valvular disease. This latter condition is not an infrequent cause of puerperal apoplexy. It occurs in suppurative and gangrenous conditions of the lungs, and is an essential factor in the production of "pulmogenic" cerebral abscess. Embolism is rather more frequent in women on account of the greater incidence of mitral stenosis in that sex, and from the puerperal cases.

The embolus comes from the left heart and may be a vegetation from a quite recent endocarditis, but is more commonly a detached vegetation from a chronic and especially from a septic endocarditis. Very frequently it is a detached portion of clot which has formed in the left auricle in mitral stenosis. The middle cerebral arteries are the usual sites of lodgment of the emboli, and the left middle cerebral is rather more frequently affected than is the right. Embolism of the other cerebral vessels may occur, but is extremely rare.

The pathological events which may follow the plugging of a cerebral vessel with an embolus are varied and are highly important. In the first place, secondary thrombosis may proceed from the embolus throughout the whole distal distribution of the vessel, and lead to complete softening of its area of supply, and the clinical aspect will be that of severe and unchanging damage to the brain. The softened area may shrink or may undergo cyst formation, or it may be completely absorbed, giving rise to a porencephalus. Secondly, the embolus in the absence of secondary thrombosis may become adherent to one spot of the vessel wall at the site of its lodgment, and retracting from the vessel wall elsewhere, the blood channel becomes reopened, and the clinical results of the embolus, at first very severe, may disappear with unexpected and dramatic rapidity, and complete recovery ensue. Thirdly, the embolus may contract at the site of its primary lodgment, and become detached and shifted on by the blood stream to find a second resting-place in a much smaller artery. This event is manifest clinically by rapid clearing up of the physical signs in many regions, with persistence or even deepening of the involvement of one particular region. For example, a severe and complete hemiplegia clears up suddenly on the third or fourth day, leaving a brachial monoplegia only. Fourthly, an embolus may be impacted at the termination of the internal carotid artery, giving rise to severe hemiplegia with blindness of the opposite eye from blocking of the ophthalmic artery—*carotid hemiplegia*. Owing to the re-establishment of the circulation by the circle of Willis the hemiplegia is likely to recover rapidly, while

the eye remains permanently blind owing to secondary thrombosis extending through the ophthalmic artery. When an embolus is finally lodged and completely occludes the artery, the condition, both pathologically, clinically and from the point of view of treatment, is one of thrombosis, the cause of the embolism being taken into consideration. As with thrombosis, the immediate result of permanent occlusion is a condition of infarct and acute œdema in the region from which the blood supply is cut off. The acute œdema causes local pressure and increased general intracranial pressure, and is a common cause of the transient coma which often supervenes a few hours after the stroke, in both embolic and thrombotic apoplexy.

Stroke from embolism is the most suddenly occurring of all apoplexies, and the ictus is not preceded by any prodromal cerebral symptoms. Consciousness is apt to be lost at once if the whole middle cerebral artery be occluded, especially if the lesion be upon the left side. Or it may be retained throughout if the embolus lodge in a small vessel only.

Diagnosis.—This rests upon the occurrence of sudden apoplexy without prodromal symptoms in the presence of an obvious cause for embolism such as cardiac valvular disease, aortitis, pulmonary thrombosis or the puerperal state. The diagnosis can be a matter of probability only in those conditions where either embolism or thrombosis is likely, such as enfeebled cardiac states and the puerperal state.

Prognosis.—The prognosis in cerebral embolism depends upon the size of the vessel which is plugged, as deduced from the severity of the initial symptoms and their extent, and upon the immediate pathological changes which occur in the obstructed vessel as above described; and according to the nature of these changes it may be the most severe and least recoverable, or, on the other hand, the least severe and most recoverable of all forms of apoplexy. The prognosis of the condition causing the embolism is often the more important.

Treatment.—The treatment is that of cerebral thrombosis (see p. 1606) together with that of the condition giving rise to the embolus.

ARTERIAL THROMBOSIS AND HÆMORRHAGE

Cerebral thrombosis and cerebral hæmorrhage seem hitherto to have been described in text-books of medicine as quite separate conditions, almost antagonistic and mutually incompatible, between which it was highly essential and even possible to make a differential diagnosis for the purpose of applying a very dissimilar line of treatment in the respective conditions, each line of treatment being the worst possible for the other condition. It cannot, however, be too forcibly pointed out that primary arterial thrombosis and primary arterial hæmorrhage depend in every case upon degeneration of the arterial wall, and that every condition of degeneration of the arterial wall may cause either thrombosis or hæmorrhage indifferently. It is a usual experience to find in patients who have had severe strokes that thrombosis was the cause of the earlier, and hæmorrhage of the final apoplexy. Even in that condition, which has always been held to be the most important antecedent of cerebral hæmorrhage—renal disease with

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high arterial tension—Janeway has recently found that thrombosis and not hæmorrhage was the cause of apoplexy in many of his cases.

On account, therefore, of the identity of the underlying pathological condition in every case, and the clinical association of thrombosis and hæmorrhage of the cerebral arteries, and the difficulty of distinguishing them clinically, the two conditions are here described together.

Ætiology and Pathology.—The arterial degeneration which may result in cerebral thrombosis and hæmorrhage is due to the following causes: (1) Syphilis, which is the commonest cause of thrombosis in the first half of adult life, and which is less commonly the cause of hæmorrhage. It may affect both the large and the small arteries, even to the smallest. All the coats of the artery are affected, and in the case of the finest vessels there is conspicuous lymphocyte accumulation or “cuffing” round the vessel. In the neighbourhood of the affected vessels there is always syphilitic cerebritis in the form of lymphocyte exudation and oedema, and meningitis, if the lesion come to the surface. This is the most recoverable of all thrombotic lesions of the brain. (2) Atheroma, which is the common cause both of thrombosis and of hæmorrhage in the second half of adult life, and which is by far the commonest cause of hæmorrhage. It must be especially borne in mind that cerebral atheroma may be local in the cerebral vessels, and unassociated with general atheroma of the systemic vessels. (3) Arterial hypertrophy, with secondary focal degeneration of the media, with or without its commonly associated renal disease, which is of the nature of “small white kidney” in children and younger adults, and of the various types of “granular kidney” in older subjects. (4) Abnormal conditions of the blood, especially when associated with feeble cardiac action and low blood pressure, as in the puerperal state, and in septicæmic conditions, and at the time of the menopause. Hæmorrhage into the brain may also complicate polycythæmia and acute leukæmia. (5) In association with new-growths of the brain, both thrombosis and hæmorrhage are common events, especially when the neoplasm is soft and rapidly growing. The vascular lesion may occur quite early in the course of the new-growth, and apoplexy may be the first sign of its presence. (6) Inflammatory conditions of any nature may cause thrombosis and hæmorrhage. The vascular lesions are usually small, but they may be extensive, and may cause death. (7) Traumatic lesions, such as the passage of a bullet through the brain, or a blow upon the head, or concussion from high explosives, may cause extensive thrombosis or hæmorrhage.

While cerebral hæmorrhage results often enough from the direct rupture of a true aneurysm, or of one of those irregular local thinnings of the vessel wall which is called a “false aneurysm,” and may take place from an artery the wall of which is softened by disease though there be neither thinning nor bulging of the vessel wall, yet it is probable that cerebral hæmorrhage is very often the direct consequence of thrombosis, and especially of thrombosis which has occurred some time previously. The sequence of events is as follows: An area of thrombosis occurs within the brain, and the usual softening and necrosis follow. On the confines of this area, the necrosis spreads to come in contact with the wall of a living and unthrombosed artery, perhaps of considerable size. The arterial wall of this vessel was nourished by the capillaries of the necrosed area, and with its nutrient supply now

cut off there is local degeneration of the wall of the living artery. Moreover, the shrinking of the necrosed area of brain causes loss of support to the degenerate wall of the vessel, which ruptures as the result, under the influence of any sudden increase of blood-pressure. It is for this reason that one commonly finds in patients who have had multiple attacks of apoplexy, that the final and fatal attack is one of hæmorrhage, and that the preceding attacks have been attacks of thrombosis. These spots of thrombosis, which cause hæmorrhage, need not be of large size, and they may be so small as hardly to cause symptoms on their occurrence. Marie first called attention to these small spots of thrombosis as *plaques jaunes*, small yellowish-brown spots, softened and sometimes cystic, and pointed out their importance as a cause of cerebral hæmorrhage. In a similar way the thrombosis of syphilitic arterial disease may cause subsequent hæmorrhage.

Syphilitic cerebral thrombosis is not usually a pure pathological process, for the vascular disease is often accompanied by acute syphilitic encephalitis, with much lymphocyte extravasation in the vicinity of the diseased vessels, and acute local œdema, which increase the evascularisation when thrombosis occurs. The symptoms of cerebral loss of function are not all due to the thrombosis, but are in part owing to the recoverable acute inflammatory condition, and it is for this reason that syphilitic apoplexy often shows much more recovery than do other forms of apoplexy.

Thrombosis is a more common cause of apoplexy than is hæmorrhage, but it is much more frequently survived, while hæmorrhage is frequently fatal, within from a few hours to a few days of its onset. It follows therefore, that in the autopsy room of a general hospital, hæmorrhage is seen much more often than is thrombosis, while in infirmaries, where the survived cases of apoplexy collect, thrombosis is almost invariably the lesion found to be primarily responsible for the apoplexy.

Thrombosis tends to occur when an habitually high blood pressure is temporarily lowered and the circulation less active, and is always strongly suggested when apoplexy occurs during sleep and conditions of quiet, and after exhaustion, exposure to cold, severe purgation, and in debilitated states generally. It is preceded by slowing of the circulation in the area affected, and this may be productive of prodromal symptoms. Or there may be slight local thromboses preceding the main thrombosis, also giving rise to prodromal symptoms. Thrombosis may thus have an ingravescent onset, especially when clotting occurs in distal branches of an artery and extends towards the main vessel; but, on the other hand, it may have an absolutely sudden onset when the clotting occurs primarily in a large artery. The immediate effect of the thrombosis is a condition of infarct with œdema, extending widely in the vicinity, and it is this œdema which causes the loss of consciousness so commonly seen a few hours after the apoplexy has occurred. The œdema tends to pass off in a few days, and the area bereft of circulation by the thrombosis tends to become narrowed by collateral circulation from surrounding regions, and any recovery of function within the affected region must be by collateral circulation from elsewhere. The affected area at an early stage is bright red in colour, and soon becomes soft and shrunken (red softening). Later, the blood pigments degenerate with the production of bilirubin and are partly absorbed, producing a yellow-coloured lesion (yellow softening). Finally, much of the thrombosed tissue becomes necrotic and is

absorbed, leaving one or several cystic cavities. These cavities are never so sharply defined as those resulting from embolism, because of the more complete necrosis occurring with the later lesion. Still, a severe arterial thrombosis occurring at an early age may result in a porencephaly. Cavities found in cases of apoplexy after years have elapsed, are too often attributed to hæmorrhage. In reality they are nearly all due to thrombosis or embolism. The cerebro-spinal fluid in thrombosis is never found to contain blood, but some little time after the apoplexy it is often coloured yellow or yellowish-brown from escape of changed blood pigments, when the lesion has reached the surface of the convexity or the surface of the ventricle.

Hæmorrhage, which is usually described as an apoplexy of sudden onset, may be so when the escape is from a large vessel. When the bleeding commences from a smaller vessel, the symptoms are not sudden in their onset, but gather rapidly. Such a hæmorrhage is much like an avalanche. Commencing from a small vessel the hæmorrhage tears a small cavity, and in so doing opens up fresh bleeding points, and with increasing destruction more and more bleeding occurs from every piece of torn tissue, until the hæmorrhage reaches such a size as to burst commonly into the ventricle and much more rarely on to the surface. Indeed, it is difficult to conceive how a hæmorrhage into such a soft and vascular tissue as is the brain should ever stop. As a matter of fact, it very rarely does so, but causes death in the first attack of hæmorrhagic apoplexy, within from a few hours to a few days after the onset, from widespread tearing up of the nervous system and bursting into the ventricle. One of the most important clinical distinctions between apoplexy due to thrombosis and apoplexy due to hæmorrhage is that the former is often survived, and that the latter is almost invariably fatal within a short time of the onset.

Hæmorrhage may occur anywhere within the nervous system, but its common seat of commencement is in the centrum semiovale, and the vessel which bursts is one of the perforating arteries, of which the lenticulo-striate which carries the name of the "artery of hæmorrhage" is the most common. Such bleedings are often called "capsular hæmorrhages." It must be pointed out that this term capsular refers to the region outside the corpus striatum or external capsule, and not to the compact internal capsule as it converges to the crus cerebri. The cerebro-spinal fluid in cases of hæmorrhage contains blood within a very short time of the onset, and lumbar puncture often withdraws what is practically pure blood in large quantities. I have found blood present in large quantities very often within an hour of the onset.

Both thrombosis and hæmorrhage may occur in any part of the brain, while massive embolism is rare, except in the middle cerebral artery. The semioval centre, the calcarine region and the pons are the common sites of both hæmorrhage and thrombosis in that order of frequency. Hæmorrhage is rare except in these regions, while thrombosis is not uncommonly met with elsewhere.

Symptoms.—The nature of the symptoms in apoplexy will depend upon the site of the vascular lesion; and as the semioval centre or region of the middle cerebral artery is the commonest site for all the vascular lesions, hemiplegia is the common result; and this is associated with aphasia, if the lesion

is in the left hemisphere, and involves or isolates the cortex. When the calcarine artery is the site of the lesion, hemianopia results; and this is apt to be accompanied by word-blindness, if the lesion be on the left side. Pontine apoplexy involves the appearance of double hemiplegia, bilateral ataxy and bilateral loss of sensibility, with signs of involvement of cranial nerve nuclei and cranial nerves. Cerebellar apoplexies and thrombosis of the posterior inferior cerebellar artery produce acute ataxy with forced movements and vomiting.

Prodromal symptoms in the form of transient weakness of one or both limbs of one side, transient aphasia and giddiness occur in thrombosis only. An ingravescent onset occurs in thrombosis only and when the clotting occurs in the periphery of arterial distribution first and spreads towards the main trunk. When commencing in the parietal region, tingling and numbness of an extremity first occur, followed by a spread of these symptoms over half of the body, and subsequent weakness deepening into hemiplegia. When commencing in the left temporal region gradually oncoming aphasia is first noticed, and when commencing in the ascending frontal convolution a peculiar sensation of heaviness in the limbs gradually increases until hemiplegia is obvious.

The onset in embolism is always instantaneous; it may be sudden in thrombosis, and in hæmorrhage from a large vessel. In hæmorrhage it is always rapid. Consciousness is lost or not, according to the severity of the initial lesion and the site it occupies, and to the magnitude of the processes which follow the initial lesion, namely, the œdema of embolism and thrombosis and tearing of the brain tissue in hæmorrhage. In hæmorrhage, consciousness is lost soon, and the rapid development of severe symptoms which progressively deepen, is a most important early indication that this is the nature of the lesion.

In calcarine thrombosis the initial symptoms may be so slight as to pass unnoticed by the patient, whose first indication of defect may be, that he runs into objects on his blind side.

Convulsion sometimes occurs at the onset, and this nearly always indicates thrombosis, rarely embolism, and never hæmorrhage. There may be some local spasm in the region of the cranial nerves in pontine hæmorrhage, but this is not convulsion.

Conjugate deviation of the eyes is a common feature of all apoplexy. When the lesion is irritative at its onset, and not too destructive, and always when convulsion occurs at the onset, there may be active conjugate deviation, the eyes being turned away from the side of the lesion and towards the paralysed or convulsed side in hemiplegic cases, or the blind side when hemianopia is present. But this active conjugate deviation lasts but a short while and is followed by a paralytic conjugate deviation in the opposite direction, both eyes being directed away from the paralysed side and towards the side of the lesion. This variety of conjugate deviation may last for a considerable time, but usually disappears with the onset of deep coma.

The pupils are often unequal; they may be contracted, or dilated widely, and may be insensitive to light. In severe apoplexy, when as the result of the cerebral shock or when hæmorrhage or œdema have so raised the pressure as to greatly reduce the physiological activity of all the intracranial elements with the production of deep coma, the pupils are widely dilated and insensi-

tive. In pontine lesions, the pupils are often contracted to pin-point size, and this condition is of important localising significance.

In proportion to the severity of the general intracranial disturbance, respiration tends to be hurried, noisy and stertorous, and with increasing pressure to become irregular, grouped or of the Cheyne-Stokes type. The blood pressure tends to be raised and the pulse full in all conditions of apoplexy, provided the heart will respond to the requirement of an increased blood pressure in the face of an increased intracranial pressure.

Swallowing is often impossible, and the sphincters may be relaxed or retention may occur.

In the usual variety of apoplexy where the lesion is in the area of the middle cerebral artery and the local sign of the lesion is hemiplegia, it will be obvious that when the general intracranial pressure becomes severe and the coma becomes deep, the hemiplegia becomes less apparent, or masked by the universal condition of paralysis consequent upon the general intracranial condition. The physician often sees the patient for the first time when there is considerable coma, and he must determine upon which side the lesion is situated, and endeavour to have some perspective as to prognosis by determining the severity of the lesion.

The following points will serve to determine the side of the lesion when these signs are present: (1) The paralytic conjugate deviation is towards the side of the lesion. (2) The corneal reflex, when any is present, is diminished or lost on the hemiplegic side. (3) Painful stimulation will elicit less response or no response upon the hemiplegic side (hemianæsthesia). (4) The patient may respond by blinking to a feint made with the observer's hands towards the patient's eyes upon the sound side, and not on the hemiplegic side (hemianopia). (5) The limbs on the hemiplegic side when raised and allowed to fall passively, do so in a more lifeless, inert and flaccid fashion than upon the sound side. (6) And when there is any difference between the knee-jerks, abdominal reflexes and plantar reflexes, the former tend to be diminished and lost on the hemiplegic side while the plantar reflex will be of the extensor type on the hemiplegic side. It must be remembered in this connection, that a severe lesion of one cerebral hemisphere abrogates for a time at least most of the functions of the whole hemisphere, and that the hemianæsthesia and hemianopia, here referred to, do not necessarily indicate that the destructive lesion involves the visual and sensory paths. And further, that the condition of coma due to increased intracranial pressure of itself causes such signs as bilateral loss of abdominal reflexes and knee-jerks, and bilateral extensor responses in the plantar reflex.

The severity of the lesion may be judged—(1) From the depth of the coma; (2) from the degree to which the patient responds to any form of stimulation and from the general signs of nervous depression present—for example, a condition of complete bilateral flaccidity with complete loss of all reflex action and of all response to stimulation indicates a most severe lesion; and (3) from signs of failure of respiration as shown by irregular, grouped or Cheyne-Stokes breathing. It is further important to arrive at a determination if possible as to whether the condition present is stationary, deepening or showing signs of amelioration.

Vomiting is not an uncommon occurrence in the early hours of apoplexy and before coma becomes deep. Hyperpyrexia is often seen in fatal cases

before the end. It is especially common and may reach a high degree in pontine apoplexy. It may be preceded by initial depression of temperature. It is of fatal prognostic import.

HEMIPLEGIA is the commonest sequel of vascular lesions of the brain. The signs which serve to indicate its presence in the comatose subject have already been enumerated.

After cerebral thrombosis it may happen that the initial hemiplegia is completely recovered from, but unless this recovery begins early and progresses rapidly it is not likely to be complete.

The essential feature of hemiplegia is the loss of voluntary movements, but as this loss begins to pass off, certain new features make their appearance. These are muscular hypertonus, increased tendon jerks, and associated movements.

The restoration of movements follows a certain order. Deviation of the tongue and facial asymmetry clear up early; next, the leg begins to recover; and finally—and often very incompletely—the arm. The return of movements in the limbs is selective. In both upper and lower limbs, movement at the proximal joints recovers soonest and most completely. In the leg, extension and plantar flexion recover more completely than flexion and dorsiflexion. As a result, the patient can often stand when he cannot lift the foot and leg to step properly, and has instead to circumduct the limb when walking. In the arm, flexion movements recover soonest and best, while the fine skilled movements of the hand and fingers are frequently lost for ever.

The development of hypertonus, or spasticity, is as selective as the return of movements. In the leg, the extensor group becomes spastic; in the arm, the flexor group. Thus, the arm tends to take up a position of adduction, with flexion at elbow, wrist and digits. The leg is always spastic in extension, and does not go into flexion contracture, as may happen in spastic paraplegia from spinal cord lesions. The degree of hypertonus varies, and is greatest when the loss of movement is greatest.

The tendon jerks are exaggerated, and there is clonus (knee and ankle) in the affected limbs. The Babinski plantar response persists, but the abdominal reflexes, which are initially lost on the affected side, sometimes return after a period of months.

The forced immobility of shoulder and distal joints in the arm may lead to the formation of adhesions.

The so-called associated movements are involuntary changes of attitude of the paralysed limbs which accompany forceful voluntary movements, or such involuntary movements as, yawning.

CEREBELLAR APOPLEXY.—This is usually the result of thrombosis of the posterior inferior cerebellar artery, which is a branch of the vertebral artery, and the clinical picture is very unlike that of cerebral apoplexy. The patient is seized with a sudden intense vertigo which carries him to the ground, as in Ménière's disease. Incessant vomiting and forced movements follow, the forced movements rotating the patient, so that he comes to rest prone, with that side of the face corresponding with the side of the cerebellar lesion in contact with the pillow. There is intense ataxy, usually bilateral at first, and later becoming confined to the limbs and trunk on the side of the lesion. The patient is unable to lift his head, or to maintain the sitting or standing

position. When placed in such a position he positively dives to the ground when released. Nystagmus with the long slow movement to the side of the lesion, and a short fast movement in the opposite direction is conspicuous, and the skew deviation of the eyes is sometimes seen. There is much general hypotonia of limbs and trunk which soon becomes limited to the side of the lesion. Head retraction, pain and stiffness of the neck and opisthotonos may occur. When the patient's condition recovers sufficiently to allow of examination, all the signs of a unilateral cerebellar lesion will be found. Consciousness is not often lost. Since the posterior inferior cerebellar artery also supplies the lateral region of the medulla, signs indicative of disturbance of this region are usually present, and these may dominate the clinical picture rather than the cerebellar signs. Chief amongst them are analgesia and ther-manæsthesia of the face and head, due to implication of the as yet uncrossed quinto-thalamic path, and of the limbs and body upon the opposite side, due to involvement of that part of the spinothalamic tract which has crossed below this level. Between these two areas of sensory loss there is often a gap where sensibility is normal, corresponding with that part of the spinothalamic tract which is crossing obliquely at this level, and therefore is too near the middle line to be affected. Paralysis of the motor vagus is often found from involvement of the nucleus ambiguus, and, from the extension of the lesion or of consecutive œdema towards and across the middle line, sometimes causes severe dysphagia and dysarthria, and one of the great dangers of this form of apoplexy is extension of the thrombosis to that part of the medulla which contains the respiratory and other vital centres. When, however, such extension does not take place, and if the destruction of the lateral lobe is not too extensive, the most remarkable recovery may take place.

Diagnosis.—*The nature of the lesion.*—Embolism should be diagnosed in all cases where there is an obvious cardiac valvular lesion, particularly mitral stenosis, septic endocarditis, aortic disease and aneurysm. It is true that syphilitic cerebral thrombosis may occur with syphilitic aortitis, but the combination is rare, for syphilitic aortitis usually occurs at a much later age than does syphilitic cerebral thrombosis.

Further conditions of cardiac feebleness and corresponding feebleness of circulation must obviously predispose to thrombosis if arterial disease be present. Mistakes in diagnosis will, however, not often occur, and they are not of moment to the patient, for embolism, when once the embolus is lodged, is for all purposes of treatment and prognosis the same condition as is thrombosis. Thrombosis should be diagnosed in all primary apoplexies in young syphilitic subjects, for syphilitic hæmorrhage usually occurs at some time considerably subsequent to a syphilitic thrombosis. In this connection the serum reaction and the cytology and reactions of the cerebro-spinal fluid are all-important in the diagnosis.

Thrombosis should be diagnosed, notwithstanding the presence of high arterial tension or renal disease, in all cases of apoplexy without organic cardiac valvular disease, when the onset occurs during sleep or under circumstances of quiet, depletion or exhaustion, and in all cases where prodromal symptoms are marked, or where the onset of the apoplexy is gradual, and in apoplexies occurring in advanced age, for then hæmorrhage is almost unknown. All slight apoplexies and nearly all those that survive the first 10 days after the ictus, are due to thrombosis.

Puerperal apoplexy and that occurring at the time of the menopause in women are mostly due to thrombosis.

The cerebro-spinal fluid affords important indications, since hæmorrhage into the brain in most of the cases soon bursts on to the surface or into the ventricle. If blood is absent from this fluid a few hours after the ictus, thrombosis or embolism is highly probable and hæmorrhage is very unlikely. Any infarct condition coming to the surface may in the course of time cause the fluid to be blood-tinged or yellow. It is important to bear in mind that the infarct conditions of embolism and thrombosis are followed by packing of the infarcted region with polymorphs, and that these may escape from the surface in such numbers as to load the cerebro-spinal fluid with such a high polymorph pleocytosis as to suggest the presence of suppurative meningitis. Hæmorrhage is a likely cause of apoplexy occurring during exertion, especially if it occurs at a moment of severe physical strain, or at the height of passion. It is always a probable lesion in cases where a previous thrombotic apoplexy has occurred, the final event, where multiple strokes have succeeded one another, being almost invariably hæmorrhage. An apoplexy with rapid onset and with symptoms rapidly deepening, with a quick onset of deep coma, and the development of pyrexia and signs of respiratory failure, is usually due to hæmorrhage. The certain test that an apoplexy is due to hæmorrhage is the presence of blood in quantity in the cerebro-spinal space as proved by lumbar puncture. In cases of small white kidney in the young and of granular kidney before the age of 50 years, where the blood tension is very high, and where there is severe retinitis, hæmorrhage is the most likely cause of stroke.

The position and extent of the lesion.—The position of the lesion may be judged by the nature of the initial signs, whether visual, sensory, motor or aphasic, cerebellar or pontine, and later by the permanent symptoms resulting from the lesion. It must be carefully borne in mind in this connection, that a severe lesion of a cerebral hemisphere may entirely abrogate the functions of that hemisphere, initially by a process of shock and afterwards by the occurrence of œdema in the vicinity of the lesion, which may spread widely.

The extent of the lesion may be gathered by the severity or otherwise of the early symptoms and their rate of increase, and by early or immediate loss of consciousness, and by the completeness of the paralysis resulting. The more severe the extent of the lesion the sooner do grave signs of general cerebral failure appear.

Differential Diagnosis.—The diagnosis of coma due to a cerebral vascular lesion is usually made without difficulty from the history, and from the presence of unequivocal signs of local lesion of the brain. In a patient without history, and when the coma has become so deep as to remove the unilaterality of physical signs, from the severity of the general intracranial pressure, the diagnosis may be difficult from other causes of coma such as uræmia and diabetes, poisoning by opium, alcohol and its derivatives and illuminating gas, and in cases of difficulty search is to be made for the usually obvious signs of these conditions. Uræmia may present especial difficulties, for it is often associated with cerebral vascular lesion, and transient hemiplegic attacks may occur in this condition. This is true also of the crises of essential hypertension, which are described in more detail on page 1608. Absolutely

sudden death which is so often recorded in death certificates as due to apoplexy, is usually associated with a stoppage of the heart following the obliteration of one of its coronary arteries. Apoplexy never causes sudden death. There is one recorded case of death from cerebral hæmorrhage in 5 minutes, but it is rare in any apoplexy for death to occur in under 2 hours. Other conditions causing hemiplegia with coma must be taken into consideration. Epilepsy and especially hemi-epilepsy may be followed by marked unilateral paralysis (Todd's paralysis), which may last for a considerable time. Here the history of recurring attacks and the complete recovery will easily prevent confusion.

Cerebral malaria and sunstroke may closely resemble apoplexy, and should always come to mind when rapid coma follows the development of cerebral symptoms in circumstances where these causes are likely.

The congestive attacks of general paralysis of the insane are peculiarly difficult to diagnose from apoplexy. Perhaps they are due to suddenly occurring acute cerebral local oedema. They are liable to mistaken diagnosis, of course, only when occurring as the initial manifestation of the disease. These attacks take the form of rapidly occurring attacks of hemiplegia, aphasia, hemianopia, hemianæsthesia or of some combination of these conditions, usually associated with initial convulsions and followed by coma. The diagnosis of a syphilitic thrombosis is made with reason on the positive serum reactions, and cerebro-spinal fluid examination. If energetically treated it recovers with marvellous rapidity and completeness, to slowly develop the characteristic signs of general paralysis. It is the too rapid recovery in a case of apparent syphilitic thrombosis which should suggest the possibility of the stroke being a congestive attack in general paralysis of the insane. In all cases of coma without history, especially when there are signs of local cerebral involvement, a very careful examination of the head should be made for traces of recent injury, and if signs of injury be found, the skull and meninges should be opened, and the nature of the lesion sought out and dealt with surgically.

Prognosis.—A majority of the cases of apoplexy from syphilitic thrombosis make a fair recovery, which obviously depends upon how much permanent thrombosis occurs in the lesion of acute syphilitic encephalitis which is responsible for this condition, and upon the early application of appropriate treatment for syphilis. In some of these cases even, no recovery occurs.

In embolism the course and prognosis depend upon the extent of the vascular supply cut off when the embolus comes finally to rest; and upon the amount of collateral circulation afforded, and upon the cardiac condition.

In thrombosis due to atheroma the apoplexy may be rapidly fatal from extension of the thrombosis and secondary oedema, which raise the intracranial pressure beyond the limits of survival. In cases which survive, considerable recovery may occur in proportion to the extent of the lesion, but in these subjects an apoplexy is usually the beginning of the end, since the underlying pathological causes, arterial disease and failing cardiac action, still exist and are not amenable to any radical treatment. It is astonishing, however, how many of the cases of apoplexy due to atheromatous thrombosis survive for years without any recurrence of the thrombosis or occurrence of hæmorrhage. In cases of hæmorrhage, the immediate prognosis

is the gravest possible, the great majority of the cases surviving but a few hours.

Treatment.—When arterial disease is known to be present, the only measure which can in any way tend to safeguard the patient from apoplexy is moderation in all things: in diet, alcohol, mental and physical exercises, and above all moderation in all measures tending to lower the blood pressure, for hæmorrhage is due not so much to the immediate high blood pressure as it is to an antecedent period of low blood pressure in a high tension subject, which has allowed of thrombosis and which, when the tension is high, at some subsequent period causes rupture of a vessel in the thrombosed area. It is highly probable that no treatment influences the course and fatal issue of apoplexy due to hæmorrhage. Thrombosis and embolism, however, allow some scope for treatment, which should be the same in the two conditions; and as I have argued above that medical treatment in cases of hæmorrhage is useless and cannot avert the fatal result, I advise one line of treatment to be taken in all cases of apoplexy.

From the onset of symptoms in every case, a careful stimulant line of treatment must be adopted, and all depletive measures that may be calculated to lower the blood pressure and diminish the force of the cardiac action should be scrupulously avoided. It has been pointed out in the preceding passages how much the local and general symptoms of apoplexy are the result of cerebral ischæmia produced by the raised intracranial pressure, either from hæmorrhage or from œdema, and how nature attempts to combat this ischæmia by a reflex raising of the blood pressure, to keep the cerebral circulation going, and that how when the intracranial pressure exceeds that of the mean intracranial venous pressure, death must at once result from stoppage of the cerebral circulation. As Thomas truly emphasises, "How can the lowering of arterial blood pressure possibly help such conditions?" Absolute rest is, in the first place, essential when prodromal symptoms appear, and at the onset of an attack diffusible stimulants in the form of alcohol and liquid food; the heart's action may be improved by strychnine, while restlessness may be combated with bromides. If the patient is conscious, he should make as little effort as possible. His head and shoulders should be raised, special care being taken that the neck is not bent, and that nothing shall interfere with the return of blood from the head. If there is unconsciousness with stertor, the head and shoulders should be turned upon one side, so that the tongue should not fall back and impede respiration. If there be much cyanosis from impeded respiration, as is often seen in plethoric subjects, it is advisable to withdraw blood by venesection, for such relief of embarrassment acts as a stimulant to the circulation. Purgation should be avoided, and the bowel relieved at intervals by enemata. Stimulating food in a liquid form should be administered with stimulants at regular intervals; and if there is any difficulty in swallowing, the food should be administered with the nasal tube. The bladder should be carefully watched from the first, lest retention should occur, and the catheter passed when necessary. Lumbar puncture should, when necessary, be performed for diagnostic purposes, and it frequently gives relief from symptoms due to the high intracranial pressure. I have many times seen consciousness return within a few minutes of lumbar puncture, when much fluid can be withdrawn. It is advisable to withdraw all the fluid which will run out at a rate above the normal. Bed-sores and

hypostatic bronchitis must be avoided by the usual measures. In the cases that survive the first few days, passive movements should be used daily to all the joints of the affected side in hemiplegic cases, for this will obviate the painful rest adhesions which form in the joints of the paralysed limbs, and especially in the shoulder joint, and subsequently cause so much pain and misery to the patient. With the return of the power of voluntary movement, active exercises take the most important place in treatment. The final state of hand-and-finger movements depends not alone on the severity of the damage done to the brain, but in part upon the thought given to devising active exercises for it and the assiduity with which the patient can be persuaded to employ them. To avoid fatigue it is best to ordain a given daily period of some minutes to systematic exercise. A rubber sponge of appropriate size, fixed in the palm by a strip of webbing passing round the hand, will limit the passive flexion of the fingers, and will provide a resilient resistance against which the patient may move his paretic digits. Massage is an adjuvant, but never a substitute, for active exercises in the patient who can undertake them. Electrical stimulation of the muscles is absolutely contra-indicated. It has no other effect than to aggravate the spasticity that is so serious a hindrance to free movement. A hemiplegic patient after apoplexy, should be got upon his legs and encouraged to make attempts to walk as early as ever the returning power allows any possibility of the attempt.

HYPERTENSIVE ENCEPHALOPATHY

In the preceding section on the differential diagnosis of apoplexy, mention was made of the sudden and transient cerebral symptoms associated with essential hypertension, and some further reference to them is necessary. It is known that the subjects of this variety of hypertension may ultimately succumb to cerebral hæmorrhage, but it should also be borne in mind that they are subject from time to time to what are known as "hypertensive crises." The patient is the possessor of a persistently high blood pressure. The attack is precipitated by a further rise in this, and develops with intense headache, sickness and sometimes drowsiness or even semi-coma. Examination will reveal the presence of hypertensive retinitis in most cases, but in a proportion there is a definite papilloedema with retinal hæmorrhages and exudate. Accompanying these symptoms there may be hemiparesis, hemianopia, focal or generalised fits, or other indications of local cerebral lesion. The crisis is brief, lasting from a few hours to several days, and usually ends in recovery, but recurrence is likely, and finally many subjects develop cerebral atheroma and succumb to cerebral hæmorrhage. Intervals of several months may intervene between succeeding crises.

The presence of papilloedema is taken to indicate that cerebral oedema is complicating the situation. The transient nature of the crisis, and particularly the rapid appearance and disappearance of such symptoms as hemiparesis, exclude the possibility of arterial thrombosis or other material lesion of the kind, and spasm of the arteries has been invoked to account for the symptoms. There is of course no conclusive evidence that this occurs. Yet while the cerebral arteries are not under the same measure of

vasomotor control as arteries elsewhere in the body, it is known that some such control exists, and it may be that in arterial hypertension more intense spasm is possible than in healthy arteries. At least, it may be said that no hypothesis better founded or more in harmony with the facts of clinical observation has been formulated.

Differential Diagnosis.—As has been indicated, the transitory character of the symptoms exclude gross vascular lesions such as thrombosis, and the same may be said of intracranial tumour and lead encephalopathy. Yet it may be admitted that while it is present the hypertensive crisis shows many points of resemblance to the last two named conditions, especially when papilloedema is found. Plumbism in children and young persons not uncommonly develops with headache, vomiting, convulsions and focal signs, and the development of an intense papilloedema, sometimes also with high blood pressure and albuminuria, and search for other indications of lead poisoning and careful history-taking are necessary to exclude this condition. In intracranial tumour, the systolic blood pressure is rarely above normal limits, the history is longer and the condition progressive. Uræmia can usually be excluded, since in essential hypertension the blood urea is within normal limits, and the only abnormality in the urine may be a trace of albumin.

Treatment.—Venesection is indicated as the first step, and when there is papilloedema or other signs of cerebral oedema (convulsions, high cerebrospinal fluid pressure) lumbar puncture and the withdrawal of cerebrospinal fluid, and also the intravenous or intramuscular administration of hypertonic solutions are necessary. As a measure of urgency from 50 to 70 c.cm. of a 50 per cent. solution of dextrose may be given intravenously. For less urgent cases and as a measure that can be repeated for the relief of headache, six ounces of a 20 per cent. solution of magnesium sulphate may be given per rectum at 6 hourly or less frequent intervals. The convulsions may be treated by rectal administration of paraldehyde (240 to 360 minims in water), or by the hypodermic injection of 3 grains of soluble phenobarbitone in solution.

The subsequent management of the case is that of the underlying essential hypertension.

SINUS THROMBOSIS

Thrombosis of the cerebral sinuses may occur rarely as a primary condition, or it may be secondary to infective processes spreading to the sinuses from contiguous infected regions.

Ætiology.—Primary thrombosis is a rare condition. It is said to affect the superior longitudinal sinus most commonly. It is more common in the first year of life than at any other period, when it may follow diarrhoea, bronchitis or the conditions of exhaustion met with in tuberculous disease, and in congenital syphilis, and it may follow acute diseases such as measles diphtheria, etc. It may also occur at any age, up to advanced old age, in the terminal stages of cancer, phthisis and other chronic diseases.

The essential cause of secondary thrombosis is the advent of micro-organisms to the sinuses. The infection is often a mixed one, but the common organisms present are streptococcus, pneumococcus and *Bacillus*

coli. The sinus may become infected as a part of a general pyæmia, or infection may spread directly through its wall from a focus of local disease, most commonly from an extradural abscess. In most cases, however, the sinus becomes infected from a local spreading septic thrombosis of the veins which open into the sinus, from an infected spot at a distance. Thrombosis of sinuses may also occur from injury, as by bullet wounds and fractures of the skull, and may also result from surgical procedures in the region of the sinuses.

Pathology.—The affected sinus is bulged and distended, and feels to the touch, as if it were injected with a solid mass. In the infective forms, the clot may very quickly break down into pus, and general pyæmia result. When the superior longitudinal sinus is thrombosed, there is marked congestion of the convolutions of the convexity of the brain, often with cord-like clot-distended veins. There is bloody serum in the sub-arachnoid space contaminating the cerebro-spinal fluid withdrawn by lumbar puncture. Later, there is extensive bilateral softening of the cerebral hemispheres, most marked in the paracentral and surrounding convolutions. The cavernous and lateral sinuses do not drain the brain directly, and blocking of one of them does not cause so much cerebral disturbance, on account of the presence of alternative paths for the blood. Thrombosis of the cavernous sinus, however, may extend to the ophthalmic veins and cause blindness with an anæmic and infarcted condition of the retina. The nerves which lie in its outer wall, namely, the third, the fourth, the ophthalmic division of the fifth and the sixth nerves, may be paralysed.

Symptoms.—The clinical aspect of this condition is made up of three groups of symptoms—(1) the general signs of some bodily condition likely to be associated with thrombosis, such as marasmus, pyæmia, local cranial injury or septic disease of cranial bones and neighbouring tissues; (2) general signs of intracranial disturbance, which will depend upon how much the cerebral circulation is upset by the blocking, and which will be severe in cases where the superior longitudinal or the straight sinus is affected, and perhaps altogether absent where the cavernous sinus or the lateral sinus is affected; and (3) local signs of blocking of an individual sinus.

The general signs depend upon congestion, cedema, meningeal exudation and increased intracranial pressure. Headache, drowsiness, deepening into coma, and vomiting are common, while delirium and convulsions may occur. Papilloedema is not infrequent, while head retraction and rigidity of the neck, trismus, strabismus, inequality of the pupils, nystagmus, and irregularity of pulse and respiration may occur. In infective thrombosis, high pyrexia and rigors are the rule.

LOCAL SIGNS.—*Superior longitudinal sinus.*—The general signs are severe and convulsion is common, and bilateral hemiparesis or paralysis is likely to develop. There may be cyanosis and cedema of the forehead. The angular parietal and temporal veins may be distended, and in rare cases thrombosed.

Lateral sinus.—The clot may extend into the jugular vein and cause pain and stiffness on that side of the neck, and occasionally the thrombosed jugular vein may be felt beneath the anterior border of the sterno-mastoid as a tender solid cord. There may be tenderness and swelling over the region of the mastoid emissary vein.

Cavernous sinus.—There is œdema of the orbit, with proptosis and œdema of the conjunctiva, forehead and face. Amblyopia or blindness is the rule. Ophthalmoscopic examination reveals swelling of the disk with multiple hæmorrhages. Paralysis of the ocular muscles and anæsthesia of the eye on the same side may also occur.

Diagnosis.—If local signs, which give conclusive external evidence of sinus thrombosis, are absent, it may be very difficult to distinguish this condition from meningitis, abscess, encephalitis or other intracranial lesions. The septic forms of meningitis should be distinguished by the polymorpho-nuclear leucocytosis in the cerebro-spinal fluid. It must be remembered, that in the primary forms of sinus thrombosis in children, a copious lymphocytosis is met with, which may cause confusion with tuberculous meningitis. Abscess and sinus thrombosis often exist together.

Prognosis.—This disease is, as a rule, rapidly fatal from ever-increasing intracranial pressure; but some subjects, both in the non-infective and in the infective forms, survive. This is especially the case when the thrombosis is confined to one cavernous sinus. Cases of infective thrombosis of the lateral sinus following middle-ear disease have often been saved by timely surgical interference with ligature of the jugular vein and of the lateral sinus on either side of the thrombosed area, and with incision and turning out of the clot.

Treatment.—Beyond vigorous prophylactic measures against the causes of this condition and the palliative treatment of symptoms, surgical measures in cases of local infective origin alone are of avail. Further, in dealing with injuries of the skull in the region of the superior longitudinal sinus, trephining and exploration should be undertaken with a clear understanding of possible thrombosis of the sinus, and its appalling results.

APHASIA AND OTHER DEFECTS OF SPEECH

APHASIA

GENERAL CONSIDERATIONS.—The function of speech, which is the highest and most recently evolved human function has as its anatomical substratum a region of the cerebral convolutions situated in the left hemisphere and having its centre a little behind the middle of the first and second temporal convolutions. It is limited above by the posterior limb of the Sylvian fissure, occupies probably the tip and the whole external convexity of the left temporal lobe, and spreads backwards into the supramarginal and angular gyri, while it extends forwards over all the convolutions of the insula and possibly to the posterior ends of the second and third frontal gyri of the left side.

This "speech region of the brain" comprises not only the cortex but also the subcortical white matter which carries the paths of communication between the speech region and other parts of the brain. Posteriorly it receives an important white tract from the visual region of the cortex. An interruption of this tract results in the condition known as "pure word-blindness," or inability to appreciate written speech. Upon its deep aspect

the speech region of the convolutions receives the temporal projection of fibres conveying the auditory impressions, and destruction of this system by a lesion undercutting the convolutions in the centre of the temporal lobe produces "word-deafness," or inability to appreciate spoken language. In this same region another set of afferents impinges upon the speech area which convey the muscular sense impressions and other sensory impressions which are produced in the movements of articulation and which are the only guidance which the "deaf mute" has in the knowledge of correct execution in his articulation.

A lesion deep in the temporal lobe which interrupts both the foregoing paths, isolates the speech region from any appreciation of correct execution, with the result that spoken language becomes unshapen and degenerates into a voluble jargon, "jargon aphasia."

In the anterior half of the speech area a tract of white fibres gathers by degrees, and passing forward constitutes the bulk of the "temporal isthmus," which joins the temporal lappet to the insula, and runs beneath the insula to the region beneath the first and second frontal convolutions, from whence it is connected with the pyramidal path of the left side, and by way of the corpus callosum with the pyramidal path of the right side. This is the executive outgoing path for speech movements and a complete lesion of this path, as by a limited subcortical lesion underlying the posterior end of the left third pre-frontal gyrus and anterior part of the insula, will result in complete inability to exteriorise either spoken or written speech—"pure aphasia" and "pure agraphia."

In the speech area of the brain thus limited, little or nothing is known of any localisation of function. It is generally held that there is a gradual passing over from receptive function (appreciation of spoken and written language) in the posterior regions, to executive function (exteriorisation of spoken and written language) in the anterior regions.

Inasmuch as the phenomena of "word-blindness" and "word-deafness," as well as executive "aphasia" and "agraphia" result from lesion of the speech area, these seem to result from lesion of the tracts concerned and not from interference with the function of the cortex. These phenomena are of common occurrence in connection with lesions of the speech region and led to the formulation of localised areas of the cerebral cortex with specific functions in regard to speech. Thus, Broca's centre in the cortex of the posterior part of the left third prefrontal convolution was the motor centre for spoken language, while Exner's centre in a similar position in the second left prefrontal gyrus was the motor centre for written language. The "auditory word-centre" in which auditory memories of words were stored was in the cortex of the first and second temporal gyri, and the "visual word-centre" in which visual memories for words were impressed was in the cortex of the angular gyrus. These various centres were connected together by to-and-fro paths which could be separately affected by a lesion, and the attempt was made to explain the multitudinous and varied phenomena which occur in lesions of the speech region by damage to one or other of these hypothetical word-centres or to their connecting paths. The result was highly unsatisfactory, for the cases generally refused to correspond with the theories clinically, and practically never corresponded pathologically. Dejerine, by his discovery that a subcortical lesion in the right place could produce "pure

aphasia and agraphia " at the front end of the speech area and " pure word-blindness " at the posterior end, the cortex being intact, went far to make untenable the theories of narrow localisation of function within the speech centre. Subsequently the work of Pierre Marie, Head and others has placed modern conceptions of speech function upon a less artificial basis.

The speech function seems to be concerned with the left hemisphere of the brain alone in right-handed persons, and this is explained by the major potential of the left hemisphere for receptivity and education associated with the major use of the right hand through the countless ages of humanity. Left-handedness is usually associated with a transfer of the speech function to the right hemisphere, but there are exceptions to this rule.

The possibility of the transference of the speech function from the left to the right hemisphere is great during childhood, to the extent that no lesion of the speech region of the left hemisphere, however extensive, causes lasting loss of speech in a child under the age of six years, provided sufficient intelligence remain. After this age the possibility of such compensation by the right hemisphere for lesions in the left hemisphere seems gradually to diminish and to occur but little after adult life is reached, but even in adult life remarkable exceptions to this rule are seen.

The descending paths from the brain by which speech is executed are the pyramidal paths. The speech area of the cortex seems to command both right and left pyramidal systems equally, so that no lesion of one pyramidal system is ever productive of speech defects, either *asphasic* or *dysarthric*.

From this it follows that aphasia, word-blindness, word-deafness, amnesia, etc., only result from lesions of the convolutions and of the white matter closely underlying the convolutions, and never from lesions of the deeper parts of the corona radiata and capsules.

When, however, both pyramidal systems are involved, as, for example, by bilateral lesions of the brain, or by lesions of the brain stem which involve both pyramidal systems where these are contiguous, or by *neuronic degeneration* of the pyramidal systems in general, then defects of articulation arise comparable to the spastic paralysis of hemiplegia. These do not concern the pattern of speech, but solely the articulation, which becomes slow, clumsy, and slurring and indistinct, from weakness of movement, stiffness of the muscles concerned, and inability finely to adjust the stop positions at which the consonants are made. This condition, which is known as " *spastic dysarthria*," is commonly met with in double hemiplegia, in lesions of the brain stem involving both pyramidal tracts, and also in the tonic form of progressive muscular atrophy. In older writings it is often referred to under the most inappropriate name of " *pseudo-bulbar paralysis*."

When the lower motor neurons subserving the speech mechanism are bilaterally affected, a very similar dysarthria results, from the weakness and inaccuracy of the movements thus entailed, which is known as " *flaccid or atrophic dysarthria*," and which is met with in lesions of the medulla oblongata of all kinds, in progressive muscular atrophy, and in peripheral neuritis.

LESIONS RESPONSIBLE FOR APHASIA.—By far the most common cause of aphasia, in all its degrees and varieties, is vascular disease, usually *thrombosis*, less commonly *embolism*, and only in the rarest cases *hæmorrhage*—

for the reason that the two former lesions often affect the vascular supply of the superficial parts of the hemisphere, whereas hæmorrhage is generally situated deeply; moreover, cases of cerebral hæmorrhage rarely survive the occurrence for more than a few hours. Cerebral tumour is the usual lesion causing aphasia of gradual onset, and is perhaps the only known cause of isolated "word-deafness" and of "vulgar jargon aphasia," for it is the only conceivable lesion which can undercut and, therefore, isolate the temporal convolutions without otherwise interfering with their function.

PHYSIOLOGICAL CONSIDERATIONS.—Within a short time after birth the child begins to recognise the nature and uses of some of the objects in the world around it, and to express its simple conscious process by gestures, and it early appreciates the "gesture language" of those around it. The "mimesis," or gesture language, thus early impressed and expressed, remains throughout life the most stable, the least vulnerable, and the longest lasting of the methods of receiving and communicating ideas. Long before it is able to utter any articulate sound, the infant learns to connect certain sounds which it hears with certain objects and with certain events, and the memories of these auditory patterns first implanted serve by far the most important function in the processes and expressions of thought throughout life. Whereas we rely upon our visual memories for our remembrance and intelligence in general matters almost exclusively, yet as regards speech we rely upon auditory memories to a very large extent, and of course those who have never learned to read do so exclusively. The process of recall, both in silent thought and in speaking, is the revival of auditory patterns. We are, therefore, strong "visuals" as regards general memory, but strong "auditives" as regards speech memory, and the relative strength of the two functions varies somewhat in individuals, according to personal idiosyncrasy and to education, and this individual variation is sometimes apparent in the phenomena of aphasia. From the original connection with hearing, the memories of speech patterns come to be located in that part of the brain associated with the auditory function—in and around the temporal lobe. Later, guided by the auditory memories, the child begins to express himself in articulate speech and he does so by the revival of auditory memories.

All living motion is sensory-originated, sense-guided and sense-governed, and has a motor process of itself has no proved conscious concomitant. Our consciousness is that of the sensations which accompany the movement, or which result from the movement. The knowledge of correct execution so gained fortifies and increases the functional stability of the speech area, and is of immense importance in the speech function. If it be absent owing to a lesion isolating the speech area on the incoming side, speech degenerates into a jargon and soon becomes impossible; just as in tabes the walking becomes irregular from loss of the muscular sense conveyed in the posterior columns, and ultimately standing becomes impossible.

When at a considerably later age the child learns to read and to write, certain visual patterns (letters, words, sentences) become connected with certain objects and ideas, and become linked on to the already well-established auditory memories of speech. The meaning of the visual symbols is learned by the child from the meaning of the word or pattern spoken, which he already knows well, and the already developed auditory speech

function serves as the instructor of the visual speech function, and throughout life remains the more potent, more dominant and less vulnerable function of the two.

Later still, in learning to write, the child relies upon his visual memories, and as his knowledge of correct execution in writing is largely visual and only in minor degree common sensory from the movements of the hand in writing. It follows that the function of exteriorising speech by writing becomes intimately connected with and a part of the visual speech function, and is usually depressed or lost with the visual speech function as the result of disease. It will thus be seen that there are not separate regions of the speech area in which the auditory memories of language and the execution of spoken speech on the one hand, and the visual memories of language and the execution of written language on the other hand, are represented, but that there are four functions intimately coupled in pairs, which have their seat in the same anatomical substratum.

It is a general principle that when the speech area is damaged the speech function becomes depressed as a whole, with the result that function is lost in order of its depth of impression.

Symptoms.—Small lesions of the convolutions seem to produce no defects at all, and this is perhaps true of all the regions of the cortex of the brain. There can be, therefore, no narrow localisation of function, and there must be capacity for compensation for such small lesions in the surrounding undamaged cortex. With larger lesions of the cortex, and in proportion to their extent, mutilation of the patterns of speech, slowness of utterance, inability to find the words (inability to recall), especially nominals, and above all isolated nominals, and finally confusion of speech intelligence occur, in that order.

In the mutilated speech of the aphasic may be sometimes noticed stammering. This condition is at once distinguishable from true jargon aphasia, since the former is slow and halting whereas the latter is facile and voluble. Misplacement of words and the use of wrong words is common and is called "paraphasia." A tendency to repeat a word once pronounced is sometimes present and bears the name "echolalia." The same faults occur also in writing, as faulty spelling, misplacement of letters and words, wrong words, "paragraphia" and "echographia." Much defect of general intelligence always accompanies severe damage to the speech area, and this will be readily understood from the very large rôle which speech patterns play in the working of thought. Difficulty in the recall of words and speech patterns, which has been termed "verbal amnesia" or "nominal deficiency," is a characteristic feature of lesion of the speech area. This difficulty is greatest with spontaneous revival than with recall, which is "kicked up" by direct sensory stimulation. For example, an aphasic person who is unable spontaneously to utter a word, may repeat the word at once when it is spoken to him, when he sees it in writing, or when the corresponding object is shown to him. It is important in this connection to bear in mind that we do not speak in the letters of the alphabet, nor in the words of our dictionary, but in a running pattern of sound. The pattern or context provides the meaning, while the individual words are negligible and have no meaning. The power of the pattern in aiding revival is very great both from sequence rhythm and musical quality. As examples, an aphasic who

has no utterance spontaneously is told to count with his interlocutor. The interlocutor begins counting, the aphasic joins in. The interlocutor then stops, but the aphasic continues counting, carried by the sequence rhythm.

The confusional defects of speech function are met with in extensive damage to the speech area, and are usual as immediate and transient phenomena in all suddenly occurring lesions of the speech area. There is general mental dullness, with varying degrees, usually severe, of depression of speech function, and much confusion, both on the acceptive and expressive side, when any of these functions remain, and the results of the examination of the speech faculty are apt to vary very much from moment to moment, for attention is very difficult to hold and the patient is easily fatigued and bored. Severe degrees of this form of defect may be associated with inability to recognise objects—"object-blindness," and with loss of ability to convey ideas by gesture—"amimia."

Prognosis.—In attempting to estimate the degree of recovery which is likely to occur in cases of aphasia, it is necessary first to bear in mind that sudden cerebral injury is apt at first, by the process which has here been described as functional depression or "diaschisis," to cause very wide loss of function, though the lesion may not be very extensive. A total aphasia, for example, is often the immediate result of a lesion of moderate size. Such phenomena last usually not longer than a week, and until they have passed off it is impossible to make a definite statement, either as to the extent of the lesion or the likely degree of recovery. Speech may be regained by two entirely separate processes—either by recovery of function in partly damaged and functionally depressed areas, or by compensatory activity in the potential speech area of the undamaged hemisphere. The possible recovery of function will depend upon the nature of the lesion and upon its extent. It will be greater when a lesion may be judged to be one of pressure rather than of actual destruction, if such pressure be removable, as in subdural hæmorrhage, abscess and gumma, and least when widely spread arterial disease and a failing heart suggest that the lesion is a thrombosis, and when an irremovable tumour is present. The greater the extent of the lesion if it be presumably from vascular occlusion, as judged by the associated signs, paralysis, anæsthesia and hemianopia, the less is the chance for functional restitution, as there is then little hope of any useful restoration of the circulation through collateral vessels. In children under the age of six years, unilateral lesions produce no permanent speech defects, provided sufficient intelligence remains, but even to this rule some important striking exceptions have been recorded. When adult life is reached, transference seems to occur but little, yet in a few recorded instances destruction of the posterior half of the speech area has been followed by an almost complete restoration of speech function.

Treatment.—A careful and patient system of re-education in speech, such as is used in teaching mentally deficient children, is often of great value in all forms of speech defect. From the amount of labour that the teacher has to expend for very little progress made, this treatment is not often given a fair trial. A fair degree of intelligence must be present, and care must be taken that the lessons are not prolonged to the production of the boredom, with accompanying inattention and confusion, which occurs so readily in aphasic patients. The utterance of a simple vowel sound should

first be taught, then that of the several vowel sounds, and afterwards that of consonants and their combinations, and the patient should be directed while learning to watch the movements of the lips, etc., of the teacher. The simultaneous presentation of an object with its spoken and written name is often helpful in stimulating the remains of speech function into activity. An intelligent patient soon comes to recognise under such tuition that he has no paralysis of the articulatory mechanism.

TESTAMENTARY CAPACITY.—No rule can be laid down as to the capacity of a person suffering from aphasic speech defects to exercise civil rights and to make a will, and each case must be judged upon its own merits. The first and all-important consideration is the degree of intelligence, and when this is good it is essential for such capacity that there should be some mode of cognition and of expression left. Pure word-blindness and the extremely rare condition of pure word-deafness do not interfere with the exercise of civil rights, for the patient can understand what he hears in the first case, and what he reads in the second, and in both conditions can express himself both in speech and writing. In cases of pure aphasia and pure agraphia there is complete civil capacity, but when, as usually happens, the two conditions co-exist, though intelligence and the receptive side of speech may be but little impaired, yet the expressive side of speech is reduced to gesture, and extreme difficulty may be met in ascertaining the patient's wishes. Auditory amnesia, and combined auditory and visual amnesia, and confusional defects, except in the slightest forms, interfere seriously with testamentary capacity and with capacity for exercising civil rights. In such cases there is great loss both on the acceptive and on the expressive sides of speech, with confusion of memory and impairment of intelligence. Most satisfactory results have, however, many times been brought about in apparently hopeless cases by careful, sympathetic and repeated procedures, in which the properties to be bequeathed and the likely legatees are assembled before the patient, thus allowing the testator to match the gift with the recipient. The proceedings should be conducted in the presence and under the direction of a physician thoroughly conversant with the subject of aphasia. All concerned should bear two points in mind, the one being that the wishes of the legator must be paramount, and the other that an obviously just will is most difficult to upset in a court of law.

THE METHOD OF EXAMINATION of patients suffering with speech defects should be in accordance with some definite scheme so drawn up as to test each function of the complex physiological process of speech.

The following scheme is convenient: (1) Is the patient right- or left-handed, and, if the latter, did he write with the right hand? (2) What was the state of education as regards reading, writing and foreign tongues? (3) Does he understand the nature and uses of objects, and can he understand pantomime and gesture, or express his wants thereby? (4) Is he deaf? If so, to what extent, and on one or both sides? (5) Can he recognise ordinary sounds and noises? (6) Can he comprehend language spoken? If so, does he at once attempt to answer a question? (7) Is spontaneous speech good? If not, to what extent and in what manner is it impaired? Does he make use of wrong words, recurring utterances, or jargon? (8) Can he repeat words uttered in his hearing? (9) Is the sight good or bad, is there hemianopia, or papillœdema? (10) Does he recognise written or printed

speech and obey a written command? If not, does he recognise single words, letters, or numerals? (11) Can he write spontaneously? What mistakes occur in writing? Is there paraphasia? Can he read his own writing some time after he has written it? (12) Can he copy written words, or from print into writing? Can he write numerals or perform simple mathematical calculations? (13) Can he read aloud? (14) Can he name at sight words, letters, numerals and common objects? (15) Can he write from dictation? (16) Can he match an object with its name, spoken or written, when a series of objects and names are simultaneously presented? (17) Any other tests, emotional, rhythmical, or musical, which may raise the physiological level of the speech centres. (18) Any other means of proving in what way he can receive and express ideas.

OTHER DEFECTS OF SPEECH

1. STAMMERING OR STUTTERING.—A spasmodic defect of articulation leading to a sudden check in the utterance or words, or to a rapid repetition of the consonantal sounds in connection with which the difficulty arises. To the trouble with articulation are often added spasmodic movements of the face and head, or indeed of any part of the anatomy.

Except in the rarest instances this condition is not associated with any structural changes in the nervous system nor in the organs of articulation, but it has been observed as the end-result of a lesion of the speech area. The disorder seems to consist in a lowering of the functional stability of the executive speech mechanism by a physiological embarrassment in consciousness. It is begotten of shyness and self-consciousness. It is never congenital, nor met with in early infancy, but arises at the age when shyness and self-consciousness trouble youth most. It is infinitely commoner in boys than in girls, for the latter are much less liable to self-consciousness. The stammerer never stammers in the speech of thought, nor when talking aloud to himself alone, nor at any time when singing, for in the two former cases the embarrassment of self-consciousness is absent, and in the last case the element of rhythm and music greatly increases the stability and confidence of the function. It not uncommonly appears in conditions of debility, and especially after measles and diphtheria. It has a frequent origin in sudden fright. It is the historical utterance of fright and of those who find themselves suddenly "*in flagrante delicto*." In rebellious cases the element of self-consciousness seems to disappear, while the stammer persists as an ineradicable habit, and it is remarkable how many sufferers of this class have strong aspirations towards public speaking.

In articulate speech three muscular mechanisms are concerned—(1) the respiratory mechanism for supplying the blast of air, (2) the larynx for producing the voice, and (3) the muscles of the lips, tongue, jaw, and palate for articulation. For distinct speech there must be absolute co-ordination of these mechanisms one with another. Consonants are in nearly all cases the source of the difficulty in stammering, and while these are all buccal sounds, yet some begin with a laryngeal sound, while others are purely buccal. The former are termed "*voiced consonants*," and are B, W, V, Zh, Z, Th (as in "*thus*"), D, L, R, G, Y; and the latter "*voiceless consonants*,"

and are P, F, Th (as in "thin"), S, Sh, T, K; while N, M, and Ng terminal, are "voiced nasal resonants." If one articulates these consonants it becomes at once clear that it is the presence of the initial laryngeal element or "voicing" which makes the difference between B, V, Z, D, G, and P, F, S, T, K, respectively.

A careful attention to the manner in which the letter sounds are produced is absolutely essential in the investigation and treatment of stammering. The difficulty occurs most commonly with the explosive consonants, P, B, T, D, G, K, and nearly always where these occur as initial letters—that is, in starting the articulatory mechanism; and to avoid this difficulty which arises after every pause, most stammerers speak in a rapid monotonous fashion. The fault chiefly lies in the direction of energy to articulation rather than to phonation. The patient held up by his stammer usually remains silent, but occasionally, having produced the first sound, he continues to repeat it—the reduplication stammer which has been the origin for the names "stammer" or "stutter" by which the malady is known.

Often the patient uses a trick or contortion to prevent the stutter or to relieve the feeling of nervous tension and embarrassment in consciousness which the defect causes, and these tend to become engrafted on him, as (1) associated sounds—whooping, grunting, crowing, etc.; (2) habit spasms—contortions of the face, limbs, or body, which sometimes take a complicated form and exactly resemble the co-ordinated forms of tic.

Prognosis.—The majority of the cases tend to a spontaneous cure, and recovery is hastened in all cases by systematic treatment. In every class of case the results of treatment may come slowly at first, but perseverance will in almost every case bring success.

Treatment.—Attention should be paid to conditions of general health, and to the mental well-being and satisfaction of the child, with plenty of scope for pleasure and satisfying occupation.

It is well for the patient to speak, read, or recite in a large room alone, loudly, slowly and distinctly. The following system for such exercises is useful: (1) The chest must be kept well filled with air. This most important point is often most difficult to the patient. (2) He must speak slowly, with a full resonant voice. (3) When he comes to the word on which he tends to stutter, he should raise his voice and direct his energies to vocalisation, and not to articulation. If the difficulty be over a voiced consonant, he must be directed to voice it firmly. If the consonant over which he stumbles be a voiceless one, attention must be directed to the vocalisation of the subsequent vowel sound; for instance, in "pat" he must attempt to vocalise the "at," and he will find little difficulty in prefixing "p" as the syllable is uttered. (4) Gymnastic and singing exercises are valuable additions to treatment. Should associated movements be present, the speaking exercises may be carried on in front of a mirror, so that the patient may see these himself and endeavour to suppress them.

The development of confidence and self-reliance is everything in the treatment of stammering. The skilled teacher first gains the liking, respect, and submission of his patient. He then assures him that his defect will disappear, and that he can cure himself, and demonstrates to him by correcting the faults that he can speak normally.

2. **LALLING.**—A defect due to want of precision in the action of the oral

articulatory mechanism. It characterises the speech of many children before the art of articulation is completely learnt. It is only a persistent condition in some cases of defective intelligence.

3. LISPING.—A defect due to the indistinct enunciation of certain consonants, or to the substitution of wrong consonants. It usually occurs in connection with the sounds of Th, R, and S, which change to V, L, and Th respectively. The condition, which is almost usual in infants learning to speak, is due to faulty articulation, and may become a habit, in which case the subject has probably a bad “car” for sound. Defective conformity of the mouth may cause it; for example, a “tongue-tied” person can never pronounce the English R correctly.

4. IDIOGLOSSIA.—A condition in which from the first moments of learning to speak, a child uses wrong consonants, or rather he tends to substitute three or four consonants for the whole series. Very slight degrees of idioglossia are common in little children, whose early speech is intelligible only to their nannies. In marked cases the child comes to speak a language entirely its own.

The course of time and education removes the defects of lisping, lalling, and idioglossia, and the prognosis in all these conditions is invariably good.

Any deformity of the articulatory organs should be remedied if possible.

JAMES COLLIER.

Revised by F. M. R. WALSH.

APRAXIA

Definition.—A disorder of cerebral function, characterised by inability to perform certain familiar purposive movements, in the absence of motor and sensory paralysis and ataxia (Kinnier Wilson). This disorder does not depend upon defective perception (agnosia) nor upon general reduction of intelligence.

Ætiology.—Apraxia may result from both general and local diseases of the brain. It may be met with in general paralysis of the insane, in cerebral sclerosis and in several forms of dementia, and in paralytic chorea. It occurs in its purest form from local lesions of the brain, and may then be confined to one region of the body. It may result from lesions of the posterior part of the prefrontal area of the left side, the so-called “motor or verbal” aphasia and agraphia being good examples of apraxia of speech, and lesions in this region may also cause apraxia of the limbs on one or both sides. Lesions of the anterior half of the corpus callosum have been associated with conspicuous apraxia, as have also bilateral lesions in the posterior parts of the hemispheres. In the latter cases, the apraxia is likely to be associated with some degree of lack of recognition of an object, and of its uses (agnosia), and this causes apraxia from a loss of correct comprehension of the act required. Apraxia is sometimes met with in cases of hemiplegia in which, notwithstanding the complete recovery of motor and sensory paralysis, the performance of familiar acts—from the highest skilled movements, such as the fingering of the pianoforte or of the violin, or the use of his tools by a craftsman, to the simplest act—may be no longer possible. The features of the condition may be well demonstrated by the consideration of left-sided

hemipraxis. There is neither loss of power nor loss of sensibility in the left upper extremity. When such a patient is asked to perform some familiar act with the right hand, he at once does so correctly, but when ordered to perform the same act with the left hand he is unable to do so. Either he makes aimless wandering movements with the left hand, or he may succeed in making movements somewhat resembling those required of him, with much slowness and clumsiness. Sometimes he may perform some act which is entirely different from that required of him, and this phenomenon is called *parapraxis*. When the apraxia is partial, the patient may be able to perform some acts and not others, his inability usually, but not always, increasing with the complexity of the act required. Or he may be able sometimes to perform an act in which he commonly fails. Not infrequently such a patient, wearied with the unsuccessful attempts of his left hand, will abruptly perform the act correctly with his right hand, to get rid of it. And he will define his defect by saying, "I know quite well what you want me to do, but I cannot do it." Spontaneous volitional movement is similarly affected, and this leads invariably to a marked loss of initiative in the use of the affected limb—the patient will not try to use it. The apraxic patient is often to an astonishing degree unaware of his disability, and frequently becomes conscious of it for the first time when it is pointed out to him by another person.

Diagnosis.—Apraxia may be confused with astereognosis, with agnosia and with cortical ataxia. A correct conception of the nature of the two former conditions will exclude the possibility of error. In cortical ataxia the patient obeys the word of command at once and succeeds more or less with the act required, the defect being clumsiness of execution. The clinical examination of patients for apraxia must include—(1) the general psychological condition as regards attention, memory and reasoning; (2) an inspection of sensory appreciation for defects of simple perception in the regions of smell, sight, hearing, taste, cutaneous sensibility and muscular sense; defects of recognition of sensory impressions in these regions (agnosia); defects of memory; and (3) an examination of executive power for any defects in the movements determined by visual, auditory, tactile and kinæsthetic stimuli. What response does the patient make to objects held in front of him or to gestures made to him? Can he imitate movements? Can he when requested make simple and purposive movements, with and without the objects in his hands? When given an object, how does he hold it and use it?

AGNOSIA

In certain conditions of cerebral disease, it is found that each and all of the sensory organs, when called into play, may fail to arouse an intelligent perception of the object exciting them. This inability to recognise the import of a sensory stimulus is called *agnosia*. Those patients who present apraxia and agnosia, often show other interesting phenomena which are of importance; these are (1) inattention, (2) defective capacity for retaining recent impressions, (3) lack of initiative, and (4) perseveration. Perseveration consists in the repetition of an already executed movement when and only when the patient desires to make a fresh movement.

CEREBRAL DIPLEGIA

Synonyms.—Congenital Spastic Paralysis ; Lobar Atrophic Sclerosis.

Definition.—A series of clinical conditions, dependent upon lack of, or imperfect development, or degeneration of the nerve cells of the cerebral cortex, basal ganglia or cerebellum. This agenesis of nerve cells may affect those cells of the pyramidal system which are the latest to develop before birth, namely those for the supply of the lower extremities and the resulting clinical condition is cerebral spastic paraplegia or Little's disease, or all the cells of the pyramidal system may be affected, producing generalised spastic rigidity. Again, the higher regions of the cortex may be affected, and the result is congenital idiocy. Similar affections of the cells of the basal ganglia result in congenital bilateral athetosis, and congenital chorea. When the cerebellum is involved, congenital cerebellar ataxy results. Further, there may be any combination of the above conditions.

Ætiology.—The malady may be apparent at the time of birth, as the child may be born with contractures present. More often, the signs of deficient or perverse movement, or of mental deficiency, appear during the first year of life, as the signs of cerebral activity commence to be exteriorised. In other and rarer cases, the degeneration of the nerve cells seems to be truly post-natal in onset, as in amaurotic family idiocy. In most cases no heredity can be traced, but sometimes several children of the same mother may be affected, and direct heredity has been known. Amaurotic family idiocy is always familial, and is almost, but not quite, limited to the Hebrew race.

Abnormalities of birth are frequent. Premature, or precipitate birth, prolonged birth from uterine inertia rather than from dystocia, and asphyxia neonatorum are all common. The child is frequently the first born of its mother.

Collier has expressed the probable pathogenesis of cerebral diplegia as follows : "If we regard the brain from the time of its earliest stages of development as a field sown with seeds (neuroblasts), which germinate at different periods of foetal life, and the germination is not even complete at the time of birth, the germination of all the elements in due time and their complete development being necessary for the formation of the perfect brain, then we may liken the cause of diplegia to some baneful influence, such as a frost, which acting at a particular time, may spare those seedlings which are well developed and able to withstand it, and those seeds as yet not germinated, but which causes havoc among the tender germinating seedlings, either to their death or severe maiming. In some cases, as, for example, in Little's disease, the neuroblasts thus affected may, after a period of retarded development, ultimately become strong plants and complete their development. It is of interest that in the highest degrees of cerebral agenesis—anencephaly, pituitary abnormalities seem to be constant."

Pathology.—The essential histology of the affected regions is that of non-development, paucity in numbers and degeneration of the nerve cells, with corresponding absence, poor development, degeneration or a combination of these states, of the tracts which spring therefrom. The pyramidal tract, for example, may be found absent throughout, or it may reach to the medulla,

or to the cervical region only, and so show at what period development was arrested. The changes in the nerve cells are followed by secondary gliosis. The final result is termed atrophic sclerosis. More often certain regions are profoundly affected, while others escape relatively or completely; but the distribution is always symmetrical upon the two hemispheres. The convolutions are unduly hard to the touch, and their surfaces often present a worm-eaten and faceted appearance. This irregular form of the convolutions, with wide, separating sulci, gives the brain a characteristic appearance, like that of a walnut kernel.

Symptoms.—The clinical picture of the several forms of cerebral diplegia presents a combination in varying degrees of certain characteristic symptoms, always bilaterally distributed, though sometimes more severe on one side than on the other. These symptoms are: muscular rigidity, paresis, perverse movements, contractures and increased deep reflexes. Mental deficiency, optic atrophy and ataxy are other important symptoms. The signs of the disease become obvious during the first year of life or soon after. In severe cases, soon after birth, the nurse, in washing the child, is the first to notice the stiffness of the limbs, or the regular assumption of a curious bodily attitude. Otherwise, the abnormalities may not be obtrusive, until the child should sit up or learn to get about, when weakness, rigidity, perverse movements and pes cavus may call attention, or backwardness in learning to walk and to talk, and mental deficiency may first suggest that there is something wrong with the child. The following are the common types of the disease, but it must be remembered that any combination of, or transition between, the types may be met with. Cerebro-macular degeneration has certain peculiar features which necessitate a separate description for this malady:

1. *Generalised rigidity; general congenital spastic paralysis.*—There is extensive defect of the pyramidal system. The rigidity and weakness affect the whole of the musculature.

2. *Paraplegic rigidity; congenital spastic paraplegia; Little's disease.*—The pyramidal deficiency is confined to that supplying the lower part of the trunk and lower limbs.

3. *Congenital bilateral athetosis and congenital chorea.*—The agenesis affects the cells of the basal ganglia, with the appearance of irregularity of movement, and of spontaneous involuntary movements, which may be of an athetotic, choreic or irregular type. A certain variable degree of general rigidity is present in these cases.

4. *Congenital cerebellar ataxy.*—The agenesis affects the cerebellum with the appearance of cerebellar ataxy. In this type, the limbs are flaccid, and in mixed cerebral and cerebellar types there is a tendency to hypotonicity of the muscles, instead of rigidity.

5. *Congenital idiocy; restless idiocy.*—The agenesis affects those parts of the brain concerned with the higher functions. These children are emotionless, restless and unteachable. The skull often shows frontal or occipital microcephaly.

6. *Microcephalic idiocy*—where the agenesis is of the whole brain and the skull very small.

7. *Cerebro-macular degeneration.*

PARESIS AND RIGIDITY.—Except in severe cases, in which the weakness

amounts to complete paralysis, there is more rigidity than weakness, and it is often astonishing that there should be so much power in the presence of such a degree of rigidity. The lower extremities are generally the most affected, the upper to a less degree, and the facial region still less. Movement is slow and clumsy and resembles that of the tardigrade animals, and spontaneous involuntary movements are often present in the limbs. Contractures accompany the rigidity, and if walking is possible the gait is digitigrade from contraction of the calf muscles, the knees are flexed from contracture of the hamstrings, the thighs are rotated inwards, and the knees pressed together, rubbing against one another. More severe adductor spasm gives rise to the cross-legged progression. The rigidity and contractures, when severe, may give rise to peculiar attitudes and deformities. A mask-like expression of face, with wide palpebral apertures and large open mouth, is not infrequent. Slobbering is very common. The head may be rigidly retracted, but more commonly the chin is pressed down upon the chest. The spinal column generally shows some deformity in the way of kyphosis, lordosis or scoliosis, and pes cavus or equino-varus is the rule.

PERVERSE MOVEMENTS.—Under this heading must be grouped the very constant maladroitness of voluntary movement, the facial over-action and grimacing in speech and in mimetic expression, choreic movements, athetotic movements and intention tremor. Common sensation and the muscular sense are unimpaired. The sphincters are unaffected. The deep reflexes are increased, but are often difficult to obtain when rigidity is very marked. The trunk reflexes are often absent, the plantar reflexes usually are extensor in type. Since the growth of the skull follows and conforms with that of the brain, cranial abnormalities are common. There may be microcephaly, asymmetry and flattening in the region of the central convolutions, or a furrow corresponding with the interhemispheric fissure, or frontal or occipital smallness and flattening. Every degree of mental reduction may be met with, from precocity and slight mental dullness to complete amentia. But this by no means corresponds with the severity of the bodily symptoms, for the mental defect is often most severe when the bodily symptoms are slight, and conversely. In some cases, very high intelligence persists, when there is utter uselessness of the limbs, and when speech is hardly intelligible. Primary optic atrophy occurs in a small number of cases. Inequality of the pupils and slowness of light reaction are not uncommon. Nystagmus is often met with. Convergent strabismus occurs in about one-third of the cases. Convulsive attacks are of common occurrence, and in about one-eighth of the cases epilepsy becomes established.

Diagnosis.—When the symptoms are well marked, the diagnosis presents little difficulty, since the disease dates mostly from birth, or is discovered during the first year of life. Paraplegic rigidity may possibly be confused with other forms of paraplegia, and, especially, with that resulting from spinal caries. Certain cases of pontine tumour may closely resemble generalised rigidity. The occurrence of such conditions during the first two years of life is, however, very rare.

Prognosis.—In many cases of generalised rigidity, and in all cases of paraplegic rigidity, there is a tendency to slow amelioration of the rigidity, an increase of voluntary power and control of the muscles in the course of time, especially under the influence of careful training, and in paraplegic

rigidity, if the mental acuity be not seriously impaired, laborious treatment may result in an almost normal condition of the limbs by the age of puberty. On the other hand, some cases of generalised rigidity become progressively worse, and succumb, usually before the end of the fourth year. Bilateral athetosis and choreic diplegia, as a rule, follow a very slowly progressive course, without tendency to a fatal result. Paraplegic rigidity apart, a great many of the cases of all forms of diplegia succumb before the sixth year, and in those who survive this age, the tenure of life is short, few reaching far into the third decade of life.

Treatment.—In those cases with a marked degree of mental impairment, and in those which show a course of progressive degeneration, no treatment is of avail. In slighter cases of generalised rigidity, and in paraplegic rigidity, treatment is to be directed to the prevention of the rigidity, to regaining of voluntary control, and the improvement of mental acuity. There is, perhaps, no disease which demands greater patience and persistency in carrying out of suitable treatment, and there are few diseases in which more brilliant results may be produced from apparently hopeless cases by pertinacity in treatment. It is in the early years, when treatment is for the most neglected, that good results are more quickly and readily obtained. From the first, regular massage and passive movements should be employed. Voluntary movement should be encouraged, as far as possible, and as power and movement increase, gymnastic exercises of every kind should be employed. Rigid apparatus for prevention of deformity and to reduce contracture is harmful, for it increases the weight of the limb, and interferes with movement, which is the remedy with which paralysis is to be combated. Tenotomy is of great service in the relief of deformity and contracture, and should be soon followed by passive movements. It should never be performed, unless a fair degree of voluntary power is present. Many of the patients seem to improve more rapidly if thyroid be administered in moderate daily doses.

CEREBRO-MACULAR DEGENERATION: AMAUROTIC FAMILY IDIOCY

1. WARREN TAY-SACHS' DISEASE: THE INFANTILE FORM

Definition.—A family disease of infancy occurring chiefly, but not entirely, in the Hebrew race, affecting children during the first year of life, who are apparently quite healthy when born, and characterised by—(1) progressive mental impairment, ending in absolute idiocy; (2) progressive paralysis of the whole body; (3) progressive diminution in sight, ending in absolute blindness. Pathognomonic retinal changes are constantly present, consisting of a large and conspicuous "cherry-red spot" in the region of the macula, and, in addition, optic atrophy occurs later and (4) a fatal termination in the marasmic state before the age of 2 years.

Ætiology.—Nothing is known of the ætiology of the disease apart from its familial and racial incidence. The tendency to the disease is unquestionably congenitally installed.

Pathology.—This is very striking. It consists of a progressive degeneration of the nerve cells from the highest to the lowest, and ultimately there may be no normal cells remaining anywhere in the nervous system.

The degeneration takes the form of swelling of the cell protoplasm, and of the dendrites with chromatolysis, swelling of the hyaloplasm and destruction of the cell fibrils, followed by disappearance of the nucleus, and finally by absorption of the remains of the cell. Every cell of the central nervous system both of the brain, spinal cord and spinal ganglia is in the end similarly affected.

Symptoms.—There are few diseases in which the *clinical manifestations* are so perfectly uniform as in this malady. The children have all been born at full term, and in perfect health. They thrive well during the first 3 to 6 months of life, when they gradually become listless and apathetic, cease to take interest in the surroundings, and begin to show signs of the visual failure which ends in blindness. Later, the child is unable to sit up, or to hold up its head. The limbs, which may be slightly spastic at first, become flaccid and motionless. There is a gradual increase of all these signs. The mental defect becomes more and more noticeable, the paralysis more extreme, complete blindness follows, and the patient sinks into a condition of marasmus, in which he dies. Convulsions, nystagmus and strabismus are sometimes present.

The retinal changes are pathognomonic and are due to a degeneration and disappearance of the nerve cells of the retina and their processes, which constitute the fibres of the optic nerve. This change is most intense in the region of the fovea centralis, where the retina thins and disappears over a circular area, exposing the vascular choroid. This gives rise to the characteristic appearance, on ophthalmoscopic examination, of a cherry-red spot in the region of the macula. This spot is actually a hole in the retina exposing the choroid. The optic disk shows progressive atrophy.

Diagnosis.—Distinction has to be made between this and other forms of progressive diplegia. The symptoms are so distinct that a physician, who is acquainted with the disease, and able to recognise the retinal picture, can hardly fail to make the correct diagnosis.

Treatment.—No treatment is of any avail.

2. OTHER FORMS OF CEREBRO-MACULAR DEGENERATION

In addition to the classical infantile form described in the preceding article, two other forms are well known in which the pathological changes are similar but much less severe than in the Waren Tay-Sachs' disease, and there is a similar familial incidence, but the incidence of the malady occurs later in life and the course is less rapid and the result far less serious. The later the onset in life the slighter and less progressive are the symptoms. The cherry-red spot at the macula, so constant in the infantile form, does not occur in the later forms. The characteristic retinal change is a disturbance of the retinal pigment commencing in the macular region, rather like retinitis pigmentosa, accompanied by honeycomb changes at the macula and sometimes by optic atrophy. The *juvenile* form occurs in later childhood and is characterised by the association of the retinal changes and visual defect with some degree of mental deterioration. The *adult* form is the least progressive of any, and the clinical manifestations are the visual defect and retinal changes in the absence of mental deterioration.

INFANTILE HEMIPLEGIA

While in childhood hemiplegia of slow onset is due to the same causes as in adults, cerebral tumour being the common cause and chorea not an infrequent one, yet the majority of the cases of infantile hemiplegia of rapid onset are examples of diseases peculiar to children, to which no comparable disease occurs in adults, and to such cases the term "infantile hemiplegia" is restricted. These conditions are due to gross organic lesions of the brain, and for this reason must be strictly separated from the cerebral diplegias which are the result of cell lesions and not of gross lesions.

Ætiology.—In two-thirds of all the cases, the onset occurs within the first three years of life. The malady becomes increasingly rare as childhood advances. A few of the cases are of prenatal origin, and some of these have been proved to have been due to injury to the fœtal brain from a blow upon the mother's abdomen, while others are due to syphilitic fœtal vascular disease. In a third class of mysterious origin, mothers have given birth to several hemiplegic children, examples of which we have recorded. Some of these children are born with definite hemiplegia and contractures. Again, a very few cases are due to obstetrical events during birth, by which the cerebrum is injured. Acute infective diseases play a very important rôle in the causation of the disease, for about one-third of all the cases develop the malady during the course of a known infection. By far the most important of such fevers are measles and scarlet fever, but hemiplegia may occur in the course of pertussis, small-pox, röteln, diphtheria, dysentery, pneumonia, typhus, typhoid, mumps, malaria, chorea and endocarditis. While there can be no doubt that primary vascular lesions are responsible for a few of the cases in which this condition complicates the specific fevers, whooping-cough, for example, may cause cerebral hæmorrhage; marasmic conditions in any fever may cause thrombosis of cortical veins, and chorea and endocarditis may cause embolism, yet it is certain that in the majority of cases, an inflammatory focal lesion of the brain or encephalitis is the pathological lesion. In cases which arise with no definite ætiological connection, it seems clear that a primary encephalitis is responsible, but there is no evidence at present as to its causal factors.

Pathology.—The following lesions are met with, either alone or combined in order of frequency: (1) Atrophic sclerosis; (2) cyst formation; (3) shrunken patches resembling wet wash leather, with some degree of atrophic sclerosis in their vicinity, and (4) porencephaly. Of these, the atrophic conditions seem to be the results of encephalitis, which may also cause some cyst formation; the cystic conditions may result from the above, or from hæmorrhage or thrombosis, and porencephaly is certainly due to embolism.

Symptoms.—The onset is rapid, and in two-thirds of all the cases the disease is ushered in by convulsions, which may be unilateral, but are more frequently general, and are frequently repeated during a period of from a few hours to 24 hours, after which the patient sinks into a subconscious state, from which he gradually emerges in the course of a few days, to show the signs of some cerebral defects, usually hemiplegia, sometimes hemianopia, or aphasia, or any other sign of local cerebral or cerebellar lesion. Pyrexia

often accompanies the convulsion, and vomiting is common. The onset may be without convulsions or loss of consciousness.

The relation of the onset of the paralysis to the convulsion varies. It may reach its height immediately after the initial convulsion, or slight hemiparesis may occur which deepens after each subsequent convulsion. Sometimes the early convulsions leave no paralysis, but this appears towards the end of the first week, either suddenly with fresh convulsion, or gradually, as the patient recovers from the comatose state. The paralysis at its onset is flaccid, and involves the whole of one side of the body to a greater or smaller extent. An initial monoplegia is of extreme rarity. The paralysis may not reach the greatest intensity until the end of the second week. Subsequently it lessens, in some cases disappearing completely in from a few weeks to 3 months; in others, it may show no signs of improvement. The limbs, at first flaccid, subsequently become spastic and develop contractures. In the course of years there may be great arrest of growth on the affected side, and this is not in relation with the degree of paralysis, but apparently depends upon the degree of destruction which has occurred in the parietal lobule. Post-hemiplegic spontaneous movements of an athetoid, choreic or irregular kind are common, and are attributable to lesions in the corpus striatum and subthalamic grey matter, for which regions encephalitis shows an especial predilection. Epileptic fits recur at varying intervals in about half of all cases of infantile hemiplegia. These always commence upon the affected side and are sometimes confined to it. Mental deficiency is met with in all degrees, in relation to the position and extent of the cerebral cortex which is involved in the lesion.

Diagnosis.—The nature of the malady at the onset, with convulsions, may be possibly suggested by prodromal pyrexia, by the severity and long duration of the convulsions, and by the prolonged subconscious state that often follows. Convulsions occurring several days after the onset of specific fevers should strongly suggest the diagnosis. When the signs of hemiplegia or of other local cerebral lesions appear, the diagnosis presents no difficulty.

Course and Prognosis.—In a very small proportion of the cases the patient does not survive the initial manifestations of the disease, and dies in convulsions. Apart from this event, infantile hemiplegia has little tendency to destroy life. The initial flaccid hemiplegia tends to improve and gives place to a slowly improving spastic hemiplegia, which, with the return of some power, shows perversity of movement, stiffness and slowness, ataxy, athetosis and choreic movements or tremors according to the position of the lesion. The spontaneous movements appear within a year of the onset. Slow improvement may go on for years, but cases with much mental reduction or when recurring epilepsy is frequent, improve but little.

Treatment.—We know of no measures that avail to prevent the occurrence or lessen the severity of the cerebral destruction which occurs from encephalitis. Too often the damage to the brain has happened as soon as a diagnosis is possible. When the paralysis has developed, treatment is to be directed to the prevention of rigidity and contractures by regular passive movements, to regaining voluntary control by encouragement and patient exercises, and to the improvement of mental acuity. Where there is much contracture and deformity, tenotomies are of great service, provided there be some voluntary power in the muscles, the tendons of which are

to be divided. Recurring convulsions should be treated as idiopathic epilepsy.

SYPHILIS OF THE NERVOUS SYSTEM

Of the many problems presented by syphilis of the nervous system one may be chosen for special reference, namely, the alleged dual nature of nervous syphilis and of the causative organism. It has been maintained that there are two distinct pathological forms of nervous syphilis, namely, primary *parenchymatous* syphilis, as exemplified in tabes and general paralysis, where the initial lesion is held to be in the nerve elements themselves, and primary *meningo-vascular* syphilis, in which the initial lesion is in the blood vessels and meninges. But the dual nature of syphilis implied by these distinctions is subversive of pathological principles, for the initial and fundamental lesion of syphilis, wherever found and at all stages, is a lymphangitis or an arteritis, and very strong evidence would be needed to enforce the conviction that the reactions of nervous tissues to the presence of the spirochæte differs essentially from that of all other tissues of the body. Nor is this view supported by the morbid anatomy of the diseases concerned, for in every case of tabes and general paralysis, vascular and meningeal lesions can be found after death. Moreover, in the vast majority the increased number of cells in the cerebro-spinal fluid shows that the meninges are attacked even in the earliest stages.

For these reasons the trend of opinion is to deny the existence of parenchymatous syphilis as an initial lesion of syphilis in the nervous system, and to hold that the vessels and meninges are first injured in all forms of syphilis of the nervous system.

The contention that the organism of syphilis exists in two forms next demands consideration. Of all syphilitics the proportion in whom the nervous system is attacked is small. To explain this low incidence it has been assumed that neuro-syphilis results from infection by a biological variant of the *Spirochæta pallida* with special affinities for nervous tissues—the “neuro-tropic variety,” while other forms of syphilis follow infection by the “dermo-tropic variety.” At first sight this attractive conjecture seems to be supported by numerous clinical observations. These are, that in some instances several persons infected from the same source have later developed syphilitic nervous diseases; that the superficial manifestations of syphilis are often mild or absent in those who ultimately develop neuro-syphilis, and that secondary and tertiary syphilis, outside the nervous system of these patients, is rare; also that in Oriental countries where syphilis is common, some forms of neuro-syphilis, namely, tabes and general paralysis, are seldom seen.

But it has never been shown that the persons who formed the source of infection for several cases of nervous syphilis have themselves developed this disease; it can no longer be held that tabetics and paralytics are free from secondary and tertiary lesions—witness the frequency of aortitis in these cases; finally, Europeans who contract syphilis in the East are just as likely to suffer from tabes and general paralysis as if they had contracted

it at home. It cannot be denied that the secondary phenomena are often so slight as to pass unnoticed by patients who later become tabetics or paralytics, or that patients with severe integumental lesions rarely develop tabes or general paralysis. These facts, however, together with a large amount of additional evidence, suggest that the ultimate result of infection depends rather on the individual attacked than on any peculiarity of the infecting agent, and they lend no support to the notion of the duality of the syphilitic organisms.

For the purpose of description it is still convenient to describe syphilitic diseases of the nervous system under two headings; Interstitial or meningo-vascular syphilis and parenchymatous syphilis. To avoid misunderstanding it must be emphasised that these are merely clinical aspects of one disease—neuro-syphilis, and that in both forms the primary lesion is in the vessels and meninges.

THE BLOOD AND CEREBRO-SPINAL FLUID IN SYPHILIS OF THE NERVOUS SYSTEM

A normal fluid may be regarded as one with a pressure equal to 150 to 180 mm. of water, a cell count not exceeding 5 per c.mm., an albumin content of from 0.025 to 0.05 per cent., and negative Wassermann, globulin and Lange's gold tests.

In *secondary syphilis*, without nervous symptoms, changes are found in the fluid in 80 per cent. of the cases. The infection of the nervous system occurs early in the second stage of the disease and it is a common happening. In many of the cases this infection dies out in the course of time either spontaneously or as the result of treatment and the cerebro-spinal fluid reverts to a normal condition. In other cases the infection remains and the W.R. continues positive in the cerebro-spinal fluid, and it is exclusively in this class of patient that tabes, general paralysis and the other degenerative maladies of the nervous system which are due to syphilis arise. Increased pressure, lymphocytosis, excess of albumin or a positive Wassermann reaction are found with a frequency which diminishes in this order. If the blood and fluid are both normal at the end of a year, neuro-syphilis is not likely to arise.

In *latent syphilis*, without nervous symptoms, the blood is positive in about 70 per cent., but changes in the fluid are found in 20 per cent. only. A fluid which is normal in the latent stage almost never becomes pathological later; if it deviates from the normal it is highly probable that neuro-syphilis will develop later.

In *early cerebro-spinal syphilis* the blood is usually positive, but it is sometimes negative when treatment has been thorough or recent. This indicates that an examination of the fluid is essential when the blood is negative in a case where the diagnosis is doubtful. Cells and albumin are usually greatly increased, the gold test is usually positive, and the Wassermann reaction is positive in almost every case when 1 c.c. of fluid is used in making the test. The blood and fluid often become normal after the first course of treatment, but a relapse occurs frequently and long before the recurrence of symptoms. Vigorous mercurial treatment and 4 or 6 full courses of arsphenamine at intervals of 3 months usually render the blood and fluid persistently negative. If both are normal a year after the fourth course, recurrence is unlikely.

In *cerebro-spinal syphilis* of longer duration, the reaction in the blood is

almost always positive, and the fluid is seldom normal in active cases. A positive reaction in the fluid probably indicates an active process, even in the absence of recent symptoms. A normal fluid indicates a healed process. The inference is that all cases with a positive fluid and a history of cerebro-spinal syphilis should be treated energetically whether fresh symptoms are present or not. The treatment outlined above under early cerebral syphilis usually renders the fluid negative and the indications for prognosis are the same.

In *tabes* Wassermann's test is positive in the blood in about 70 per cent. of cases; it is often negative in the fluid when the test is done by the original method with 0.2 c.c. of fluid, but with 1 c.c. it is positive in almost 100 per cent. An increase in the number of lymphocytes in the cerebro-spinal fluid as well as an increase in the total albumin with a relative increase of globulin is almost constant. In some cases in which the disease had remained stationary for a long time the cerebro-spinal fluid was normal in every respect.

Lange's colloidal gold test gives useful information when the diagnosis lies between *tabes* and general paralysis, for the latter gives a typical reaction, but the test is of no value by itself in distinguishing *tabes* from other diseases of the nervous system (see page 1558).

Treatment by mercury and intravenous injections of neoarsphenamine sometimes renders the fluid normal. It is stated that in such instances relapse is less likely to occur than in other cases, but no exact correlation has been shown to exist between the clinical course of the disease and the presence or absence of changes in the blood and fluid.

General paralysis of the insane.—The Wassermann reaction is strongly positive in the blood and in the fluid practically in 100 per cent. of cases, and an increase of cells and a positive globulin reaction are almost constant. Lange's gold test gives the characteristic paretic curve.

Treatment by mercury and arsphenamine may produce slight modification in the reactions, but it has very little effect in checking the progress of the disease.

THE ESSENTIAL LESION OF SYPHILIS

Every lesion in syphilis commences with the collection of spirochaetes in the lymphatic spaces surrounding small arteries. This is followed by an inflammatory reaction with oedema and exudation of many lymphocytes and plasma cells around the small vessels, and the "cuffing" or "muffing" of these vessels with such cells is characteristic. These cells may wander freely into the nervous tissue away from the vessels and may form clumps, often containing giant cells, and these are miliary gummata. Such a perivascular lymphocytic exudation is typical of syphilis, poliomyelitis, tuberculosis and lethargic encephalitis, but the distinction can be made by the nature of certain histological elements present. Syphilis is distinguished by the presence of numerous plasma cells among the lymphocytes, poliomyelitis by the large admixture of polymorphs, tubercle by the absence of plasma cells and the presence of Koch's bacilli, and lethargic encephalitis by the absence of any elements except the lymphocytes. The initial periarteriolitis of syphilis is often followed by invasion of the whole vessel wall (panarteritis), and often proliferative endarteritis which may give rise to thrombosis is the most conspicuous feature in the panarteritis. Later, the wall of the vessel may scar and may develop patchy calcareous deposit. The lymphocyte

deposit goes into fibrosis or increases to gumma formation, and there is neuroglial felting. Further thrombosis of the vessel may cause the softening and infarct conditions which necessarily follow vascular obstruction, and local necrosis results. The hyperproteinia and pleocytosis of the cerebro-spinal fluid are expressions of this essential lesion upon the surfaces and spaces of the nervous system, and the meningeal scarring and adhesion are its results.

So far the pathology of nervous syphilis is simple, but the so-called "parenchymatous" or degenerative lesions which are apt to be widespread and progressive are as yet inexplicable. They are commonly found in the absence of any findable spirochæte, or of any sufficient inflammatory lesions in their locality, and have been observed also by Carey Coombs in the heart muscle and the aorta, and may be progressive when all signs of active syphilis such as the W.R., hyperproteinia and pleocytosis have died out finally. These degenerative lesions may not be improved or stayed in their progress by any form of treatment. The slowly oncoming progressive and unarrestable optic atrophy and the systemic lesions of the spinal cord in tabes are good examples of the degenerative lesions, and in them spirochætes have rarely or never been found, while the inflammatory lesions are absent or minimal and cannot be the factors of so wide a destruction. The nearest locus where spirochætes are commonly found in tabes is the bronchial glands.

CEREBRO-SPINAL SYPHILIS

Ætiology.—Cerebro-spinal syphilis (excluding tabes and general paralysis) occurs in about 4 per cent. of all persons who acquire syphilis. The onset of symptoms is commonest from 1 to 5 years after infection, but it may be as early as 2 or 3 months, or as late as 30 or 40 years. The brain is affected more often than the spinal cord, and usually when the main symptoms point to the latter some signs will be found to show that the brain is also attacked.

1. CEREBRAL SYPHILIS

Pathology.—The disease may begin in the meninges, in the blood vessels or in the bones of the skull. In cases where the main incidence falls upon the *blood vessels*, the arteries at the base of the brain forming the circle of Willis or arising from it, together with their branches, are most often attacked. To the naked eye they show irregularities in size, due to thickening of their walls in circumscribed areas. Proliferation of the intima with a round-celled infiltration of the outer coats—*endarteritis obliterans*—is the characteristic microscopical change. The same changes occur in smaller arteries within the brain or on its surface, and these vessels, as well as those at the base, may be compressed or invaded by disease beginning in the meninges. In each case their lumen is narrowed or obliterated, thrombosis occurs readily, and softening may result in parts cut off from their blood supply. Obliterative changes also occur in the veins and lymphatics, and lead to further impairment of the nutrition of the brain.

The commonest form of *meningeal syphilis* is a diffuse gummatous leptomeningitis at the base of the brain. On the convex surface of the brain

it begins most often over the frontal and parietal lobes. The meninges may be affected alone, but more often the vessels are also diseased. In severe cases a gelatinous exudate fills the sub-arachnoid space and extends along the vessels and nerves. Later, the newly-formed tissue organises, and forms sclerosed masses of thickened adherent membranes containing numerous small gummata.

Gumma of the brain is rare. When present it arises from the meninges, and may be multiple. The convexity of the hemisphere in the motor region is the site of election. A gumma may spread so as to involve the overlying bone.

Many important results of cerebral syphilis are due to impaired nutrition of parts not directly affected. Thus softening, hæmorrhage, cyst-formation, atrophy of cells and tract degenerations may occur, and non-syphilitic diseases may be simulated.

Symptoms.—The main incidence of the disease may fall upon vessels or membranes, the lesions may be diffuse or circumscribed, any portion of the brain or any cranial nerve may be affected alone, and every combination of lesions and, therefore, of symptoms, is possible. In many cases their multiplicity, their presence in unusual combinations, and the changes in their intensity and distribution from time to time give a clue to their nature.

Headache is a common prodromal symptom. It is often severe, and is usually worse at night. Sustained mental and physical effort becomes difficult, the memory is impaired and the character changes. Irritability, intense excitement or delirium may follow, but more often the patient becomes lethargic. At this stage paralysis and localising signs may be absent. In many the pupils are unequal or irregular, or they contract sluggishly to light, and in some the optic disks show blurring of the edges and other signs of early papilloedema.

At any time, with or without prodromata, more definite signs of vascular disease or of paralysis of one or more of the cranial nerves may appear. *Arterial thrombosis* is usually preceded by prodromal symptoms, but it may come on in one apparently well. Its seat of election is the middle cerebral artery or its branches, and weakness of one arm or of one side of the body with or without aphasia is a very common early symptom. The weakness is often slight and transient. If hemiplegia occurs, it takes several hours or a day or two to develop. It is more often a paresis than a paralysis, and consciousness is usually retained. Sometimes the affected limbs are rigid and tremulous. The symptoms of thrombosis in other arteries are given on p. 1559.

In *vertical meningitis*, headache is usually severe, and the skull is often tender over the affected part. It frequently attacks the motor areas, and convulsions are common. They may be confined to one limb or to one side of the body, consciousness being retained, but more often they become generalised and consciousness is lost. In more chronic cases mental symptoms may predominate, the patient becoming slowly demented.

With *basal meningitis* severe deep-seated headache is almost always present. Later, a characteristic lethargy with severe impairment of the mental functions may appear. Whilst in this stuporose state the patient can usually be roused, when he answers questions in a sleepy fashion and obeys simple commands, but his memory is bad, and he is unable to give an

adequate account of himself. Variations in the degree of the stupor are a striking feature. In some cases profound torpor may persist for several weeks.

Soon after the onset, signs of implication of the cranial nerves appear. Any one of them may be affected alone, but, as a rule, several are attacked, where they lie close together after their exit from the brain, or as they leave the skull through the foramina. Ocular symptoms are rarely absent. Inequality in size, or irregularity in the outline of the pupils, may be the only sign, but diminution of the light reflex, ptosis, squint, diplopia and weakness of the movements of the eyeball are frequent, and papilloedema is common. It is characteristic of the cranial nerve palsies in syphilitic basal meningitis that the symptoms often show great variations in degree and distribution at different times.

The symptoms produced by large *gummata* are those of any cerebral tumour—headache, vomiting and papilloedema.

Diagnosis.—The aphorism, probably justified 25 years ago, that syphilis should be thought of in every case of nervous disease that is not quite clearly non-syphilitic in origin, has long outlived its usefulness as a generalisation. But it still lingers and is responsible for the practice, far too general, of throwing the burden of neurological diagnosis upon the shoulders of the clinical pathologist to make blood and cerebro-spinal fluid examinations, as a substitute for that careful history taking and clinical examination that would usually suffice for a complete diagnosis without these accessory investigations. Neuro-syphilis should not be guessed at in a haphazard fashion, but the attempt should be made by the clinical methods mentioned above to assess the pathological nature of the lesion or disease process in any case of nervous disease, and to use serological methods to confirm clinical diagnosis, or to clear up uncertainty when this still obtains after full clinical consideration. A history of syphilis or signs of the disease elsewhere are of first importance. Obstinate headache alone should arouse suspicion, and when signs of vascular or cranial nerve troubles follow a prodromal period of headache and mental impairment, syphilis is the most likely cause.

Jacksonian epilepsy or fits of any kind in patients without an epileptic history are suggestive of syphilitic meningitis or gumma, even though these are not the commonest causes of these symptoms. As a rule, these convulsions are readily distinguished from those of idiopathic epilepsy, by their partial distribution, by the weakness which remains in the parts which were convulsed, and by the presence of papilloedema, of cranial nerve palsies, or of other signs of organic disease which are found in the intervals between the fits. Cerebral new-growths produce similar symptoms, but this diagnosis should not be finally accepted until syphilis has been excluded. In all cases an examination of the blood and *cerebro-spinal fluid* is essential (see p. 1629) because a positive Wasserman or Kahn reaction in the blood may be taken to indicate no more than that the subject has at some time had syphilis. It does not usually mean that the tumour is syphilitic in nature. It is on the whole less rare to find a glioma in a syphilitic subject than to find a gumma of the brain. Only the presence of the changes characteristic of neuro-syphilis in the cerebro-spinal fluid, as well as in the blood, can justify the diagnosis of cerebral gumma.

Prognosis.—The outlook in cases with mild symptoms is good if the treatment is efficient. In severe cases of syphilitic arteritis it is grave. The vessels most often invaded are large end-arteries, and the softening which results when their lumen is occluded is not amenable to anti-syphilitic remedies. Relapses are common, and patients apparently cured are often seized in a few months with fresh cerebral troubles. The lesions in the meningeal and gummatus forms of the disease are mainly cortical, and large areas of softening are not produced. The prognosis is much better in this form, and complete cure is almost the rule when treatment is begun early. It is often impossible, however, to decide from the clinical signs whether the vessels or the meninges have suffered the more. In the absence of this knowledge the prognosis depends on the results obtained by the use of appropriate remedies.

Treatment.—See p. 1636.

2. SPINAL SYPHILIS

Pathology.—The disease may begin in the bony or membranous coverings of the cord, in the blood vessels on its surface, or in the interstitial tissues within its substance, and spreading from one to the other, usually attacks several of these structures in various combinations. As in cerebral syphilis, many of the changes in the cord are secondary to disease in the vessels or meninges, and appear in parts not directly attacked by specific processes. The commonest form of spinal syphilis is *meningo-myelitis*. The meninges are thickened and adherent, while the spinal and meningeal arteries and veins show obliterative changes, and are surrounded or infiltrated by small round cells. The same changes are apparent in and around the vessels and pial septa within the cord. In severe cases, the membranes are united to form a thick fibrous sheath around the cord, and the outlines of the nervous structures as seen in transverse section are almost obliterated by the presence of numerous small gummata or of myriads of small round cells. The nervous elements are compressed by the cell infiltration, or undergo softening or necrosis as a result of obliteration of the blood vessels. Meningo-myelitis is usually confined to a narrow area in the dorsal region, and is often associated with a more extensive meningitis.

In some cases with severe arterial changes, extensive softening results from thrombosis or hæmorrhage and produces severe paralysis of sudden onset—*acute syphilitic myelitis*.

Large gummata are rare. They occur in the cord, or on its surface, and produce the signs of compression.

In another rare form in which the membranes alone are affected—*pachymeningitis* and *leptomeningitis*—the dura and pia-arachnoid unite to form a thick fibro-gummatous sheath around the cord to which they become adherent.

Syphilitic disease of the vertebral column is very rare. It produces changes which resemble those of tuberculous spinal caries, with osteitis and periostitis, and the formation of granulation tissue and gummata on the outer surface of the dura. Necrosis of the bones may lead to deformity of the spine. It is seen most often in the cervical region, where it begins in the spine, or spreads from a syphilitic ulcer in the throat.

Symptoms.—In *meningo-myelitis*, pain in the back, tenderness of the spine, and radiating pains or a feeling of constriction in the limb or around the trunk, are often present in the premonitory stage. After these have lasted several days or weeks, cord symptoms appear. They may come on rapidly, or very slowly. Often the first complaint is of numbness or tingling in the lower limbs, or of weakness or stiffness after exertion. Several attacks of temporary weakness may precede severe paralysis, and in different cases every degree is seen, from slight stiffness to complete paraplegia. When the paralysis comes on slowly the lower limbs become spastic, the knee- and ankle-jerks are exaggerated, and the plantar response is “extensor.” In severe acute cases the limbs are flaccid and the tendon reflexes are at first diminished or lost, spasticity and increased reflexes developing later. In both forms the abdominal reflexes are diminished or lost below the level of the lesion. In most cases bladder control is impaired, and in some this is the first symptom. Sensory troubles may be slight when the paralysis is severe. Numbness and tingling are common, and some objective loss can usually be detected, the temperature sense, especially for cold, and the sense of vibration being most often at fault.

In one group of cases—*Erb's syphilitic spinal paralysis*—spastic weakness develops slowly in the lower limbs, without meningeal symptoms. The bladder is usually affected, and sensory loss is slight. In this form the spinal disease appears later after infection than is usual in other forms of *meningo-myelitis*.

Pachymeningitis (diffuse gumma of the theca) as an isolated disease is usually found in the cervical region. The earliest symptom is pain in the neck, radiating down the upper limbs and between the shoulders. After a time, usually several months, weakness, wasting and loss of sensation appear in the arms. Still later, spastic paraplegia may develop from compression of the cord. When the lumbar region is attacked, the same symptoms appear in the lower limbs.

In *syphilitic caries* tenderness over the diseased bones, pain on movement of the spine, and radiating pain in the distribution of the sensory roots at the level of the lesion, are the chief symptoms. When the cord is compressed, power and sensation are diminished in the parts below.

Diagnosis.—Spinal syphilis appears in many clinical forms and often resembles other diseases. Hence it must be considered in every case of spinal disease without an obvious cause. The diagnosis may be founded on—(1) a history of syphilitic infection; (2) the presence of syphilitic lesions in other parts of the body; (3) Wassermann's reaction in the blood or pathological changes in the *cerebro-spinal fluid* (see pp. 1629, 1630); (4) signs of associated cerebral syphilis; (5) rapid improvement under treatment by specific remedies.

Prognosis.—The outlook for recovery of power is good when the meninges only are attacked, but bad when the symptoms are due to softening, hæmorrhage, atrophy of motor cells, or tract degenerations, for these are secondary changes and are not influenced by anti-syphilitic remedies. In a given case, however, it is not possible to assess accurately the amount of damage sustained by different structures, and the prognosis is always doubtful. Complete recovery occurs in about one-third of the cases of slight or moderately severe *meningo-myelitis*. The majority make a partial recovery, and are able to

walk fairly well, in spite of the weakness and stiffness which remain. In cases with a sudden onset of severe paralysis, the prognosis is very bad, and death from bedsores or bladder and kidney infection is the usual result. On the other hand, the outlook for recovery of power is better when slight troubles come on rapidly than when they develop extremely slowly, as in Erb's form of the disease, in which many years pass before the paralysis becomes severe; for in the first case the lesions are interstitial and they respond well to treatment, whereas parenchymatous degeneration of the nervous elements themselves is present in the latter, and treatment is of less avail. The prognosis in early cases is determined by the effects of anti-syphilitic treatment. In cases of long standing, the most that can be expected is that appropriate treatment will arrest the course of the disease.

Treatment of Cerebro-spinal Syphilis.—As soon as the diagnosis is made, vigorous anti-syphilitic treatment should begin. The drugs used are mercury, bismuth, the arsphenamine derivatives and potassium iodide. In the opinion of some authorities mercury still holds pride of place in the treatment of neurosyphilis, especially when administered by inunction. Treatment will vary according to the acuteness and nature of the lesion present.

In *acute syphilitic meningitis*, unless the condition is grave, it is best to begin with mercurial inunction (60 grains of the ung. hydrarg., B.P., well rubbed in) for the first week, and then to give a course of neoarsphenamine (0.6 g.), at five or six day intervals, until 6 or 8 injections have been given. Simultaneously with this, intramuscular injections of a bismuth preparation (salicylate, or iodobismuthate of quinine, each dose containing 3 grains of bismuth) are employed. Subsequently, courses of bismuth or mercury and of arsenic may be given alternately until examination of the cerebro-spinal fluid shows cessation of the active disease.

In *chronic syphilitic meningitis* treatment may be started with inunctions of mercury or injections of bismuth, followed by a course of arsenical injections. A full course of mercurial inunctions consists of sixty inunctions if signs of mercurialism do not develop. Potassium iodide in large doses may also be given. Treatment must at the same time be correlated from time to time with cerebro-spinal fluid examinations.

In *vascular syphilis* also treatment is best begun with either mercury or bismuth, and arsenic not given for the first two weeks. Iodides should also be administered.

Massage and passive movements should be carried out daily, when the limbs are weak. When sensory loss is present, careful nursing is necessary to prevent the formation of bedsores; and when control of the bladder is defective, the usual precautions must be taken for the prevention of infection of the urinary tract.

GENERAL PARALYSIS OF THE INSANE

Synonym.—Dementia Paralytica.

Ætiology.—As in tabes, the essential factor in the causation of general paralysis is previous syphilitic infection. Males are affected much more frequently than females. The onset is commonest between the ages of 30 and 50 years, from 10 to 20 years after infection. As a result of congenital

syphilis or of early innocent infection, it may appear in childhood, youth or even adult life. It has been estimated that about 5 per cent. of *syphilitics* develop general paralysis, but the incidence in those who have been well treated in the early stages is not more than 1 per cent. The incidence varies in different races, and seems to be greater with increasing civilisation. It is more apt to occur in town and city dwellers, and in those who have led a strenuous intellectual or business life.

Pathology.—The skull-cap is thickened, especially in its anterior part, its density is increased, and the diploë is obliterated. The dura mater is thickened and adherent to the skull, and may show the changes of *pachymeningitis hæmorrhagica*. The arachnoid is tough and thick, and white lines are seen between the sulci and along the vessels. The pia is thickened, its meshes are distended by pale yellow fluid, and on attempting to strip it off portions of the cortex are torn away. The amount of cerebrospinal fluid is increased. The brain looks wasted and shrunken, and its weight is abnormally low. The sulci are wide and the convolutions are narrow. The ventricles are dilated, and their ependymal lining presents a granular or a frosted appearance. On section the grey matter of the cortex is seen to be thinner than normal—decortication. On histological examination gross changes are found in the membranes, in the blood vessels, in the neuroglia, and in the true nervous elements. The earliest changes are found in the cortical vessels and membranes. Nuclear proliferation occurs in the walls of the smallest pial vessels and in the perivascular lymphatics. There is overgrowth of the endothelial cells in the capillary walls, and around them lie peculiar cells—the so-called plasma cells. Small lymphocyte-like cells, mast cells, and many others of doubtful nature appear in and around the perivascular channels, and by blocking them interfere with the nutrition of the cortex. The larger vessels also show proliferation of the endothelium, degeneration of the muscular coat and perivascular infiltration. The spirochætes are readily demonstrated in the brain substance.

The fibres of the neuroglia proliferate, its cells multiply, and some assume an abnormal size or shape. These changes in the interstitial tissues are followed by degeneration of the cortical cells and atrophy of their processes. The changes are most marked in the cortical cells and association fibres of the anterior part of the cerebrum, but similar degenerations are found in the basal ganglia, the cerebellum, the brain stem and the spinal cord.

Symptoms.—The disease is characterised by progressive deterioration of the mental and physical powers.

MENTAL SYMPTOMS.—The most recent acquisitions are usually lost first. Hence the earliest sign of mental failure will differ according to the intellectual and emotional make-up of the individual attacked. Memory, judgment and reasoning are impaired from the first, the æsthetic, moral and intellectual attributes alter early, and changes occur in the domains of conduct and emotion which astonish or distress the patient's friends. To one who sees the patient for the first time the defects may not be apparent, but those who know him will speak of the *changes* in his intellectual capacity, character or behaviour. Cheerfulness has given place to depression or irritability. the quiet and unassuming man has become passionate and boastful, the good father has turned against his family, promises are no longer kept, a good

business is neglected, money is spent unwisely, high artistic skill is lost, the moral code is transgressed, and so on. There is no end to the variety of the early symptoms, but in each case they represent a change for the worse.

In the classical form of the disease, elation and expansive delusions concerning health, wealth, social position or physical and athletic powers are prominent, but in a larger number of cases the patients are depressed in the early stages, and the delusions when they appear may be melancholic or hypochondriacal. Unlike the paranoiac, who may refuse to disclose his delusions or who may reason skilfully from his false premises, the parietic reveals his delusions readily, and can be made to betray their falseness by his own words. To the direct question regarding his occupation the Emperor, the possessor of untold wealth, the world's greatest general, will reply unconcernedly that he is a boot-black, or the champion athlete of the universe will give answers showing a complete absence of familiarity with any branch of sport.

In the course of time dementia increases, memory is abolished, delusions are forgotten, emotion disappears, and in a year or two the patient is unable to move from his bed where he lies speechless, paralysed and incontinent. The various concomitants of the delusions are described in the following paragraphs.

The most constant and most characteristic signs are changes in the pupils, tremors of the face, tongue and hands, and disorders of speech. Changes in the pupils occur early, indeed they are often present as signs of past syphilis before symptoms of general paralysis appear. Inequality in size, irregularity in outline, and the complete or incomplete Argyll Robertson phenomenon, are very common pupillary signs. Primary optic atrophy is frequent, but except in tabetic cases it is rarely complete. Paralysis of the external ocular muscles sometimes occurs as a result of associated tabes or cerebral syphilis. Tremor is often an early sign. In the face and hands, though often present when the parts are at rest, it is best seen in speaking or when movements, such as showing the teeth and holding the arms outstretched, are carried out to order. The typical tongue tremor is a backward and forward "trombone" movement of the organ, when the attempt is made to protrude it. Speech is often affected early. At first it is merely hesitant. Later it becomes blurred, syllables are omitted, interpolated or slurred, and the voice becomes feeble and lacks intonation. As the memory fails, confusion arises in the construction of long sentences, proper names are forgotten, the choice of adjectives and verbs becomes more and more limited, and the vocabulary diminishes until only interjections are left. Written language suffers in the same way, and may show defects of execution and of ideation before spoken speech is noticeably altered. At the onset voluntary power in the muscles is usually maintained, but undue fatigue after moderate exertion is a common early symptom. As the disease progresses, weakness appears in the lower limbs and soon affects all the muscles. Some of the signs of injury to the pyramidal tracts, such as increased tendon reflexes, diminution or loss of the skin reflexes, and Babinski's plantar response, are found sooner or later in almost every case. In a small number the tendon reflexes are abolished and other signs of tabes, such as sensory disturbances, are present. Retention of urine or incontinence sometimes

occur in the early stages. Towards the end, control of the bladder and rectum is always lost.

Epileptiform seizures of various kinds are common. They may be the first obtrusive symptom, and may occur at any time in the course of the disease. The attacks may have all the aspects of idiopathic epilepsy, or they may be local and of the nature of Jacksonian fits. Attacks resembling *petit mal* have also been observed. In the so-called "congestive" apoplectic attacks, paralysis of one limb or of one side of the body comes on suddenly with or without convulsions and passes off in a few days or weeks. The patient may become comatose and breathe stertorously, or he may be merely somnolent or confused.

Insomnia is frequent in the prodromal period, but in the early stages sleep is often excessive. Later, sleeplessness and motor restlessness are often troublesome symptoms.

CLINICAL TYPES.—*Exalted or expansive form.*—This form includes the cases in which elation, euphoria and grandiose ideas are prominent.

Demented forms.—The patients often seek advice of their own accord, complaining of diminished mental and physical power or failing memory. Mental deterioration runs its course without marked depression or exaltation.

Depressed form.—This common form is characterised by melancholic and hypochondriacal delusions. Some have delusions of persecution. Very often the patient exaggerates his afflictions to a degree not seen in other forms of insanity, melancholic megalomania. Remissions are common.

Maniacal form.—The features of this form are attacks of acute maniacal excitement, which may resemble acute delirious mania. Remissions are common, and apparent complete recovery may be made; but the attacks recur, each one leaving the patient more demented.

Many other varieties have been described, the stuporose, the convulsive, the tabo-paretic, and so on. The characters of these types are sufficiently indicated by the names.

Diagnosis.—It is upon the detection of character alterations in the patient that the early diagnosis of general paralysis commonly depends. As has been said, these alterations vary from subject to subject, but constant components are impairment of judgment, defects of memory, and signs of impaired emotional control. Emotional tone may be one of depression, or one of exaltation, and while the latter is the more striking it is probable that the former is the more frequently encountered. It is the patient's family and his fellow-workers who first notice the insidiously developing change, and their evidence may be essential to early diagnosis. It is, however, important to remember that an altered emotional tone is invariably accompanied by some indications of mental deterioration, and this latter component serves in the differentiation of the psychoneuroses from general paralysis. Commonly the patient himself has little or no insight into his altered state, and may express his subjective sense of well-being and of intellectual acuity in glowing terms that arouse a suspicion of the true state of affairs in the trained observer.

Accompanying these essential alterations are a number of somatic physical signs, such as a slurring articulation, some tremor of the lips and tongue, perhaps some tremor of the hands. Pupillary anomalies are almost constant,

but fall short of those seen in *tabes dorsalis*. Thus, myosis is uncommon, but irregularity of outline, inequality and sluggishness of the light reaction are usual. There may or may not be changes in the tendon reflexes and plantar responses, but diagnosis in the initial stages of the malady has often to be made in the absence of many of these somatic signs. In doubtful cases confirmation must be sought, and can be obtained from the examination of the cerebrospinal fluid and from the Wassermann reaction (see pp. 1629, 1630).

Course and Prognosis.—After an insidious onset, the disease progresses steadily and usually ends fatally in about 3 years. Acute forms may run their course in a few weeks. When convulsions are frequent, death usually results in about 6 months. When periods of extreme restlessness and excitement alternate with depression, one year is the average duration. In simple demented and depressed forms, the duration is usually about 3 years. The course is most prolonged in those who have attacks of wild excitement or mania, as remissions are very common in this form. During the remissions, patients may be able to return to work, and 10 years may elapse between the first attack and the fatal termination. The course is often prolonged in women, in congenital cases, and in cases of tabo-paresis.

Treatment.—As some cases of cerebral syphilis simulate general paralysis, in every instance where the latter disease is suspected rigorous anti-syphilitic treatment should be tried in the hope that the patient is suffering from the more curable condition.

Malaria therapy in general paralysis.—Encouraging results have been obtained by infecting paralytics with benign tertian malaria. Blood is obtained from a patient suffering from malaria (not necessarily during a rigor), and is injected intramuscularly, or infected mosquitoes may be applied to the skin in a wide-mouthed jar, the orifice of which is covered with muslin. The incubation period may be as long as a fortnight or more after subcutaneous injection, but failure to infect with 5 c.c. of blood is rare. If the blood has to be transported it should be received in a sterile test-tube containing glass beads; after defibrination it is transferred to another sterile tube and packed in ice, when it will remain active for 6 hours or more. The recipient is allowed to have six, ten, or more rigors, the number depending on his general condition during the treatment; the infection can be cut short at any moment with quinine; relapses rarely occur in this experimental malaria. In favourable cases a remission occurs, improvement continuing for several months; the end result of the treatment cannot be assessed until 6 months or more have elapsed. When the patient has recovered from the debilitating effects of the malarial infection, anti-syphilitic treatment should be initiated. For this purpose tryparsamide has been successfully employed, after an initial injection of 1 g., a course of weekly injections of 2 g. may be carried out—15 injections being given. This drug has been known to cause optic atrophy and therefore the visual function must be carefully watched during its administration.

Malarial therapy is not without risk and should be employed only by those experienced in its use, and under institutional conditions where adequate nursing is obtainable. Malarial therapy owes such efficacy as it possesses to the severe and recurrent pyrexia it produces, which is very destructive of spirochaetes in the brain. Recently attempts have been made to bring

about a more readily regulated hyper-pyrexia by mechanical means, such as prolonged retention in electrically heated cabinets. This procedure, not less than malarial therapy, calls for considerable experience and the requisite apparatus, and even on these terms it cannot be said to be free from danger.

TABES DORSALIS

Synonym.—Locomotor Ataxia.

Ætiology.—Tabes is more frequent in men than in women (10 to 1), and begins most often between the ages of 30 and 45. The essential factor in its causation is previous syphilitic infection. The interval between infection and the onset of symptoms varies from 2 to 20 years; commonly from 5 to 10 years. As a result of congenital syphilis, or of infection in infancy, it sometimes begins in childhood, youth, or early adult life—infantile or juvenile tabes. Occasionally husband and wife are both affected—conjugal tabes.

Pathology.—Despite what has been said earlier (p. 1630) of the essential lesion in syphilis of the nervous system, it has to be admitted that all we know with certainty of the lesion in tabes is that there is degeneration of exogenous fibres in the posterior columns of the cord. At various times it has been suggested that the primary lesion is a form of local meningitis of the posterior roots proximal to the posterior root ganglion. Recently, Richter has described a peculiar type of granulation tissue in this situation, and he believes that this “strangles” the entering nerve fibres, and thus leads to their secondary degeneration. But Stern has shown that this tissue is present in the normal subject, and therefore the hypothesis of a primary local meningeal lesion remains unsubstantiated. It is known that toxins may ascend afferent nerve fibres from a peripheral focus of infection, and that degeneration of exogenous nerve fibres *within* the cord may result, the peripheral portions of the nerve fibre showing no lesion. It has been suggested that the toxins of syphilis may thus ascend from a peripheral focus, and that the essential and primary tabetic cord lesion is thus a degeneration of nerve fibres within the cord. Some of the degenerated fibres end around cells in the grey matter soon after they enter the cord, while all the fibres with a long intraspinal course enter the posterior columns, and ascend in them to the nuclei of Goll and Burdach in the medulla. As a secondary change the neuroglia around the degenerated fibres increases in amount and density. Hence the characteristic feature in sections of the cord in tabes is sclerosis of the posterior columns. The sclerosis usually appears earliest in the postero-lateral columns of the lower lumbar and upper sacral regions. In the dorsal and cervical cord it is confined at first to the postero-internal columns, which contain the degenerated fibres from the lumbar and sacral regions, but in advanced cases when the dorsal and cervical sensory roots are also affected the posterior columns are sclerosed throughout.

In advanced cases the endogenous tracts of the posterior columns show degeneration, and in some the afferent tracts in the lateral columns are also affected. In sections stained by the Weigert-Pal method the diseased areas are paler than the rest of the white matter. By the Marchi method parts containing recently degenerated fibres show numerous black dots, which represent fatty material in the degenerating myelin sheaths.

In cases of long standing, atrophy of the entire sensory root with degeneration in the peripheral parts of the sensory nerves, and atrophy of cells in the ganglia are frequent findings. The ocular palsies of tabes are probably mainly due to gummatous meningitis, but it is probable that in the case of the third nerve there may be a degeneration of the nerve cells in the nucleus. Tabetic optic atrophy is also the result of a combined interstitial gummatous inflammation and primary degeneration of optic nerve fibres.

Symptoms.—The inadequacy of current descriptions of the clinical manifestations of tabes is shown by failure on the part of those who depend upon them for guidance to diagnose the disease before it has reached an advanced stage. So long as tabes is described as a disease characterised by severe lightning pains, absent knee-jerks and Argyll Robertson pupils; so long as the diagnosis is withheld until these symptoms are found together; so long as patients without ataxy are stated to be in the *early* or *preataxic* state—just so long will valuable years be wasted, as they are at present, before patients receive treatment at a time when it might be expected to arrest the course of the disease. It is true that these important symptoms appear ultimately in a very large proportion of the cases, and they are often present when the patient is seen for the first time. It is equally true, however, that many of these patients have complained of symptoms which, if they had been appreciated by the physician, would have betrayed the disease many years before, and that throughout these years they have presented physical signs which, although the knee-jerks and pupillary reactions were still present, would have made the diagnosis of tabes certain. In the following paragraphs stress will be laid on the signs that appear early and allow the diagnosis to be made at the onset of the disease. Chief amongst these early symptoms are disturbances pointing to interference with the functions of the posterior nerve roots.

SENSORY DISTURBANCES.—*Subjective.*—Following a general law the first manifestations of altered function are subjective—the patient complains of sensory troubles before any changes can be discovered by objective examination. The most important of these subjective troubles in tabes are the so-called lightning pains. These pains merit the closest attention. They are rarely absent, they often precede other symptoms by 5 or 10 or more years, and most important of all they possess peculiar features which render them pathognomonic of tabes and allow the diagnosis to be made in a syphilitic on their presence alone. Although they are rarely absent careful interrogation may be needed to disclose them. To the question, "Have you had any pains?" the patient may answer "No." If then he is asked if he has rheumatism, he will often answer "Yes," and proceed to give an account of characteristic tabetic pains of several years' duration. In other cases the patient mentions his pains, but their significance escapes notice because it is thought that they are too slight for tabetic pains. It must be made clear at once therefore that the peculiarity of the pains in tabes does not lie in their severity, for they vary from a trifling sensation of discomfort to almost intolerable agony, but in their distribution, in their direction of propagation and especially in their arrangement in time.

As a rule, they come on in attacks, in which single momentary pains are repeated at intervals of a few seconds or minutes for several hours, the whole bout lasting several days or weeks. Between the attacks there may

be long intervals of complete freedom from pain. The pains are felt most often in the lower limbs, but any part may be affected. They may be referred to the skin, to the muscles or to the bones. They are very common in the bony prominences around the knee and on the foot. The direction of radiation varies. In some the pain seems to shoot up or down a limb, but in a larger number it seems to strike the limb vertically as if a sharp object were piercing it from without. Some patients experience both kinds of pains. The onset of each pain is always sudden. If it is severe the patient may cry out, and if it overtakes him whilst walking he is forced to stop and he may fall. The duration of each pain is usually momentary, but sometimes it lasts a second or two and fades away gradually. During a given bout the pains usually recur in the same place each time for several hours on end, and then appear in another part, say on the following day. In a few cases, however, they confine themselves to two or three points, now appearing in one and now in the other. In a still smaller number the site varies from moment to moment, so that the patient never knows where the next one will strike him. In one group the pains are repeated very rapidly in one place for a few seconds, and then after an interval in another, so that the timing recalls the sound of a machine-gun firing short bursts—tap, tap, tap, pause, tap, tap, tap, pause, and so on with a longer interval now and then during which the gun is trained on a new objective.

After a bout the skin is often tender, and ecchymoses may appear over parts in which the pains were felt, though this is exceptional. Cold, changes in the weather, anxiety and especially over-exertion make the pains worse. They are often more severe for a day or two after treatment by intravenous or intrathecal injection of specific remedies. Other pains with characters which are not peculiar to tabes are common. They are described as aching, burning or gnawing pains. Like the lightning pains, they alter with changes in the weather and are usually attributed to rheumatism. Other common subjective sensory symptoms are "pins and needles" in the extremities, a feeling of walking on a soft substance, and of constriction around the trunk or limbs. More important than these, because it often appears very early, is hyperæsthesia of the trunk, especially in its lower part. Light touches or applications of water at certain temperatures are almost unbearable.

Objective sensory disturbances.—Signs of damage to the posterior nerve roots appear in the earliest stages of the disease, and are demonstrable in many cases long before the classical signs appear. The detection of this early sensory loss is of great importance, for its distribution is pathognomonic. The parts in which sensation is first impaired are—(1) a band on the chest and along the inner border of the arms; (2) the feet; (3) around the anus; (4) on the nose.

As the disease advances, sensory loss extends upwards from the feet, downwards from the chest, and outwards from the nose and anus in concentric circles. Ultimately these areas coalesce, and in the later stages sensation is diminished all over the body. All forms of skin sensation are not equally affected. Sometimes the defect is first discovered on testing with light tactile stimuli, but more often pain and temperature are first impaired.

The senses of deep pain and of position and passive movement, as well as the vibration sense, are often diminished in the legs in the early stages. In advanced cases these defects are present in all the limbs.

In cervical tabes sensory disturbances occur first, and are most severe in the arms.

In severe cases sensation of all kinds may be almost completely abolished. No cutaneous stimuli are felt and the deep structures are insensitive to pain. To this is added loss of the sense of position, not only in the limbs but also in the trunk, so that the patient is unaware of their position when his eyes are closed. If he sits up with the arms outstretched, on closing his eyes the arms "wander," the fingers execute slow "piano-playing" movements, and the body sways. In extreme cases the patient falls on his side as soon as the eyes are closed.

MUSCULAR HYPOTONIA AND THE TENDON REFLEXES.—Loss of muscle tone occurs in lesions of various parts of the nervous system, and is not necessarily accompanied by changes in the reflexes in the limbs, but when it results from interruption of the spinal reflex arc the two signs are found together. Hence in tabes, where the afferent limb of the reflex arc is the first structure affected, hypotonia and diminution of the tendon reflexes are characteristic signs. The decrease in the tone of the muscles is often well marked when lightning pains are the only symptom of tabes, and loss of skin sensation the only other sign. It is shown by flaccidity of the muscles, and by an abnormal range of active and passive movement of the limbs.

The leg can often be raised to an angle of 100° from the horizontal, with the knee extended, whereas a normal person cannot raise it more than 60° , and excessive range of dorsiflexion of the foot is often a striking sign. In extreme cases the legs can be made to encircle the neck, the body can be flexed so that the head touches the bed between the knees, and the patient is able to imitate the tricks of the "double-jointed" man.

The knee-jerks are very often absent when the patient is first examined, and in the later stages they are almost always lost, but compared with the signs already mentioned this one is of late onset, and may be missing even in the ataxic stage. The position formerly held by the knee-jerks in the symptomatology of tabes should be given to the tendo Achillis (ankle) jerks. Loss of the ankle-jerks is indeed an early sign in tabes, for it often precedes loss of the knee-jerks by many years. The tendon reflexes in the upper limbs are lost early in cervical tabes, and are frequently absent in cases of the ordinary type.

The skin reflexes are often exaggerated to a degree rarely met with in other diseases. This is best seen on the abdomen, and is usually associated with hyperæsthesia to touch and temperature. Later, when the tactile sense is lost, the skin reflexes are often diminished. The plantar reflex is usually normal. It is sometimes absent when sensory loss on the soles is severe, and in cases where sclerosis of the pyramidal tracts exists as a complication of tabes the response is "extensor."

ATAXIA.—The fibres conveying those afferent impressions which are essential for the proper execution of voluntary movements, are more resistant than those with other functions, and inco-ordination, though extremely characteristic of tabes, is usually a late symptom, or it may be absent throughout the whole course of the disease. Its onset is marked by unsteadiness in walking and difficulty in maintaining the balance of the body. These troubles are first noticed when co-ordinated movements are performed without the aid of vision. As the defect increases unsteadiness appears even

with visual guidance. To maintain their balance the patients walk on a wide base with the eyes directed to the ground. At a later stage some raise the feet too high, throw them too far forward and bring them down forcibly, the whole sole striking the ground at once—stamping gait. Others reel from side to side like drunken men. Still later the support of one or two walking-sticks is required, and ultimately walking becomes impossible. The inco-ordination is not only present in walking, but can be seen in all voluntary movements, *e.g.* in the heel to knee test. The same defects occur in the upper limbs. At first there is merely clumsiness in performing fine movements such as picking up small objects and in adjusting the dress. In the end the ataxia may become so great that the patient is unable to feed himself.

By appropriate tests inco-ordination can usually be disclosed before the patient has noticed it. Some of the tests are: standing with the heels and toes together, standing on one foot, walking backwards, rising quickly from a stooping position and turning quickly in walking. In each instance the unsteadiness is greatest when the eyes are closed and when the feet are bare.

SPRINTER TROUBLES.—These are the result of the lowering of pain sensibility in the bladder which is the afferent element in the reflex of micturition. An increased distension of the bladder becomes essential before the act can be started, and this fails before the bladder is completely emptied, and residual urine is present in slowly increasing quantity. Though this causes little or no inconvenience to the patient it often leads to cystitis and renal complications. Difficulty in starting micturition and nocturnal incontinence are the common complaints. Complete retention and paralytic incontinence are rare, and when retention occurs it has in our experience been due almost invariably to enlargement of the prostate, the removal of which has been well borne and has given complete relief. Sexual desire and power are usually lost early in the course of the disease.

OCULAR SYMPTOMS.—Changes in the reaction of the pupils and in their size and form are very frequent and are of great importance for diagnosis. The chief of these is the Argyll Robertson phenomenon, in which the pupil contracts on accommodation but not when exposed to light. This sign appears in both eyes in 70 per cent. of cases, and is one of the earliest to appear. It is sometimes found in one eye with a normal or diminished reflex in the other. It may be present in an incomplete form, the contraction to light being slight and sluggish when the reaction to accommodation is brisk, or the pupils may contract when first exposed to the light only to dilate again. Occasionally the pupils are fixed and do not react to either stimulus. In rare cases the reaction to accommodation is lost while the light reflex persists.

The size of the pupils varies greatly in different cases. Most often they are small, but pupils of moderate size are common, and sometimes they are widely dilated, though this is very exceptional and usually associated with optic atrophy. It is not unusual to see pupils which, when contracted on accommodation, are no larger than the head of a pin, but the "pin-point" pupils are extremely rare. Inequality of the pupils or irregularity in their outline is present in most cases. It is said that the pupils are sometimes normal in every respect even in the advanced stages of the disease. On the other hand, in old tabetics in whom the disease has been present for

very many years the pupils may be found wholly inactive both to light and on convergence.

External ocular muscles.—In the early stages transient palsies of the muscles of the eyeball often cause ptosis, diplopia or squint, lasting a few days. Permanent paralysis may come on at any time, but is most frequent in the later stages. A persistent drooping of the eyelids—tabetic ptosis—is a common sign. This is attributable to a lesion of sympathetic fibres. The patient tries to overcome the defect by contracting the frontalis muscles, and the wrinkling of the forehead with slight drooping of the lids gives the patient an expression—the tabetic facies—by which the disease may be recognised at a glance.

Optic atrophy.—Defective vision from atrophy of the optic nerve is often the symptom for which the patient first seeks relief. It occurs in about one case in ten, and almost without exception ends in complete blindness. The loss usually begins in the periphery of the visual field, and is often unnoticed until central vision begins to fail. Occasionally central vision fails early. At first one eye suffers more than the other, but ultimately, after a period which averages 5 years, all vision is lost in both. Patients sometimes relate that their blindness came on suddenly, or in a few hours or days. In these cases optic atrophy has been present for a long time, but the fibres subserving central vision have escaped until the last. On the other hand, vision may fail very slowly, with periods of arrest or apparent improvement, and total blindness is sometimes delayed for 10 or 15 years. The atrophy is primary, that is, it is not preceded by papilloedema. Pallor appears first on the temporal side, whence it spreads over the whole disk. The edges of the disk are sharply defined and the lamina cribrosa is visible as slightly darker spots, so that the disk stands out clear and bright, like a full moon. When optic atrophy is the first symptom, it is often impossible to detect any incoordination in the lower limbs, and ataxia may be long delayed. The knee-jerks are often brisk, but some of the early signs—lightning pains, sensory disturbances or loss of one or both ankle-jerks—are almost always present, and the Argyll Robertson pupil is a constant accompanying sign. In a number of the patients with optic atrophy the signs of general paralysis are added to those of tabes (tabo-paresis), and the course of their illness is that of the more serious disease.

OTHER CRANIAL NERVES.—The senses of smell and taste are sometimes lost. Vertigo, tinnitus and nerve deafness are common. Lightning pains are often severe in the distribution of the trigeminal nerve, and loss of sensation on the nose, especially to pain, is one of the earliest and most frequent signs. Paralysis of the vocal cords, though rarely sought for, is present in many cases.

VISCERAL CRISES.—There are two varieties of visceral crises which are associated with disturbance of the parasympathetic and with the sympathetic innervation respectively. The former, which is confined to the vagus distribution, consists of spontaneous sensory irritation and its reflex results which is never painful since the vagus contains no pain-conducting elements. The examples are the laryngeal crisis and the gastric crisis, which comprises painless vomiting. The latter belongs to the sympathetic distribution, always involves severe pain, and is made up of the painful gastric crises, and the rectal and vesical crises.

Gastric crises.—The organ most subject to crises is the stomach. Attacks of severe abdominal pain with repeated vomiting come on suddenly. They last a few days, or a week or two, and are often repeated every few weeks for long periods. Sometimes pain or vomiting alone is present. There is always complete anorexia. The patient looks very ill during the attack, but it is never fatal. They often occur before other symptoms of tabes appear, and are often mistaken for acute obstruction, and other conditions requiring urgent surgical treatment, but careful examination will rarely fail to reveal indubitable signs of tabes. If attention were paid to the ankle-jerks, and to sensory disturbances, instead of to the knee-jerks, unnecessary operations would be less frequent.

Next to the stomach, crises are most frequent in the larynx (*laryngeal crises*). In the commonest form there is spasm of the larynx, with noisy breathing, cough and dyspnoea. Sometimes the attacks resemble whooping-cough or laryngismus stridulus. They are much shorter than gastric crises, rarely lasting more than an hour. Death in an attack is extremely rare.

Attacks of extremely painful and prolonged tenesmus (rectal crises) are not uncommon. Attacks of frequent painful micturition (vesical crises) and of pain like renal colic (renal crises) are rare.

Cardiac, nasal, bronchial, intestinal and other crises have been described.

VASOMOTOR AND TROPHIC DISTURBANCES.—The most important of these are changes in the joints and perforating ulcers. Rarer forms are local sweating, loss of hair, nails or teeth, attacks of herpes, hæmorrhages into the skin, necrosis, rarefaction and spontaneous fracture of bones, excessive callus formation and spontaneous rupture of tendons.

Charcot's joint disease.—Arthropathies may develop at any stage of the disease. Occasionally the patient seeks advice for the first time with this complaint. The first sign is usually rapid swelling in and around a joint, with effusion and œdema. The effusion, in slight cases, subsides slowly and the joint recovers, but more often the enlargement is followed by destruction of the cartilages, wasting of the ends of the bones, peri-articular new-bone formation and destruction of the ligaments. The joint becomes disorganised, the range of movement is increased, and crepitations of startling coarseness are heard and felt when the part is handled. The characteristic feature is the complete absence of pain. Dislocations occur readily, especially at the hip. The diseased joint sometimes becomes infected. This is commonest in the foot. The joints most often attacked are, in order of frequency: knee, hip, shoulder, elbow, ankle, small joints of the hands and feet, the spine.

Perforating ulcers are commonest on the sole of the foot. Patches of hard thickened skin are frequently seen on the soles of the feet. Sometimes blisters form beneath this thick epidermis, and on bursting leave an indolent sore. Once formed the ulcer is very indolent. It is usually painless.

Complications.—Tabes is frequently complicated by other syphilitic affections of the nervous system, of which the commonest and most important is general paralysis of the insane. Some tabetics develop general paralysis, and many paretics present some of the signs of tabes. Indeed, these conditions are merely different aspects of the same disease, and are named according to the predominant features. Sometimes it is difficult to decide the category of given cases, and the name *tabo-paresis* is used to describe them. At the same time it may be observed that *tabo-paresis*

runs a much slower course than uncomplicated general paralysis, and apart from any question of treatment the survival period is longer in the former than in the latter. Occasionally the pyramidal tracts degenerate and signs of spastic paraplegia are added to those of tabes. Atrophy of the anterior nerve roots with consequent wasting of the corresponding muscles is a fairly common complication. Outside the nervous system the commonest complications are aortitis, aortic regurgitation and aneurysm.

Diagnosis.—Most tabetics come under observation for the first time when one of the many symptoms of the disease begins to cause serious trouble. The obtrusive symptom may be: lightning pains, failing vision from optic atrophy, double vision from paresis of ocular muscles, attacks of vomiting, tenesmus, unsteadiness in walking, painless joint disease, impotence, troubles with micturition, or some other less common complaint. In these the diagnosis rarely causes difficulty. A history of characteristic pains, or evidence of syphilis in the past, justifies the diagnosis of tabes on the symptoms alone. In almost all of these cases, moreover, unequivocal signs will be found which make the diagnosis certain. Two signs—the Argyll Robertson pupil and absence of the ankle-jerks or knee-jerks—are of supreme importance, for although one is often lacking, the absence of both in the kind of case we are discussing is rare. To one or both of these several of the following confirmatory signs are usually added: inequality or irregularity of the pupils, diminished sensibility to pinprick of the skin on the nose, on the chest and feet, absence of pain on compressing the calf muscles, loss of vibration sense in the feet, muscular hypotonia, defective sense of position in the limbs and unsteadiness when the eyes are closed.

When the symptoms and signs are slight and few, or when suspicious signs are found during a routine examination, the diagnosis is sometimes difficult, and may require for its elucidation a careful inquiry into the history, an examination of the blood and cerebro-spinal fluid, and a meticulous investigation of the nervous system. These cases are discussed in the following paragraphs.

THE DIAGNOSIS OF EARLY TABES.—1. Since Westphal, some 60 years ago, described loss of the knee-jerks as an early sign of tabes, and established the existence of the pre-ataxic stage, the profession, apart from neurologists, has altered its views but little, and still hesitates to diagnose tabes while the knee-jerks are present. Consequently mistakes in diagnosis are common. The diagnosis can and should be made when lightning pains are the only symptom. Pains with the characters already described occur in no other disease, and their presence calls for a careful investigation for evidence of past syphilis. In this first stage of tabes the diagnosis is founded on (1) characteristic pains; (2) evidence of syphilis in the past, obtained from the history or by examination of the blood and cerebro-spinal fluid (see pp. 1629, 1630).

2. Only rarely need the diagnosis be made on these grounds alone, for in almost every patient with lightning pains careful examination will reveal confirmatory signs. The most important of these are sensory disturbances and alteration in the pupils. Hyperæsthesia to touch and temperature on the lower part of the trunk is very common, although few patients mention it until their memory is refreshed by careful interrogation. In a patient who has had syphilis and suffers from lightning pains, a clear demonstration

of sensory impairment confined to the characteristic areas makes the diagnosis still more certain. Other signs to which a high value may be given are absence of pain when the calf muscles are compressed, loss of the vibration sense in the feet, and muscular hypotonia. Irregularities in the outline of the pupils without an obvious explanation, or pupils which react well to accommodation but sluggishly to light, are very strong evidence of past syphilis and should be duly appraised. This may be called the second stage of tabes in which the diagnosis is founded on (1) evidence of syphilis; (2) tabetic pains; (3) sensory disturbances with a characteristic distribution.

3. If to these sensory disturbances there is added an Argyll Robertson pupil, or if one ankle-jerk or knee-jerk is absent or definitely diminished when compared with its fellow, the diagnosis is established beyond doubt. This may be called the stage of the fully developed disease. The diagnosis rests on—(1) evidence of syphilis; (2) lightning pains; (3) characteristic sensory signs; (4) the Argyll Robertson pupil in one or both eyes; (5) absence of one or both ankle- or knee-jerks, or a definite diminution in one of them.

Lightning pains probably indicate that the disease is active. In the absence of pains tabes would still be suggested by the combination of an Argyll Robertson pupil with an absent ankle or knee-jerk, or by the combination of one or both of these signs with characteristic sensory loss. In such cases, however, it would be impossible to say whether the patient was suffering from tabes which was likely to progress, or whether the disease had been arrested in its earliest stages.

DIFFERENTIAL DIAGNOSIS.—*Peripheral neuritis.*—The signs common to both diseases are loss of reflexes, hypotonia, inco-ordination and sensory loss. Wasting, loss of power and tenderness of the calf muscles distinguish peripheral neuritis. A complete history and examination will usually reveal the cause of the neuritis, or disclose certain signs of tabes.

Friedreich's disease.—Loss of tendon reflexes and inco-ordination occur in both diseases, but the age of the patient, the family history, the speech defects, nystagmus and the deformities of the feet and spine make the diagnosis easy. Juvenile tabes is sometimes mistaken for Friedreich's disease.

Course and Prognosis.—In most instances the disease is well established before some serious symptom brings the patient under observation. For this reason it is usually impossible to determine the sequence and duration of the signs that are found, but if the onset of lightning pains and of ataxia are taken as landmarks, an idea of the extreme variability of the course of tabes in different cases will be obtained. In many patients the disease remains stationary in the earliest stage and causes no disability. In a larger number inco-ordination appears after a pre-ataxic stage of 10 or 20 years. Some become ataxic within five years of the onset of pains, a few within a year. Once ataxy appears, its rate of increase varies within wide limits. It may be so rapid that walking becomes impossible in a few weeks; it often increases very slowly, and only interferes seriously with walking after several years, and in a large number periods of increase in the ataxy alternate with long periods in which it is stationary or undergoes temporary amelioration.

The course of the other symptoms is equally variable. In general, irritative phenomena—pains and crises—tend to diminish, while the signs of destruction of sensory nerves—diminished sensation, hypotonia, etc.—

increase. Ocular palsies are frequently of short duration, and bladder and rectal symptoms are often temporary. It is impossible to foretell how any given case will progress, but there seems to be some connection between the period which has elapsed since syphilis was contracted and the rate of evolution of the disease—the longer this period the more benign the course. If the symptoms have increased slowly in the past, the future course is likely to be slow, whereas cases of rapid onset often progress rapidly. When optic atrophy occurs, blindness results almost invariably, and a proportion of these cases develop general paralysis of the insane.

The prognosis as to life is variable. Most tabetics die of intercurrent maladies or of some cardio-vascular complication, but life is constantly menaced by cystitis and ascending infection of the urinary tract. It should be remembered that in many cases of tabes, the malady undergoes arrest, and the patient may never become ataxic or grossly disabled. Such arrest may be found in persons who have at no time had any anti-syphilitic treatment. Conversely, tabetics who have been rigorously treated in this way may become progressively disabled. On the whole the prognosis as to both working capacity and life is best in those cases where the bladder can be kept free from infection.

Treatment.—This falls under three heads: treatment by anti-syphilitic remedies, general treatment, and treatment of individual symptoms.

ANTI-SYPHILITIC TREATMENT.—Mercury and bismuth are the most valuable drugs. Several courses of daily inunctions should be given at intervals, until 60 inunctions have been applied. This may be supplemented by injections of arsphenamine. Between the courses, which may be repeated at 6-monthly intervals, mercury should be taken by the mouth in a pill or mixture. Most observers agree that all forms of intraspinal therapy are useless, and many will doubt whether any form of anti-syphilitic medication can be conclusively shown to influence the course of the malady. Nevertheless, patients should be given the benefit of such doubt on this point as there is, and it is this attitude, rather than one of unreasoning optimism, that really determines the adoption of anti-syphilitic treatment in tabes dorsalis. It should be remembered that long continued and repeated treatment of this order is apt to evoke an unfortunate psychological reaction in the subject of such a chronic malady as tabes, and to produce in him a syphilophobia that is not less distressing than syphilis. In few circumstances can it be more essential to remember that one is treating a sick man and not merely a disease.

GENERAL TREATMENT.—In early cases the patient should be encouraged to continue at his work and avocations, so far as this is consistent with the avoidance of undue mental or physical stress. Strict moderation in the use of alcohol and tobacco should be enjoined. The diet should be generous, and efforts should be made to prevent the rapid loss of weight which is a feature of many cases. Strict attention to the bowels is necessary. In many tabetics the normal call to stool is not felt, and if regular efforts to open the bowels are not made, stasis develops readily. This should be treated by enemata or by glycerin suppositories. Purgatives should be used with discretion. They are of little use in stasis, and should not be given if there is any tendency to rectal incontinence, as this is always worst when the motions are soft. A change from purgatives to enemata or suppositories

will often relieve this distressing symptom. The bladder should be emptied at regular intervals, regardless of the call, which is apt to be less insistent than in normal persons. In general, rest in bed is to be deprecated. In some instances, however, where ataxy develops rapidly, it is advantageous, provided that daily treatment by massage and exercises is instituted at once.

TREATMENT OF SYMPTOMS.—Pains.—Of the many drugs that have been tried for the relief of pains the following either in single or in various combinations have been found useful: aspirin, phenacetin, phenazone, amidopyrine, cannabis indica, colchicum, ammonium chloride and sodium salicylate. After one has lost its effect another will often give some relief. Morphine is the only drug that is certain in its action, but it cannot be allowed except on isolated occasions, when for some special purpose it is essential that the patient should be free from pain for a few hours. In no disease is the morphine habit more rapidly acquired or more difficult to break. External applications rarely do any good. Chloroform on lint sometimes gives relief. Hot baths, hot applications to the limbs and blisters to the spine are worthy of trial.

The clothing should be warm, and sudden changes of temperature should be avoided. Residence in a warm country is an advantage. Attention to small details, such as the avoidance of constipation and abstinence from alcohol, often has a favourable effect.

Crises.—Gastric crises, like the pains, are very resistant to treatment. Chlorbutol in cachets containing 10 grains is often useful. It may be given twice or at most thrice in 24 hours. The effect of the drug should be watched carefully, as it sometimes produces alarming depression of the heart and respirations. When chlorbutol fails cerium oxalate and tincture of iodine should be tried. The use of morphine is not justified. Rectal crises are sometimes relieved by small doses of grey powder with opium or pulv. ipecac. et opii. The lower bowel should be emptied daily by enemata. In mild cases with morning diarrhoea an enema or a suppository should be used before the first evacuation. Thereafter the patient should try to resist the desire to defæcate, which soon passes away, and with a little training this troublesome symptom can usually be overcome. Laryngeal crises though very alarming are practically never fatal. They are usually relieved at once by an inhalation of nitrite of amyl.

Bladder disturbances.—When there is any difficulty in passing water a mixture containing 5 minims of liq. strychninæ thrice daily will be found useful. When the bladder is imperfectly emptied the use of the catheter should not be delayed. Only too often neglect of this matter leads to death from pyelo-nephritis. It is well to remember that serious infections may run a painless course. Their presence must be sought for even when pain is absent. This entails an examination of the urine from time to time for evidence of inflammation in the urinary tract. If pus-cells are present in the urine, urotropine and acid sodium phosphate should be given by the mouth. If this does not remove them, the bladder should be irrigated daily until the urine becomes normal. True incontinence of urine is often diminished by 5 minim doses of tincture of belladonna thrice daily, or by the use of the following pill: R Ergotin (Bonjean) gr. 1, Ext. Belladonn. gr. 1-4. Ft. Pil. Sig. i t.d.s.p.c.

Ataxia.—Just as a normal person by practice and effort can learn to perform feats of balance and muscular co-ordination which are impossible for one untrained, so the tabetic by concentrating his attention on his movements can be taught to make greater use of his remaining powers. The results of appropriate re-educative treatment are often astonishing. It is no uncommon thing to see patients who had been confined to bed for months able to get about freely again. Permanency of the result is often a gratifying feature.

As long as the patient is able to get about the necessary re-education can be acquired, if he is taught to pay particular attention to each movement of his limbs, and to attempt to carry it out accurately. In more severe cases, and when the patient is confined to bed, re-education should be given along the lines devised by Fraenkel. Constant supervision is necessary at first, and the treatment should begin in an institution, or under the supervision of a skilled attendant.

No remedy is of avail in checking the progress of optic atrophy.

The condition of the feet often requires attention. Corns should not be cut. Perforating ulcers should be curetted and dressed with a paste of iodine and starch. A cradle should be placed over the feet to prevent deformities, and over-extension of the knee-joint should be prevented by wearing a suitable splint.

Charcot's joints.—As soon as this condition is discovered, the patient should be put to rest, the joint immobilised, and those measures used which tend to relieve the œdema and the effusion into the joint; and if occasion demand, the joint should be aspirated. When the joint becomes dry it should be rested for a long period. For example, the patient with a Charcot's foot should use a peg stump for six months, when the condition will be found to have healed. The knee is a difficult joint to support, and the best treatment is excision of the joint, with the production of a stiff knee.

CONGENITAL SYPHILIS OF THE NERVOUS SYSTEM

Affections of the nervous system are much less frequent in congenital syphilis than in the acquired disease. Viewed broadly, the pathological changes and the clinical manifestations are the same in both. Regarding the first, meningitis, endarteritis and gummata are common to both forms; but while *central softening* from arterial disease is characteristic of acquired syphilis, *cortical cell atrophy and subsequent sclerosis* are prominent features in congenital cases. As for the symptoms, mental defects, with convulsions and spastic weakness of the limbs, are typical of congenital syphilis in contrast to the hemiplegias and monoplegias, with or without convulsions, which occur in the acquired form. It is noteworthy that the combination of obvious visceral and integumental lesions, with parenchymatous degeneration of the nervous tissue, is very common in the congenital, but not in the acquired disease.

Symptoms.—Many syphilitic infants suffer from *convulsions* during the first two years of life and in many cases these are given as the cause of death. In those who survive, fits may continue or they may begin again towards the end of childhood. The latter is more common. The fits in some

cases have all the aspects of idiopathic epilepsy, and may continue throughout life without the addition of any symptoms suggestive of local brain disease. In another group, convulsions are followed by symptoms of *hemiplegia* or of *spastic diplegia*. The same defects may appear apart from convulsions.

Mental impairment is one of the common features of the disease. Idiocy is rare. More often the defect is first noticed between the ages of 5 and 15 years. The child may merely cease to learn, and retain any acquirements he possesses, or he may lose his memory and become slowly demented.

Vision is often defective as a sequel of atrophy of the optic nerve or of choroido-retinitis, and bilateral deafness is not uncommon. Affections of the remaining cranial nerves are rare.

Juvenile general paralysis appears most often between the ages of 10 and 17 years. It has been seen as early as the eighth, and as late as the thirtieth year. In some cases it results from congenital syphilis, in others from syphilis acquired in infancy or in childhood. The physical signs are the same as in the adult form. The mental symptoms, as might be expected, differ from those in adults, when mental decay sets in before the appearance of the instincts and passions which form the content of the delusions in older patients. A boy of 12, for example, is not likely to have delusions regarding his wealth or his intellectual capacity or his sexual powers, although he may well have grandiose ideas concerning his physical strength. Optic atrophy is very common in juvenile cases, and as in adults, signs of tabes are present in many cases.

Juvenile tabes presents the same features as in adults. It is important to remember that in rare instances, tabes in an adult owes its origin to congenital syphilis or to syphilis acquired in infancy.

The diagnosis of congenital syphilis of the nervous system rarely causes any difficulty, as the patients almost invariably present some of the stigmata of their malady.

Treatment by mercury should be carried out perseveringly. The results are disappointing.

DISSEMINATED SCLEROSIS

Synonyms.—Multiple Sclerosis; Insular Sclerosis.

Ætiology.—Disseminated sclerosis disputes with neurosyphilis and intracranial new-growths for primacy as the commonest organic nervous disease in these islands and throughout Europe. It is said to be less frequently observed in North America.

Cases have been recorded in which the disease was noticed after acute illnesses, such as scarlet fever, influenza and rheumatism; but it is probable that these simply made more prominent a condition already present. Febrile illnesses are usually followed by increase in the symptoms, and many patients with disseminated sclerosis relate that they became much worse after an attack of influenza. In the great majority of the cases there is nothing in the family or personal history to which the disease can be attributed. In one instance, confirmed by examination after death, it attacked a mother and her child, and a few similar cases, as well as the affection of several members of a family, or of a household, have been recorded.

The onset is most frequent between the ages of 16 and 30, the sexes being affected equally. It is rare for the disease to begin after the age of 55.

The cause is still wholly unknown. Weston Hurst has recently expressed the considered view that there is no sure evidence that any of the demyelinating diseases of the nervous system are directly due to the action of a filtrable virus. The signs of inflammatory reaction in this disease are compatible with the view that it is infective in origin, but it may be added that it behaves like no known infective disease.

Pathology.—The disease has been described by Nageotte and Riche as “an affection constituted by multiple inflammatory foci, varying greatly in size and number, disseminated irregularly throughout the length of the cerebro-spinal axis. The chief features of these foci are (i) their sharp outline, (ii) their irregular and capricious shape, (iii) the fact that they do not interrupt the axis cylinders, which are only demyelinated and deformed as they traverse the focus. Hence the absence of Wallerian degeneration. The abundance of neuroglia in the foci justifies the name sclerosis which has been given to the process.”

These foci are visible on naked eye examination, the fresh ones as greyish translucent foci, the older ones as greyish or pinkish shrunken areas. Grey and white matter are both affected, the foci having some predilection for the walls of the ventricles. The foci bear no necessary relation to blood vessels.

Under the microscope the older patches are found to contain proliferated neuroglia and nerve fibres which have lost their myelin sheaths. The axis cylinders in the sclerosed areas escape destruction for a long time. For this reason secondary degenerations do not occur in the spinal tracts, and sections of the cord between lesions at different levels present normal appearances. Ganglion cells are also spared; hence wasting of the muscles supplied by the affected segments is not a feature of the disease. In recent patches, œdema is present with infiltration by small lymphocyte-like cells, plasma cells and compound granular corpuscles around the blood vessels, especially in the adventitial sheath of the veins. It is highly probable that these inflammatory changes represent the initial lesion, and that the alterations in the nerves and in the neuroglia are secondary to them.

Symptoms.—In the early stages the axis-cylinders in the diseased areas are not interrupted completely, but suffer partial and temporary impairment, which alters in intensity with the severity of vascular and other inflammatory changes in the tissues around them. Moreover, as the inflammation subsides in one patch a new one develops and produces a different set of symptoms. Hence it is not surprising that the earliest symptoms are often slight and fleeting, or that they may first appear now in one part and now in another. In spite of this, however, certain symptoms and physical signs appear with remarkable regularity* and render disseminated sclerosis, in the more advanced stages at least, one of the most distinctive and most easily recognised diseases of the nervous system.

It is remarkable that though the demyelinating lesions, which are often of considerable size, occur anywhere in the central nervous system and commonly involve the fillet, the lateral fillet, the spinothalamic paths and the peripheral neurones in their intramedullary course and the visual path, yet anything but the most transient loss of function never occurs in connection with these

systems. On the other hand, the phylogenetically newer systems—the pyramidal paths and the proprioceptive system commonly suffer permanent damage. The common transient loss of vision may be determined by an oedematous lesion of the optic nerve as it traverses the optic foramen.

MOTOR SYMPTOMS.—Weakness in the lower limbs is the symptom for which many patients first seek relief. Beginning with a feeling of heaviness or stiffness in one or both limbs, the weakness, which may be limited at first to one group of muscles, increases, in some uniformly, in a larger number with remissions or with periods of apparent recovery, until at last, after a time which varies from a few weeks to many years, it ends in severe spastic paraplegia. The physical signs are those of pyramidal lesions in general—increased tone in the muscles and exaggeration of the tendon reflexes, diminution or loss of the abdominal and cremasteric reflexes, and Babinski's plantar response. They are of extreme importance, for some or all of them may be present when the patient's complaints are still trivial, and they are found so constantly in all stages of the disease that the diagnosis of disseminated sclerosis is rarely made in their absence.

The paralysis can often be distinguished from that of other pyramidal affections by the variations in its severity from time to time, and by the occurrence of remissions or of apparent recovery, the improvement sometimes lasting for weeks or months, and, in rare cases, for many years. In most cases, moreover, examination will reveal some other sign—nystagmus, intention tremor, or pallor of the disk—which betrays the cause of the paralysis. In one large group of cases, however, the symptoms are those of a steadily increasing spastic paraplegia without remissions and without any indication, either in the physical signs or in the history, of extra-pyramidal disease. The gait may be but slightly altered, even when the tendon reflexes are greatly exaggerated and the plantar responses are "extensor." Later, it becomes spastic or spastic and ataxia. Sometimes ataxia makes walking very difficult, when the power in the limbs is only slightly impaired. In the arms there is often loss of power associated with exaggeration of the tendon reflexes. In some cases the arms are affected before the lower limbs, when astereognosis and loss of sense of position from a lesion in the course of the parietal projection produce one of the commonest of the early symptoms—the "useless arm."

TREMOR.—The characteristic tremor in the arms appears on voluntary movement only, and increases in rate and amplitude as the goal is approached. For these reasons it is called intention, volitional, or terminal tremor. It is sought for by causing the patient to touch his nose with the tip of one finger. In its minimal form the tremor appears as two or three jerky movements of the finger just as the goal is attained, or the finger reaches the nose without any abnormal movement and then oscillates, so that it slips away from the nose again or depresses it several times before coming to rest. The tremor may be noticed first in writing or in performing other delicate movements, such as threading a needle. Later, the rate and amplitude of the movements increase, and the tremor, although still greatest at the end, appears almost as soon as a voluntary movement begins. In advanced cases it prevents all useful movements, and the patient is unable to do anything for himself. The arms are affected earliest and most often, but nodding of the head is common, and any part of the body may be affected. Beside

intention tremor, other types of inco-ordination of the limbs are occasionally seen, such as those characteristic of lesions of the optic thalamus or of the mid-brain or of the cerebellum.

SENSORY SYMPTOMS.—*Subjective.*—Numbness and tingling in the extremities and alterations in the sensation of various parts are common complaints. They are often transient, and may be the only symptoms during the premonitory period. Severe pains are rare, but many patients complain of stiffness or of aching in the limbs and in the back. Occasionally intense neuralgic pain of trigeminal nerve distribution is found.

Objective.—Severe sensory loss is not common, but careful examination will often reveal areas of skin in which sensation is impaired. Occasionally the loss is severe, and may show so sharp an upper level as to suggest the presence of a spinal tumour. In many cases the sense of position and passive movements in the limbs is seriously affected, in others loss of vibration sense is the only sensory sign. An isolated loss of the last named, in the legs, is a phenomenon of diagnostic importance. Like the other signs, the sensory disturbances often show considerable variations in extent and degree at different examinations.

OCULAR SYMPTOMS.—Attacks of *double vision* are frequent, and highly characteristic of the disease. Close interrogation, avoiding the leading question if possible, will often elicit an account of these attacks when the patient has not mentioned them at first, either because he has forgotten them, or because it does not occur to him that a symptom so remote or so transient can have any bearing on his present trouble. This diplopia is of the highest importance, because it is often the sole complaint when the patient seeks advice for the first time, and because its presence, or a history thereof, is often the deciding factor in the diagnosis of early cases with spinal symptoms. Double vision in a young person should always arouse the suspicion of disseminated sclerosis, and if it is associated with signs of pyramidal tract disease, the combination makes the diagnosis almost certain.

Strabismus is uncommon. Even when the patient is seen whilst complaining of double vision it is unusual to detect any limitation in the range of the ocular movements.

Ptoxis is rare.

Nystagmus is present in more than half the cases, but not so frequently as an early sign. It is usually fine, rapid and horizontal, appearing only when the eyes are directed to the side. In some cases the eyes oscillate constantly whatever their position. Except in rare cases, there is no apparent movement of objects, even when the oscillations are of wide range.

Visual failure.—Diminution of visual acuity due to lesions in the optic nerves—*retrobulbar neuritis*—occurs sooner or later in nearly every case. It may precede all other symptoms by a period of several years. As in the case of the other symptoms, it is subject to exacerbations and periods of improvement. A young healthy person complains of rapidly increasing mistiness of vision, usually in one eye, sometimes in both or in one after the other, reaching its maximum in a few hours or days; this is often preceded or accompanied by pain about the orbit, which is increased on moving the eye. In the common unilateral case the signs are those of a lesion in one optic nerve; the pupil on the affected side is larger than its fellow; its direct reaction to light is impaired, but it contracts well consensually. Tests with

a small object, preferably coloured, reveal a central scotoma. At the onset the disk is usually normal, but in a few instances the inflammation reaches the nerve head, in which event the disk is blurred and swollen. Later the disk may be pale or normal. Rapid improvement of vision is the rule. Special tests may reveal a persistent slight loss of visual acuity, and a partial central scotoma, or, very rarely, a complete central scotoma. Subsequent acute attacks are common. In some cases the onset of visual failure is gradual. Usually the defect is slight, but it may be serious, although complete blindness never occurs. In these cases the disk is pale, especially in its temporal portion, and the field shows a central scotoma or narrowing at the periphery.

MENTAL SYMPTOMS.—Defective memory and slight impairment of intellectual power are common. Some of the patients are morose and subject to fits of depression, but the majority are surprisingly cheerful, and do not seem to suffer mentally even when their physical state is most pitiable. In many cases there is considerable loss of emotional control, and ready laughter or weeping is fairly common. More often there is merely a tendency to laugh at trivial things.

SPHINCTER DISTURBANCES.—These troubles arise from interference with the long path in the spinal cord by which volitional consent and inhibition are held upon the act of micturition. Therefore, lack of control in the form of hesitancy and precipitancy are common, and retention may occur. In rare cases, control over the rectal sphincter is lost.

OTHER SYMPTOMS.—Deafness, giddiness and tinnitus, sometimes with repeated vomiting, are common. Epileptiform convulsions are rare. In most instances the distribution of the signs will indicate that the lesions are multiple; but sometimes, although the patches are numerous, the signs are those of a single lesion, say of the internal capsule, of the midbrain or of the cerebellum.

CEREBRO-SPINAL FLUID.—The colloidal gold test may give a weak paretic curve in association with a negative Wassermann reaction. In a few cases the number of cells is increased. Otherwise the fluid is usually normal.

Diagnosis.—The combination of spastic weakness of the legs with "Charcot's triad" of symptoms—namely, intention tremor, nystagmus and scanning speech—which is so widely and so erroneously regarded as characteristic of the disease and as necessary to its recognition, is rarely seen except in the later stages of disseminated sclerosis. As this malady usually presents itself to us in its initial stages, when it may and should be diagnosed, it commonly consists in a group of signs of involvement of the pyramidal tracts: namely, increased tendon jerks, Babinski plantar responses, absent abdominal reflexes, a little weakness of dorsiflexion of one or both feet, possibly also some weakness of flexion of the proximal segments of the lower limbs, and usually a degree of impairment, or loss, of vibration sense over the malleoli. In many cases, this is all we can find, but in an otherwise healthy young adult, it is a syndrome more likely to be due to disseminated sclerosis than to any other pathological process.

Perhaps there may be confirmatory signs, such as a little nystagmus, slight intention tremor or sensory ataxy of an arm; it may be pallor of the temporal half of one or of both disks—a pathognomonic sign. If some or all of

these signs have, as it were, been arrived at after such a fluctuating course as we have seen to be so typical of most cases of disseminated sclerosis, then diagnosis can be no longer in doubt, and it is comparatively seldom that pathological examinations of blood or cerebrospinal fluid are really necessary for this end.

When, after some years, the disease is fully developed it still retains its individuality. The patient is commonly euphoric, there is frequently tremor of the head, and sometimes of the whole body, when the patient tries to stand or walk. The arms are unsteady, the legs spastic and weak—sometimes showing a tendency to pass into the condition of “paraplegia in flexion.” There is little sphincter control left, but cutaneous sensibility is commonly almost or quite intact.

At whatever stage disseminated sclerosis comes under observation, a careful inquiry into the history of the illness is important, and to elicit this requires a knowledge of the natural history of this disease as it has been outlined here.

Disseminated sclerosis has to be diagnosed from various diseases, of which we will consider the following :

Hysteria.—The serious mistake of attributing the early symptoms of this relentless disease to hysteria can be avoided by careful examination of the nervous system. Pallor of the disk, absence of the abdominal reflexes, or a distinct difference between them at corresponding points on opposite sides, unequal exaggeration of one or more of the tendon reflexes when compared with their fellows, Babinski's plantar response on one or both sides—any one of these signs alone would render a diagnosis of hysteria untenable.

Compression of the cord.—When the signs in disseminated sclerosis are purely spinal, the diagnosis from *spinal tumour* presents real difficulties. The first may be mistaken for the latter, when the paralysis increases steadily without remissions and is associated with sensory loss extending upwards to a definite level, while the reverse error may be made when the symptoms caused by a tumour are purely motor, or vary in intensity, or are associated with nystagmus.

Friedreich's ataxy.—This may be suggested by the presence of ataxy in a young patient with disseminate sclerosis. The distinction can be made at once, for in the latter disease the tendon reflexes in the lower limbs are exaggerated, whereas they are lost early in Friedreich's disease.

Course and Prognosis.—Despite the remarkable fluctuations which may mark its course, the disease ultimately disables the sufferer and is the cause of his death. Nevertheless, it is important to remember that after the initial outbreak of symptoms, some patients regain normal physical capacity, lose all abnormal physical signs, and lead a normal life for several years. Five, 10 and 15 year periods of this kind are by no means rare, and in general it may be said that the period of evolution of the disease is longer than is generally supposed. On the other hand, a few cases run a rapidly downhill course from the onset. The later in life disseminated sclerosis makes its first appearance, the more benign its course, and sufferers may be found who have reached old age without gross or total disablement. Commonly, after two or three fresh exacerbations with intervening recoveries of greater or less completeness, a slowly increasing permanent disability sets in. It is not

possible to say that those cases which run the longest and less distressing course owe this to treatment, for many untreated cases fare relatively well. But there are certain factors which do appear to influence its course unfavourably in most, though not in all, instances; thus, intercurrent illness, especially if it be febrile, injuries which disable the patient for a short period, all surgical interventions—including the therapeutic interruption of pregnancy which is designed to avert the frequently-seen exacerbations that follow the puerperium—and prolonged or recurrent physical exhaustion.

Treatment.—The behaviour of disseminated sclerosis makes the assessment of any mode of treatment extremely difficult, and a failure to appreciate the wideness of its fluctuations and the length and completeness of some of its remissions is responsible for many therapeutic claims that in the hands of those best acquainted with this malady fail to justify themselves. So far, there is no remedy which exerts any constant or certain influence upon the course of the disease.

Arsenic is the remedy which has the longest vogue, and many believe that it is of value, though the present writer has never been able to satisfy himself that this is the case, and has seen severe exacerbations interrupt the course of a series of injections of arsenical preparations.

Nevertheless, and *faute de mieux*, we may prescribe arsenic either by the mouth, as Fowler's solution, or by intramuscular injection of one of the arsphenamine derivatives. In the former case, some begin with a dose of min. 3, which is increased by one minim per dose on alternate days until min. 8 is taken three times daily. The dose is then reduced again to its original level. If this method is used, it is best to stop all arsenic for a week at the end of each complete curve of dosage. Not every patient can tolerate doses larger than min. 3 or min. 4 three times daily. This method is probably as useful as that of intramuscular injection, but considerations of expediency may dictate the use of the latter method.

A more recent suggestion is that of Brickner, who gives quinine hydrochloride in doses of three to five grains twice daily, continued over a long period. Here, again, intolerance may intervene and prevent this. The present writer has used quinine extensively in this way since its introduction by Brickner, and regards it more highly than arsenic. Other recent forms of medication include liver therapy, pyrexial therapy, protein shock therapy, and vaccine therapy. None of these has justified itself in the writer's experience, and when it is recalled that a febrile illness commonly aggravates the severity of disseminated sclerosis, it is scarcely surprising that pyrexial therapy should sometimes have the same result.

The fact that disseminated sclerosis is sometimes—though not always—adversely affected by a confinement has led to the increasing advocacy of terminating pregnancy at the third month to avert this ill effect. But this procedure is exposed to the same objection as a full-term delivery or any surgical procedure, and sometimes has the same unfortunate influence upon the course of the malady. It is therefore not a therapeutic measure that can be justified by its results. The correct procedure is to take every possible measure to maintain the health and nutrition of the pregnant woman, and to afford her at this time and after the puerperium more than the ordinary amount of rest. This is the rational, if not always the acceptable, line of treatment.

Of great importance is the right ordering of the patient's life, when practicable, and the avoidance of fatigue in the early stages of the disease.

JAMES COLLIER.

W. J. ADIE.

Revised by F. M. R. WALSHE.

ACUTE DISSEMINATED ENCEPHALOMYELITIS

Synonym.—Acute perivascular myelinoclasia (Hurst).

Ætiology.—This is unknown. The malady may follow one of the exanthemata, or may develop without any known exciting factor. A form following vaccination first attracted attention in 1922, and later cases have been recorded after measles, smallpox and varicella. It is doubtful if the meningo-encephalitis of mumps belongs to this category. Whatever be the preceding acute specific fever, or if none has preceded the attack, the symptomatology and pathological lesions are the same. It seems unlikely that a filterable virus is responsible, since no neurotropic virus is known that attacks the white matter. All such viruses so far identified have been polio-clastic, that is, they attack grey matter only.

The relation of this variety of encephalomyelitis to other demyelinating diseases, *e.g.* disseminated sclerosis and neuromyelitis optica, is uncertain. There are no grounds for assuming that the pathological process has a common cause.

In conclusion, it seems that acute disseminated encephalomyelitis may occur apart from any exanthem, and is entitled to recognition as a separate pathological process with its own clinical picture.

Pathology.—The lesions are widespread throughout the central nervous system, and consist in perivascular foci of softening involving the entire nerve fibre, axis cylinder and myelin sheath. The foci tend to coalesce. There is proliferation of the microglia cells, and some lymphocytic reaction with a secondary gliosis. The lesion of the nerve fibres is probably primary in the myelin sheath. The lumbar cord tends to be more severely hit than higher levels.

Symptoms.—This is characteristic and uniform. The onset is relatively sudden, and when an exanthem has been present, the nervous symptoms make their appearance at more or less regular times: thus from 10 to 13 days after vaccination, from 5 to 13 days after the appearance of the rash in smallpox, and at the end of the first week in measles.

In most cases the symptoms are predominantly encephalic, but myelitic forms are also seen. In the former case there is a slight rise of temperature, some unsteadiness of gait if the patient is ambulant. Drowsiness soon develops, and there is headache, stiffness of neck and back, Kernig's sign and tache cerebrale. The abdominal reflexes usually disappear and there is disturbance of sphincter control. The state of the tendon jerks is variable. The drowsiness may deepen into coma. At this stage the limbs become flaccid and the tendon jerks abolished. There may or may not be cranial nerve palsies and some oedema of the optic disks. Death may ensue from bulbar paralysis with hyperpyrexia and a terminal broncho-pneumonia. On the other hand, a dramatic recovery may ensue even when the patient

is in this grave state. With survival there is a gradual return to full consciousness.

In the spinal type unconsciousness, if it occur, is short-lived. A severe paraplegia with incontinence of urine and fæces and sensory loss develop. The paraplegia is usually flaccid with loss of tendon jerks at first. The upper limbs may escape or be less severely affected than the lower. Recovery is the rule, but this may be slow and finally incomplete.

Treatment.—There is no specific treatment and the management of the case should follow the lines applicable to any severe and acute paralysis, with special care to avoid bedsores, infection of the bladder, hypostatic pneumonia and wasting.

NEUROMYELITIS OPTICA

Synonyms.—Diffuse myelitis with optic neuritis ; Devic's Disease.

Definition.—A form of disseminated myelitis, preceded or accompanied by retrobulbar neuritis, with or without papilloedema. It is commonly acute in onset, and may end in death or in arrest with residual disabilities. Recovery is rare. Persons of all ages from adolescence onwards may be affected.

Ætiology.—Nothing whatever is known of its causation, and therefore it has been suggested that the disease is infective. None of the neurotropic viruses is known to produce the demyelination which is the characteristic lesion of the disease, nor is there any evidence that this is bacterial.

There are points of resemblance to disseminated sclerosis on the one hand, and to Schilder's disease on the other, both in the morbid anatomy and symptomatology of the disease, but there are equally significant points of difference, and whether or not the three are ætiologically related cannot be affirmed.

Pathology.—The spinal cord shows either diffuse or multiple disseminated lesions. They may be confined to a few segments of the cord, or may be found from end to end of this structure. The essential feature of the lesions is a demyelination of axis cylinders. There is also round-celled perivascular infiltration, an intense proliferation of microglial cells, and a multiplication of tiny vessels in the affected areas. The optic nerves present the same type of lesion, namely, an intense demyelination of the nerve fibres.

Symptoms.—The blindness which indicates the optic nerve lesion may precede or may follow the appearance of paraplegic symptoms. The latter develop rapidly, and may spread upwards until sensory loss and muscular weakness reach the upper thoracic level. Blindness, with some swelling of the optic disc, and central scotoma may ensue. The patient may become progressively worse and die ; or the paralysis may become stationary and then proceed to complete recovery of both power and of vision ; or the subject may be left with disability of varying severity.

The paraplegia is that characteristic of a diffuse spinal lesion in that there is sensory loss, paralysis, and loss of sphincter control.

Treatment.—No treatment has any clear influence upon the course of

events. Arsenical preparations have been employed—as for disseminated sclerosis. The management of the case is that of any paraplegia.

F. M. R. WALSHIE.

SCHILDER'S DISEASE

Synonym.—Encephalitis periaxialis.

Definition.—A malady characterised anatomically by a progressive and massive demyelination of the white centre of the cerebral hemispheres, proceeding from a single focus or from two symmetrical foci, and producing the clinical picture of progressively increasing failure of cerebral function, local at first, but advancing in terms of the functions of the contiguous regions which are next affected, by the spread of the disease from its starting-point.

Ætiology.—Nothing is known of the essential nature of the disease, nor is it certain that all cases included under this heading form a homogeneous group. Originally regarded as an inflammatory, probably an infective, disease, the increasing evidence of its familial incidence suggests that it may be primarily degenerative. It has also been suggested that those cases in which an inflammatory reaction is present may be infective, and those in which it is absent—as it may be—degenerative. Many of the reported cases have occurred in childhood, even as early as the second year. The latest case was in the fifth decade of life. The sexes are equally affected.

Pathology.—The characteristic lesion consists of: (1) A primary demyelination and, later, destruction of the axis cylinders of the central white substances of the cerebral hemispheres, which till very late spares the subcortical zone of white fibres and the radial cortical fibres, and produces a translucent jelly-like appearance of the oval centres. (2) A very early and perhaps primary overgrowth of the neuroglia, forming a feltwork, which is particularly intense round the vessels. (3) A general infiltration of the white matter of the brain with round cells, all of which are of neuroglial origin, and most of which are engaged in the removal of altered myelin or in the formation of neuroglial fibres.

The process commences most commonly as a symmetrical patch of demyelination, in either occipital white centres, less frequently in both temporal white centres or in both prefrontal white centres, and spreads directly thence until the whole of the oval white centres becomes demyelinated. The corpus callosum is involved, and the demyelination spreads downwards through the crura into the brain stem. Sometimes, especially in the central regions, the disease starts on one side, and, after playing havoc with the white centre of one hemisphere, spreads across the corpus callosum into the other. The resulting picture of a brain, normal on the surface, and on section with apparently normal cortex and intact subcortical white bands, but with the oval centre completely changed and translucent, is peculiar to this disease. Not infrequently other patches of the disease may be scattered throughout the central nervous system. This scattered distribution and the prominence of demyelination bring Schilder's disease very close to disseminated sclerosis, and it has actually been described as "disseminate sclerosis in childhood"; but the massiveness and mode of spread of the lesions, together with their distribution, with predilection for the brain and avoidance of the spinal cord,

its incidence in childhood and its entirely different symptomatology, separate Schilder's disease sharply from disseminated sclerosis. It is largely to Collier that we owe the clinical recognition of this malady.

Symptoms.—The clinical aspect is precisely that which might be expected from a progressive destruction of cerebral function, spreading by contiguity from the initial seat of the disease. In many of the cases blindness—by which is meant blindness without any change in the optic disks and with pupils reacting normally to light—has been the first symptom, and is the result of the symmetrical demyelination of the occipital white matter. As the disease spreads forwards into the temporal regions, bilateral deafness appears; and, later, bilateral ataxy and astereognosis—due to parietal involvement, bilateral spastic paralysis—the result of central involvement, and complete amentia—due to callosal and prefrontal involvement, develop.

In those cases in which the initial seat of the disease is in the temporal, central or frontal regions, the first symptom to appear is obviously determined by the location, and the order of development of symptoms will be changed, but the mode of progress is the same in all. Where the disease starts on one side only, hemianopia or hemiplegia is the first symptom, and these are followed by the train of added signs produced by the extension of the disease into other regions. Complete mindlessness and paralysis always dominate the clinical picture in the end. The disease-process within the brain sometimes causes swelling with increase of intracranial pressure, and signs of the latter may appear in the form of headache, vomiting and papilloedema. Such cases are not common, and most of them have been regarded in life as cases of intracranial tumour. Fits are by no means uncommon. Sometimes they constitute the initial manifestation of the disease, and they may occur at any time during its course, and may be local or general. Fever is usually absent, but there may be irregular pyrexia and some of the more acute cases have been pyrexial throughout. The cerebro-spinal fluid is normal in the majority of the cases, but sometimes there is an increased protein content and a small excess of lymphocytes.

Diagnosis.—The onset with cerebral blindness or with bilateral deafness, followed by signs of progressive cerebral destruction, is so rare in any other disease as at once to suggest the diagnosis of Schilder's disease, indeed no less than two-thirds of the reported cases have shown this picture. When the disease begins unilaterally, and more particularly when headache, vomiting and papilloedema are present, the distinction from intracranial tumour is difficult or even impossible, for in both diseases the local commencement and the progressive destruction occur. In Schilder's disease, however, high-grade papilloedema is not met with, and consecutive optic atrophy does not occur. It should be borne in mind that any locally commencing progressive destruction of the brain may be an example of this malady.

Course and Prognosis.—In most cases Schilder's disease is regularly progressive to a fatal termination. In some, however, periods of standstill have been noted, while in a few others marked improvement for a time has occurred, as the result of administration of mercury, arsenic and iodides. The duration has varied from 7 days to 36 months, with an average of 9 months.

Treatment.—No treatment is at present known that will influence the course of the disease.

THE PRIMARY CEREBELLAR ATROPHIES

In the present state of knowledge a satisfactory description or classification of the primary cerebellar diseases is not possible. They are extremely rare, and in the recorded cases the nature and the incidence of the pathological change varies so from case to case that in all probability what have been described as distinct "types" are in many cases no more than different stages of a single process.

Of all the primary atrophies of the cerebellum it may be said that their ætiology is unknown, but the cause probably endogenous. In some forms there is clear evidence of heredo-familial factors, but not in all. Some of them appear in early infancy, others in later life. The lesion is bilaterally symmetrical, essentially an atrophy, and in the late cases is slowly progressive.

The most useful working classification is as follows :

- (i) Atrophy of the cerebellar cortex.
- (ii) Atrophy of the central white matter of the cerebellum.
- (iii) Atrophy of the spino-cerebellar tracts.

Of these the least rare is Friedreich's ataxy, in which there is also a lesion of the posterior column fibres, but nevertheless this disease is most conveniently dealt with here.

(i) *Atrophy of the cerebellar cortex.*—This may be total, or may be confined to particular cortical regions. It may be found as a post-mortem discovery in idiots dying in infancy, or may develop slowly in middle-aged or elderly persons (Marie's delayed cortical atrophy). The last-named form is most marked on the upper anterior parts of the cerebellum. There is a characteristic loss of Purkinje cells. The clinical picture is that of a slowly developing ataxy of gait, severe disorder of articulation, and later ataxy of the upper limbs. Nystagmus is rarely present.

(ii) *Atrophy of the central white matter of the cerebellum.*—This is commonly known as olivo-ponto-cerebellar atrophy, and is characterised by a severe loss of nerve fibres in the central white matter, with a secondary proliferation of glia fibres and nuclei, the cortex being relatively intact. In addition there is degeneration of the pontine nuclei and their fibres (middle cerebellar peduncles) and of the olives. This also is a slowly progressive lesion of middle-aged and elderly persons, occasionally familial in incidence. It can only with difficulty be distinguished clinically from cortical atrophy, except when it is accompanied by Parkinsonian symptoms or by dementia, as is sometimes the case.

Scherer has found that a similar type of lesion is constantly found in the substantia nigra and corpus striatum in these cases, and when severe is responsible for the Parkinsonism sometimes observed. Similarly, focal atrophy in the cerebral cortex may be present, and may account for the dementia which may be present. Scherer suggests that all these changes have the characters of a focal, premature senile change, and points out that the lesions of Pick's focal atrophy and Huntington's chorea also belong to this category.

(iii) *Atrophy of the spino-cerebellar pathways.*—A rare form is the so-called spino-cerebellar ataxy, which may vary in its clinical expression from a con-

genital tremor, to gross ataxy of all four limbs, accompanied in some cases by optic atrophy, and ocular palsies. The lesion is a degeneration, falling most severely upon the dorsal-spino-cerebellar tract and less severely upon Gowers' tract and upon Clarke's column. It is a malady of children and adolescents.

More familiar is Friedreich's ataxy, which is described below under its own heading.

I. FRIEDREICH'S ATAXY

In addition to the slow, clumsy ataxy, Friedreich's type is characterised by the absence of the knee-jerk and other deep reflexes, and by the presence of the extensor plantar response and of contractures, especially in the form of pes cavus, and by the presence of curvature of the spine in the later stages of the disease.

Ætiology.—The first signs of the disease usually appear in early childhood and before the sixth year; but symptoms may not be evident until a few years later. In a considerable number of cases, however, the onset is delayed until the time of puberty, while in a few examples the onset may be delayed until after the age of thirty years. As a rule the age incidence is approximately the same in each child-rank of the same family; but sometimes the phenomenon of "anticipation" is well marked, the disease appearing at an earlier age in each succeeding generation as a whole, or in successive children of the same parents. The disease is said to be slightly more common in males. Isolated cases in which no heredity can be traced are not rare. Indirect heredity is the most common, for the reason that the subjects of this disease are usually afflicted in childhood and incapacitated by the time adult life is reached, and that they therefore do not procreate. Transmission occurs both through the males and through the females. Direct heredity is, however, by no means so uncommon as has been supposed, and in one family under my observation the disease had been transmitted from father to son for seven generations.

Pathology.—The spinal cord is unusually small, and apparently this smallness may be congenital, and the posterior roots tend to be small, grey and poorly myelinated. The essential change is a primary degeneration of certain neurones in the dorsal column of the spinal cord, of the pyramidal tracts and of the spino-cerebellar tracts, both dorsal and ventral. This degeneration commences first in the periphery of the axon, which slowly dies back towards the nutrient nerve cell, as the branches of an aged tree tend to die back towards the trunk.

The degeneration of the dorsal columns is usually the earliest change, and remains the most prominent feature throughout. The degeneration of the fibres of the pyramidal tract appears later. It has its origin in the ascending frontal convolutions, where atrophy and disappearance of the giant pyramidal cells have been shown.

The spino-cerebellar tracts are constantly degenerated, the direct cerebellar tract being the most seriously involved. The cells of Clarke's column, from which the direct cerebellar tract takes origin, and around which the pyramidal tracts end, degenerate and disappear, as does also the network of collaterals which surrounds these cells. Consequent upon these degenerations, and secondary to them, well-marked neuroglial proliferation or sclerosis

occurs. The cerebellum may be normal, or it may show varying degrees of atrophy of Purkinje's cells, or of any other of its cell elements, and of the tracts connected therewith.

Symptoms.—The onset is always insidious, and physical signs of abnormality usually precede any complaint on the part of the patient or his relatives. The first symptoms generally appear between the sixth and the tenth year of childhood; but if a careful examination be made of the younger members of the families upon which Friedreich's disease is incident, physical signs of the disease, especially the extensor response in the plantar reflex, the retraction of the great toe and some degree of pes cavus may often be found before the sixth year. Not infrequently the onset of symptoms does not occur until puberty, and in some families it is delayed until after the age of 30 years.

Ataxy is always the first sign to appear, and this is shown by an awkwardness of gait and a tendency to stumble and fall readily. Sometimes it is obvious from the history, that the ataxy dates from the earliest years of infancy when it is said that the child was never strong on his legs from the time of learning to walk, and that he could never run properly or join on equal terms with other children at play. As the disease progresses, the gait slowly becomes more irregular and clumsy. The patient walks with his feet upon a broad base, and staggers and reels from side to side; but, notwithstanding this, he keeps a fairly direct line of progression. He takes short steps which are unequal, and which are irregular in relation to the line of progression, and the movement of each foot as it is raised is poorly co-ordinated. There is never the undue excursion and noisy stamping of the feet which are so characteristic of the gait of tabetic patients.

In standing the body oscillates from side to side in slow and clumsy fashion, and coarse tremors of the head and trunk are constant features in advanced cases (titubation). Sometimes Romberg's sign is present; but this is never so well marked as in tabes, and it is frequently absent. The ataxy invades the upper extremities, as a rule, later than the legs. There is first clumsiness with the finer movements, and then little by little with all the movements. It closely resembles the ataxy due to gross disease of the cerebellum, and differs from that which occurs in tabes, and that irregular breaking of a movement towards the end of its accomplishment, which has been long termed "intention tremor," is frequently seen.

Very characteristic of the disease, and highly important in diagnosis, is the occurrence of irregular involuntary movements, which are often described as like those of chorea or of myoclonus. They differ entirely, however, from the movements of chorea, etc., in that they occur only when the limb or some of its segments are unsupported. In advanced cases such movements are constantly seen in the head and neck as nodding movements and tremors, and in the trunk as swaying instability, when the patient is sitting unsupported or standing. Similar ataxy and irregular movements affect the muscles of the eyes, of the face, tongue, larynx, etc., and the respiratory muscles. In the eyes they are seen as fine, regular nystagmus and as coarse, irregular jerkings, chiefly upon lateral deviation. There is no other disease in which ataxy of the facial muscles is so conspicuous for, in engaging the patient in conversation, all the facial muscles may be observed in irregular contraction. The ataxy of these muscles causes an invariable impairment

of articulation, which gradually becomes indistinct, clumsy, drawling and slurred. The syllables tend sometimes to be separated, adding a staccato element. Explosive utterance is almost constant, and from the irregularity of the respiratory movements short inspiratory whoops are not uncommon. Articulation thus closely resembles that of advanced disseminated sclerosis, the cause being identical in the two diseases, namely, interference with the cerebellar co-ordinatory mechanism of speech.

The strength of movements is at first little impaired ; but as the disease advances and the pyramidal degeneration increases, the power is gradually lost in proportion to the degree of the pyramidal degeneration, which varies greatly in different cases. The lower extremities are affected first and most, and later the arms, and in severe cases at a late stage paralysis may be almost universal.

The condition of the muscular tone depends upon the relative degree of degeneration in the posterior roots and in the pyramidal tracts respectively, the former tending to abolish and the latter to increase it. As a rule the influence of the posterior root degeneration is preponderant and, therefore, the limbs are flaccid and hypotonic, but occasionally they are somewhat rigid. Contractures are the rule, but these are confined to the lower extremities. The most constant of these produces the deformity of the feet characteristic of Friedreich's disease, and known as "pes cavus." The great toe is strongly retracted, the tarsus is pulled up, and the metatarsus is dropped and the plantar arch is increased. The outline of the inner border of the foot comes to resemble the letter Z, the tarsus, metatarsus and great toe forming the three limbs of the Z. Sensibility is but little affected ; but in most cases minute examination reveals slight relative loss to touch, pain and temperature, most marked at the periphery of the limbs and diminishing upwards. Similarly there may be slight loss of sense of position in the limbs, with diminution of osseous sensibility to the slowly vibrating tuning-fork.

The ocular movements are almost always intact apart from the already described nystagmus. In rare instances strabismus, diplopia and ptosis have been recorded. The pupils are not affected. Optic atrophy is a rare phenomenon in Friedreich's disease, yet it has been reported in quite a number of otherwise typical cases.

Mental symptoms are usually not conspicuous, but some of the patients are of poor mentality from the first, while others show a tendency to severe mental degeneration in the later stages of the disease. Emotional instability, irritability and outbursts of temper may occur.

Absence of the tendon reflexes is a most characteristic feature, and is often the first objective sign of the disease. When one considers, however, that the absence or presence of the tendon reflexes depends upon the relative degree of affection of the posterior column upon the one hand, and upon the pyramidal degeneration upon the other, it is not surprising to find in cases where there is a major degeneration of the pyramidal tracts, that the knee-jerks may persist or even be brisk into the advanced stages of the disease. The abdominal reflexes gradually disappear. The plantar reflex is invariably an extensor response. The sphincters usually escape. The cerebro-spinal fluid presents no abnormality.

Spinal curvature is very common, and may reach a severe degree. It consists of a scoliosis of the dorsal region, and often with some kyphosis, and

with a compensatory reverse lumbar curve. The cause of this deformity is probably the defect in the postural tone of the muscles, which occurs when the afferents subserving the function of postural tone, and which are contained in the spino-cerebellar tracts, are severed.

Diagnosis.—In uncomplicated cases the diagnosis is a matter of no great difficulty on account of the strikingly distinct nature of the symptoms. Friedrich's disease can hardly be mistaken for tabes, since the history of heredity, the peculiar deformity of the feet and spine, the extensor response, the speech affection and the nature of the ataxy contrast strongly with the loss of pain sensibility and of deep sensibility, the pupillary changes, the sphincter trouble, the abnormal Wassermann reactions and the abnormal cytology of the cerebro-spinal fluid in tabes. The distinction from disseminated sclerosis presents more difficulty; but in this disease the onset never occurs in childhood, there is no heredity, the deep reflexes are never lost, and the spinal deformity does not occur.

Course and Prognosis.—The course of the disease is usually progressive in slow and irregular fashion, and the prognosis is therefore in every case serious; but the average duration of the disease is over 30 years, and in some cases it seems to have no tendency to shorten life. The prognosis is worse and the course more rapid in those patients who have shown disability from the time of learning to walk. In some cases the disease appears to become arrested, as, for example, in one family which came under my observation, twelve members in three generations were affected with typical Friedrich's disease, yet none of them was incapacitated from following a normal life, and those that were deceased had all survived the age of 70 years. Intercurrent maladies, febrile illnesses and debilitating influences generally, may have a strong effect in hastening the advance of the disease, and bringing about a fatal termination. Confinement to bed from any cause whatever has a most derogatory influence upon the ataxy, and upon the capacity for walking. It is therefore of great importance that these patients shall be kept off their legs as little as is possible. Cases in which the ataxy becomes extreme, or in which paralysis from pyramidal degeneration becomes severe, necessarily become bedridden, and in this condition the patients may survive for many years. In other cases rapid increase of the symptoms of degeneration within the nervous system is followed immediately by drowsiness, asthenia and coma, and death occurs in that peculiar toxic state which is commonly the end-result of all degenerative nervous diseases.

Treatment.—No treatment is known which specifically affects the malady. General tonic treatment, and all measures which improve the general health and mental well-being, often have a surprising effect in improving the ataxy. Re-educational training of the limbs and trunk in the form of Fränkel's exercises are most beneficial. Properly designed boots to ensure the most advantageous use of the deformed feet must be provided.

2. FAMILIAL SPASTIC PARALYSIS

This malady is here described with the hereditary ataxies, since it seems to fall naturally into the group of diseases in which primary degeneration of the pyramidal tracts is a usual anatomical feature, and of which a familial and hereditary incidence is the rule. Moreover, among the hereditary

ataxies every grade of transition is seen to the type of pure familial spastic paraplegia. Whilst in the majority of the hereditary ataxies cerebellar, spinal and cerebral lesions coexist, yet there are the purely cerebellar and the purely spinal type; and the purely cerebral type, in the form of familial spastic paralysis, forms a natural end to the series.

Ætiology.—The disease is sometimes hereditary, but is more commonly familial and incident upon several children of the same parents. Sporadic cases are not very rare. The onset is gradual in early life, and usually occurs after the sixth year.

Pathology.—The pathological changes consist in a primary degeneration of the pyramidal neurones which apparently takes place in terms of the length; those supplying the lumbo-sacral region, being lower and longer, are earliest affected; those supplying the brain stem, being shortest, are the last to be affected. Degenerative changes in the neurones of the posterior columns of the spinal cord are often present, showing the transition to the pathological type of the hereditary ataxies.

Symptoms.—The clinical aspect consists in the slow development of spasticity and weakness, first and most in the legs, which gradually increases and progresses to the trunk and upper extremities, and involves the face last and least. The usual signs of pyramidal involvement are present in the loss of abdominal reflexes, increased deep reflexes and extensor type of plantar reflex. The malady is progressive, increasing to complete paralysis, and in its course contractures of the spastic muscles occur, that of the foot and leg producing some degree of pes cavus, while, above this, flexor contracture at hip and knee is met with. Optic atrophy is by no means uncommon. Mental symptoms do not occur in uncomplicated cases, neither is epilepsy observed.

Diagnosis.—This malady is most easily confused with cerebral diplegia; but the latter disease appears much earlier, so soon after birth, in fact, as defective movement in the child can be ascertained. Further, cerebral diplegia is not a progressive disease in the majority of the cases, and it is often associated with mental deficiency and recurring convulsions.

PARALYSIS AGITANS

Synonym.—Parkinson's Disease.

Definition.—A progressive disease of insidious onset and slow course, usually occurring in the second half of life, and characterised by a peculiar stiffness of the muscles, which tends to fix the body in a certain posture, which can be changed less speedily than in health, and which gives rise to a distinctive facial expression, bodily attitude and gait. The stiffness is accompanied by weakness, and often by rhythmic tremors, which have earned for this malady the name "shaking palsy."

Ætiology.—Little is known of the causal factors of this malady. It is essentially a disease of the decline of life, and though in rare instances it is met with as early as the eighteenth year, the maximum incidence is from the fiftieth to the seventieth year. Men suffer twice as frequently as women. Heredity seems to play no part in the causation; but it is remarkable that longevity in one or both parents is common.

Pathology.—No naked-eye changes are to be found other than the vascular and degenerative changes which are common in senile conditions. The facts that tremors and rigidity, almost identical with those of this disease, may be met with in tumours involving the substantia nigra of the crura cerebri—two striking cases with autopsy having been under my own care—and still more importantly, the frequent appearance of a paralysis agitans-like end-result in lethargic encephalitis, where the subthalamic region and substantia nigra are conspicuously picked out by the lesions, make it probable almost to a certainty that the locus morbi of paralysis agitans is the basal ganglia.

Symptoms.—The onset is always insidious, and the muscular rigidity is almost always the first sign to appear. This rigidity affects the face, neck and trunk to a greater extent than the limbs, and when the limbs are affected then the proximal muscles present a greater degree of rigidity than do those of the periphery. The oncoming rigidity of the facial muscles does away with the usual play of the emotional movements in facial expression, and the face assumes a fixed, anxious and mask-like expression, with absence of the usual involuntary nictitation. The voice loses its inflexions, and becomes monotonous, from rigidity of the muscles of larynx, tongue and lips; but there is no other defect of articulation. Very striking is the effect of the rigidity of the muscles of the neck, for the patient carries his head and neck in one piece with his trunk as if he were a statue, never inclining or raising it in the customary expressive manner, and if he turn round to look at anything he tends to move the whole trunk round with the head. In looking sharply to one side the eyes move before the head, whereas, under normal circumstances, the coarse adjustment of this movement is done first by the neck muscles, and the fine adjustment subsequently by the eye muscles. The stiffness of the trunk muscles gives a stooping attitude with the head inclined forwards, while that of the upper extremities causes the shoulders to be rounded, and the arms carried with the elbow semiflexed, and pressed into the sides. The gait is highly characteristic in marked cases since, on account of rigidity of muscles, it is deprived of spring and suppleness; the patient, in the characteristic attitude above described, takes small gliding steps, displacing his centre of gravity as little as possible. If, by any circumstance, such as catching the feet against an unevenness of the ground, or a push, the centre of gravity is much displaced, the patient often has a difficulty in regaining it, and in moving to recover his centre of gravity is unable quite to catch it up, and so continues the movement of necessity until he fall or come in contact with some object by which he can arrest himself and restore his balance. This phenomenon is more often seen in advanced cases, and is known as “propulsion,” “retropulsion” and “lateri-pulsion,” according as the centre of gravity is displaced and the movement occurs in a forward, backward or sideways direction. Festination is the term used for the quickening of the pace sometimes seen in this attempt to overtake the displaced centre of gravity. In the hand the rigidity is greater in the interosseal muscles, and the hand therefore tends to assume the “interosseal position” with the fingers pressed together and the thumb adducted, the metacarpophalangeal joints being flexed, and the interphalangeal joints extended. From this rigidity of the hand the writing becomes small as well as tremulous, and the patient finds it difficult to write in a straight line. Muscular weakness always accompanies the rigidity and the tremors. It is slight until the late

stages of the disease, when it may increase rapidly and render all useful movement impossible. On account of the rigidity and consequent slowness of movement, the sense of weakness which the patient experiences is much greater than the actual weakness as tested by the dynamometer. Tremor is present in the majority of cases. It usually commences in the hand and forearm, and is most conspicuous in this situation; but it may be seen in the face, tongue, jaw, neck and feet, while, in rare cases, it may be universal. The nature of the tremor is peculiar, and is highly characteristic. It is a regular rhythmical contraction of the muscles, alternating in the opposing groups with a frequency of from four to six oscillations per second with a range of from an $\frac{1}{8}$ th to $\frac{3}{4}$ ths of an inch. Its rhythmic nature, its slowness and its course range distinguish it from other varieties of tremor. In the hand the characteristic movement of the tremor is the rolling together of the opposed thumb and fingers, cigarette-rolling, bread-crumbling or drum-tapping movement. There is nearly always in addition a peculiar pronator-supinator tremor. The tremor is increased by excitement and by self-consciousness, and ceases during sleep. A highly characteristic feature of the tremor is in about one-half of the cases is that it continues during repose, and is temporarily arrested by the execution of volitional movement. In the other half of the cases, however, the tremor appears or is increased on voluntary exertion, and tends to be less during repose. There seems to be an antagonism between the tremor and the rigidity, for in cases where the rigidity is very conspicuous the tremor is little marked or absent, and conversely, when tremor is universal or is of early onset, rigidity is a less noticeable feature.

Other symptoms of the disease which are very commonly complained of are—(1) difficulty in turning over in bed, which is the obvious result of the rigidity of the trunk muscles; (2) flexion of the toes into the sole of the foot, so that they are trodden on, from spasm of the plantar muscles; (3) pain of a dull aching character in the trunk and limbs, which is presumably produced by the long-continued traction of the rigid muscles upon their attachments; (4) abnormal sensations of heat and cold; and (5) hypersensitiveness to changes of temperature—the patient cannot bear to be near a fire nor yet in a cold room. Mental symptoms are conspicuous by their absence, except in the last stages of the malady, when profound asthenia overtakes both mind and body. The constant bodily discomfort, restlessness, sensations of fatigue, which the rigidity and the tremors engender, and the consciousness of a malady which is found only too soon to resist every effort to lessen or arrest it, often result in gloomy and lasting mental depression. Objective sensibility is unimpaired. The special senses and the cranial nerves are not affected. The sphincters and the reflexes are normal. Trophic changes in the periphery of the limbs, thinning and glossiness of the skin, with fluted nails and vasomotor disturbance, are common. Bed-sore is commonly met with in the late stages of the malady.

Diagnosis.—There are three points which can be surely relied upon to render the diagnosis of paralysis agitans certain in every case, namely—(1) the aspect of the patient when he is walking, when the fixed mournful expression, the stooping attitude with round shoulders, the elbows pressed into the side, and the hands carried across the abdomen in the interosseal position, the immobility of the head and neck, and the curious gliding gait which cannot fail immediately to arrest the observer's attention; (2) the rhythmic rolling

tremor which is quite unlike any other form of tremor, and which often continues during rest; and (3) the absence of any of the usual signs of organic disease of the central nervous system. Difficulty may perhaps be experienced when the aspect is little marked, and the tremor is confined to some unusual situation, such as the face, tongue or neck; but, if the possibility of tremor in any situation being that of paralysis agitans be borne in mind, its rhythmic rolling nature will give the diagnosis. When paralysis agitans is confined to one side of the body, the appearance of the patient may superficially resemble that of hemiplegia; but in these cases the peculiar aspect of paralysis agitans is marked, and the organic signs of hemiplegia, such as the extensor response in the plantar reflex, the increase in the deep reflexes, and the absence of the abdominal reflex upon the paretic side are not present. In senile tremor the rhythmic rolling quality is absent, and the aspect is not that of paralysis agitans. In post-hemiplegic tremor the organic signs of hemiplegia are present. Toxic tremor is irregular and never rhythmical, and is (mercurial tremor excepted) a fine tremor. The intention tremor of disseminated sclerosis, cerebellar disease and lesions of the red nucleus are so peculiar, and so widely different from the tremor of paralysis agitans, as to render confusion impossible.

The one clinical condition, which may so closely resemble paralysis agitans as to be superficially indistinguishable, is a not uncommon end-result in lethargic encephalitis, where from a lesion in the basal ganglia the same weakness, rigidity and tremors appear as occur in paralysis agitans. The distinction is not difficult, for the onset of lethargic encephalitis is usually acute, and the symptoms are definite. Moreover, the paralysis agitans-like syndrome of lethargic encephalitis sometimes shows a progressive amelioration, whereas paralysis agitans tends to a progressive downward course. The following features present in post-encephalitic Parkinsonism but absent in true paralysis agitans are of value in making a differential diagnosis: (i) a fluttering tremor of the closed eyelids; (ii) tremor of the protruded tongue; (iii) defect of convergence and of accommodation; and (iv) excessive salivation.

Course and Prognosis.—Paralysis agitans often begins in one limb, usually the upper, and spreads thence to the corresponding limb of the opposite, or to the other limb of the same side. In the latter case it has approximately a hemiplegic distribution, and it may remain for years much more evident upon one side of the body. The course is slowly progressive with variable rate. In some cases the malady may remain stationary for years, and this is more often seen in middle-aged subjects, before the disease has reached an incapacitating stage. Such arrest in the early stages is not often seen in young subjects, for in the latter the disease seems to take a more continuously downhill course. Real improvement in the symptoms is never seen. A fatal issue may occur in as short a time as two years; but this is exceptional, since paralysis agitans has little tendency to shorten life. The average duration is from 10 to 15 years, and since the major incidence of the disease is in the sixth decade of life it will be seen that many of the patients are of average longevity. Death may occur from intercurrent maladies, especially from bronchitis; but more commonly, after the lapse of many years, the patient becomes bedridden from increasing weakness and rigidity, and sinks into a condition of sleepy asthenia which is soon terminated by coma. An unduly

high blood-pressure is unusual in the subjects of paralysis agitans, and it is noteworthy that they do not suffer from gross cerebral vascular lesions, such as thrombosis or hæmorrhage.

Treatment.—Paralysis agitans is one of the least tractable of maladies even as regards the relief of symptoms. Hygienic measures and tonic treatment, calculated to lessen the rapidity of the degenerative process, should be employed. Where there is much rigidity, gentle exercise, passive movements and massage are useful. Care should be taken to avoid the falls which the unstable gait is likely to engender, since these are often followed by a marked exacerbation of the symptoms. Pain is best treated with aspirin, and sleeplessness with a mixture of aspirin and small doses of barbitone (grs. 2 and 3). As might be expected, electrical stimulation of the muscles tends to aggravate the tremor, and even in the predominantly rigid cases can do no good. In the latter type of case, some subjective relief may be obtained by the administration of tincture of stramonium or of belladonna (doses of from 5 to 20 minims), or of hyoscine hydrobromide (grs. $\frac{2}{100}$ to $\frac{1}{100}$ by mouth thrice daily in chloroform water). These drugs may render movements freer, but they have no influence upon the tremor. When the patient is bedridden, great care must be taken with the skin, since the immobility of the trunk greatly increases the liability to the formation of bed-sores.

HEPATO-LENTICULAR DEGENERATION

Synonyms.—Progressive Lenticular Degeneration ; Wilson's Disease.

Definition.—A rare progressive disease of the nervous system, often familial, characterised by involuntary movements, rigidity and hypertonicity, with contractures, without signs of pyramidal disease ; and by dysarthria, dysphagia, emotionalism and progressive emaciation. Several closely related clinical forms of the disease bear distinctive names : *tetanoid chorea* (Gowers), *pseudosclerosis* (Westphal), *progressive lenticular degeneration* (Wilson), and *torsion spasm*, and *dystonia musculorum deformans* (Thomalla). Cirrhosis of the liver occurs in all forms. The Kayser-Fleischer zone of corneal pigmentation occurs in the first three forms, but has not yet been recorded in torsion spasm. The most constant nervous lesions are found in the corpus striatum.

Ætiology.—The disease often occurs in children of the same parents, but there is no evidence that it is congenital or hereditary. The age of onset has been as early as 7 years and as late as 26 years. The primary and essential lesion is in the liver ; its cause is unknown. Syphilis is not a factor.

Pathology.—A multilobular cirrhosis, with "hobnail" liver, is always found after death. There is good evidence that the cirrhosis is not slowly progressive, but is the result of a number of attacks of acute hepatitis. The hepatitis has caused death in some members of affected families before nervous symptoms appeared. The nervous lesions are purely degenerative. In Wilson's case they were almost confined to the lenticular nucleus, especially the putamen. Every degree of degeneration was seen, from discoloration and sponginess of the nucleus in rapidly fatal cases, to shrinkage and atrophy, and even to complete disintegration and excavation of the ganglion. Later observers have described lesions in many other parts of the nervous system. The lesions are often most intense in the corpus striatum, but the noxious

agent has no strictly selective action on any one anatomical group of ganglion cells, or on any limited area of the nervous system.

Symptoms.—In many cases there are no symptoms of disorder of the liver during life. In other cases an account is obtained of symptoms referable to acute hepatitis before the onset of nervous symptoms—attacks of diarrhœa and vomiting, pyrexia, jaundice, migrainous headaches, hæmatemesis and sometimes definite ascites.

The first nervous signs to appear is usually involuntary movement of the extremities, which may be of several kinds. In progressive lenticular degeneration, rhythmical tremors, increasing on voluntary movement, furnish the most common symptom. This is followed by rigidity of the face, the muscles of the neck, and later of the trunk, which rigidity increases steadily until the patient becomes helpless. The rigidity of the face and neck muscles gives rise to a peculiar expressionless appearance. Still later, extensive contractures, usually in the flexed position, in the upper and lower extremities, follow; but sometimes there is extensor contracture of the latter. During sleep the tremors cease, but the contractures do not relax. Dysarthria, of a sturring type, results from affection of the muscles of speech, and may end in complete anarthria. Progressive muscular weakness and general emaciation follow; and the patient becomes emotional, facile, docile and childish. There is no fibrillation or localised amyotrophy. The optic disks and pupillary reactions are normal. There is an absence of nystagmus, cerebellar symptoms, and impairment of sensation. The reflexes are not altered, as in the case in pyramidal disease.

Prognosis.—The disease always ends fatally in a few months or years; the average duration is about 4 years.

Treatment.—None is known to have any effect upon the course of the disease.

JAMES COLLIER.

W. J. ADIE.

Revised by F. M. R. WALSH.

KERNICTERUS

Definition and Ætiology.—A yellow pigmentation of certain of the basal ganglia, associated clinically with motor disorders of the type known as extra-pyramidal, and found as a rare phenomenon in children who, normal at birth, develop jaundice within the first three days of life.

In neonatal jaundice the brain may be diffusely pigmented, or more rarely the pigmentation may be confined to the putamen, subthalamic and dentate nuclei, the cornu Ammonis and fascia dentata. To the latter variety of jaundice of the brain the name “Kernicterus” has been given by Schmorl. The nerve cells in the affected masses of grey matter show evidence of destruction and degeneration, while the nerve fibres are demyelinated.

Symptoms.—The child is healthy at birth, but within a few days develops intense jaundice, usually the form known as icterus gravis neonatorum, though kernicterus has been found in association with septic jaundice. The onset of jaundice is followed within 24 hours by tonic and clonic movements, muscular rigidity and opisthotonos, alternating with periods of flaccidity.

If the child survive, within a few weeks involuntary movements of choreo-athetoid form develop. Emotional instability and mental retardation appear as the child grows older.

Prognosis.—The cases so far identified and on record are too few to allow of any generalisation as to the expectation of life of the subjects of this malady, but in two cases recently reported by Greenfield, one patient died at the age of three months, the second at nine years.

Diagnosis.—Athetosis and comparable forms of involuntary movement are not rarely seen in children, and are in the majority of instances not associated with kernicterus. Yet when a case of such motor disorder is seen in a child in respect of whom there is a history of neonatal jaundice, the possibility of this disease should be borne in mind. Again, the development of marked symptoms of organic nervous disease immediately after the appearance of severe jaundice in a newly-born infant should lead to a consideration of this condition as the probable pathological basis.

Treatment.—There is no evidence that the condition is susceptible to any mode of treatment.

F. M. R. WALSH.

EPILEPSY

Synonym.—Idiopathic Epilepsy.

Definition.—A condition characterised by suddenly occurring disturbances of cerebral function, prone to occur over long periods of time or even through life. Of the intimate nature of the disturbance all we know with certainty is that a series of characteristic changes in the normal electrical activity of the cortical nerve cells accompanies it. The clinical forms of the disturbance suggest that this involves both loss and release of function in various regions of the brain.

Ætiology and Pathology.—Few problems in medicine have been more obscure, or more provocative of speculation and of controversy than the nature of epilepsy. It has been widely assumed in the past that there is an idiopathic epilepsy, a definite and often heritable disease, with an individuality and natural history of its own, and one of which the characteristic and often the sole expression is the fit.

On the other hand, it has also long been realised that fits clinically indistinguishable from those of idiopathic epilepsy may occur in the course of many and diverse affections of the brain, as, for example, in certain inflammations, degenerations, intoxications and also in association with new growths. Such fits have been placed in a category of symptomatic epilepsy, the relation of which to the idiopathic variety never admitted of clear formulation.

These facts have led some writers to maintain that epilepsy is no more than a symptom, one expression of many and diverse neuro-pathological processes, and because of this manifold causation they prefer to speak of "the epilepsies." Against this view it may be urged that not one of these pathological processes is necessarily, or even commonly, productive of fits and may occur without their development. Such processes cannot therefore be wholly or even primarily responsible for the fits that may, upon occasion,

accompany them. As the late Kinnier Wilson, a strong supporter of the purely symptomatic nature of epilepsy pointed out, "The fit, the fundamental symptom of epilepsy, has no histopathology." In this statement we see that the fit has become a symptom of a symptom. Again, he stated, "The number of epilepsies for which no cause other than that of inherent irritability can be discovered is being steadily reduced." This pronouncement speaks in favour of a unity rather than of a diversity in epilepsy.

It is probable, therefore, and the evidence about to be stated supports the suggestion, that the fit, and the tendency to its recurrence that we call epilepsy, owe their production to some abnormal quality of structure or of function in the brain, and that the many pathological processes that may give rise to fits—as incidental but not essential phenomena—are no more than secondary and contributory factors capable of fit production in the susceptible brain alone. Yet even if there be but a single essential factor common to all fits, it still remains practically useful, if not necessary, to speak of an idiopathic and a symptomatic epilepsy. For purposes of diagnosis, prognosis and treatment the distinction is clearly important. We must separate the two, for example, when we proceed to generalise as to the heritable qualities of epilepsy, and the treatment of a case of uræmia with fits is clearly widely different from that of a case of idiopathic epilepsy. This distinction still allows us to suppose that in idiopathic epilepsy the "fit threshold" of brain, as Lennox expresses it, is so low that the ordinary activities of life and very minor fluctuations in bodily health are sufficient to fire off a fit; while in what we call symptomatic epilepsy, the threshold is higher and is only crossed when some gross superadded disturbance of the brain—of wholly independent origin—is superadded.

The general point of view thus summarised finds a considerable measure of support in the recent researches of Lennox, Gibbs and others upon this subject. They have employed an instrument known as the electro-encephalograph which promises to do for the brain what the string galvanometer of Einthoven has done for the heart. It was originally observed by Berger (1929) that regular oscillations of potentials of a rhythm of 10 per second could be detected if electrodes applied to the scalp were led off to a recording instrument. This work has since been confirmed and very considerably amplified by Adrian in this country and many other observers. It is known that these oscillations, or, as they are called, brain potentials, are due to the electrical activity of cortical nerve cells. The various rhythms, and their sources in the cortex, that come within the normal range of variations in the healthy subject have been studied, and it has subsequently been found that in the epileptic subject these rhythms undergo characteristic variations during the course of the fit. These observations may be summarised as follows (Lennox): Every fit is accompanied by a disturbance of the normal electrical activity of the brain. The three main types of fit have distinct forms of altered rhythm, the distinguishing feature being the frequency of the waves. In the major fit the waves are abnormally fast; in the minor fit they are alternately fast and slow; while in the so-called psychical fit the waves are slow. The pattern for each patient is apt to show individual peculiarities which repeat themselves in successive fits.

The electro-encephalograph also records fits which are clinically invisible ("subclinical") and abnormal variations of rhythms in epileptic subjects may

be found during the intervals between fits. These electrical changes are found alike in "idiopathic" and in "symptomatic" fits, and afford further evidence in favour of a unity in epilepsy rather than of a number of "epilepsies."

Lennox suggests that epilepsy may, therefore, be regarded as expressing a defect in the rhythm-regulating mechanisms in the brain, or in other words as a "paroxysmal cerebral dysrhythmia." He also suggests that the cause of epilepsy is never single, that there is a fundamental cause; namely, an inherent instability of the brain, and a secondary or contributing cause. Of such there are probably very many, and it is the combination of the two that evokes the fit. It should be borne in mind, however, that we can say no more with certainty than that these variations in cortical electrical rhythm accompany the fit. We may not in the present state of knowledge say that they cause it, and the search for the factor or factors productive of the electrical disturbance remains for the future.

This brings us to a consideration of the many factors that in the past have been held responsible for the production of fits. These do not demand or deserve any detailed discussion, for they are predominantly speculative and have always lacked confirmation, either pathological or biochemical. Disturbances of metabolism, of endocrine function, and of acid-base equilibrium have all been invoked. Yet research has failed to find any constant or characteristic disorder of protein, fat or carbohydrate metabolism, or of endocrine function of any kind, nor have studies of the blood in respect of its cellular or chemical composition been other than negative. The uncertain influence of metabolic and endocrine disorders, when these are manifestly present, in the production of fits tend to strengthen the growing view that none of these factors plays any but a secondary and contributory rôle in the malady. There are, on the other hand, certain well-known clinical features of epilepsy that have in the past been deemed to point to the importance of some metabolic variations in exciting fits; thus, the increased incidence of fits in women at the time of the katamenia, and their common cessation during pregnancy have been noted in this connection.

The features of the fit suggest that the functions of the affected regions of the brain may be disordered in several ways, by suppression, as, for example, in the blunting or loss of consciousness which is probably the most constant and invariable feature of epileptic fits of all kinds, and by release, as in the hallucinations (visual, auditory and gustatory, etc.), the delirium and automatism that are sometimes seen. The actual convulsive moments of the generalised or of the Jacksonian fit can hardly be regarded as release symptoms but point from their form and character to direct stimulation of cortical cells.

Morbid Anatomy.—As has already been stated epilepsy has no histopathology, though in the brains of chronic epileptics various minor changes have been noted. Much of the material from which information on this point has been gathered is useless, since the subjects have been aments or dements, and the relation of the changes found to the occurrence of fits during life is uncertain. Among the changes thus found are smallness of the brain, meningeal thickening, convolitional atrophy and fibrosis of the Pacchionian bodies. But none of these is invariably present, and we say that there are no certainly essential macroscopic changes in the epileptic brain. Histological examination has proved equally inconclusive. Sclerosis of the cornu Ammonis,

consisting of degenerative changes in nerves, cells and gliosis, have long since been reported, but of their significance nothing can be said. In short, as has been already stated, "there is no histopathology of the fit." Examination of the endocrine organs has also given no significant results.

Heredity.—In the past great stress has been laid upon the alleged heritable qualities of idiopathic epilepsy, and they have been made the basis of sweeping prohibitions in the matter of the marriage and child-beggetting and -bearing of epileptic subjects. Nevertheless, it seems that direct transmission of epilepsy from parent to child is exceptional, and this has certainly been the experience of the present writer in dealing with the abundant material that presents itself at the out-patient clinics of such an institution as the National Hospital. Recently Cobb has expressed the view that on so-called eugenic grounds as high a proportion as 90 per cent. of epileptics cannot reasonably be advised against marriage. Statistical reviews by others (*e.g.* Muskens, Marchand) seem to confirm this view. Marchand maintains that after the exclusion of all those cases of epilepsy in which gross external factors, such as syphilis, injury, etc., can be excluded there is nothing that can be called a familial epilepsy capable of direct transmission without the intervention of adventitious factors. In congenital affections resulting from developmental defects of the ectodermal layer, epilepsy when it is seen is no more than the symptom of a cerebral lesion. In support of these views he has been able to marshal a great weight of evidence. On the other hand, recent electrographic observations of Lennox suggest that in the parents (one or both) of epileptic subjects abnormally wide fluctuations in the rhythm of the brain potentials are unduly frequent, and it may be that while epilepsy as such is not inherited, some instability of cortical cell function may be inherited, which in combination with other factors (when one or more of these chance to be present) lead to the appearance of epilepsy. This is approximately what Marchand maintains in more general terms.

Exciting Causes of the Fit.—In the majority of cases of idiopathic epilepsy no cause whatever can be found for the occurrence of the first fit. It may follow unpleasant emotional excitement, alcoholic excess, or may occur completely unheralded while the subject is in his normal health and environment. In the case of frankly symptomatic epilepsy, metabolic disturbances in early life, and especially rickets, are potent causes. Acute intoxications with absinthe, lead, bismuth and many other poisons may invoke epilepsy, as may also the poisons occurring in the specific fevers in childhood, in uræmia, cholæmia, hyperpiesia and puerperal eclampsia. And although in these intoxications the epileptic phenomena do not usually recur after the cause has disappeared, yet there is not one of the above-mentioned conditions which has not been followed by persistently recurring epilepsy. Injury to the brain of any nature whatever, whether from violence from without or from disease within, may cause epilepsy. Traumatic cases in which the brain has been severely wounded are not associated with epilepsy in a greater percentage than 5 per cent. Cerebral tumours, agenesis, encephalitis meningitis, cerebral syphilis and vascular lesions give a higher percentage, which in children has been placed as high as 30 per cent. Another form of symptomatic epilepsy that should be borne in mind when a previously normal person who has lived abroad, particularly in the tropics, develops epilepsy is that due to cysticercosis. The cysticercus or bladderworm stage

of *T. Solium* normally develops in the pig, and infestation of man by the adult tapeworm is due to eating pork thus infested. Man may also accidentally eat tapeworm eggs and serve as the intermediate host, the embryos showing a tendency to invade the brain. MacArthur, who has drawn attention to this factor in the development of fits, records 20 personally observed cases of cysticercosis of the brain, 6 of them soldiers invalided from abroad. The fits may be Jacksonian in type or generalised.

Symptoms.—**PRODROMATA.**—The circumstances which immediately precede the occurrence of an attack are of some importance. Speaking generally, it is uncommon for an attack to occur when the attention is fixed, or when some act is being performed, and from this it follows that the epileptic is relatively or absolutely free from attacks when at work and doing, and only in the rarest cases comes to harm or injury from accident. Some patients are able, by an effort of will in fixing attention, or by the performance of some vigorous action, to arrest attacks which have already begun.

Sometimes a change in the general condition of the patient may make him aware, or may acquaint those around him, that an attack is pending, and such signs of altered metabolism may herald an attack for from a couple of hours to a week. Headache, irritability, restlessness, euphoria, lethargy, somnolence, unusual appetite and a peculiar vacant look may all be met with in this connection.

Not infrequently the attack is preceded by paroxysmal manifestations which are in reality minute attacks, such as partial lapses in consciousness, a sense of strangeness, "dreamy state," jactitations of any of the muscles exactly resembling those seen in uræmia, slight auras, giddiness, sneezing and yawning.

DESCRIPTION OF THE ATTACKS.—The varieties of the epileptic attack are legion, and several types may occur in the same subject—indeed, it is unusual for fits to be always of the same type in one subject. They tend to vary both in degree and nature. They are usually divided into the less spectacular "minor" attacks, in which spasm is not a prominent feature; and "major" attacks, in which spasm is conspicuous. This distinction is purely artificial, for most patients have attacks of both varieties, and the two merge by insensible gradations the one into the other. Further, the minor attack often is the initial manifestation of the major attack.

The following description will serve to illustrate the more definite manifestation of epileptic attacks:

1. *Simple jactitation.*—Single twitching of individual muscles or groups of muscles, occurring, now in one part of the body, now in another, are seen in the majority of epileptics at some time or other. They are conspicuous in the convulsions of childhood, where they often constitute the chief clinical feature. They are well known as the "carphology," or "subsultus tendinum," of uræmic and eclamptic attacks, and in the "typhoid state." They may be not infrequently noticed in the epileptic person when he is otherwise well, and engaged perhaps in conversation or other occupation. Gowers emphasised epileptic twitching as a prodroma of an oncoming severe attack; but while in some instances this is undoubtedly true, yet it frequently occurs when no attack follows. It has been called "epileptic myoclonus."

2. *Simple loss of consciousness.*—In this, the commonest of all minor

phenomena, there is a simple break in the continuity of consciousness. The train of thought and action is suddenly arrested for a few seconds, and there is a sudden stillness of posture and facial expression which attracts the attention of a witness. The face may show sudden pallor, a vacant expression, and curious fixity of the eyes, with large pupils. The patient does not fall, or move, or drop anything that he is holding. In a few seconds the attack is over, leaving the patient unable to describe what has happened, perhaps a little confused for some seconds, sometimes emotional and even hysterical. More often he continues what he was about as if nothing had happened. Such attacks sometimes occur very frequently, even hundreds in a day. They are characteristic of pyknolepsy, in which the prognosis is absolutely good, and also of a form of epilepsy in which rapid mental degeneration occurs and in which the prognosis is equally bad. Further, they may occur in organic disease of the brain.

3. *Simple loss of consciousness with falling.*—The patient suddenly falls, without warning, in the extended position, and almost always prone, so that his head reaches the ground first, and his forehead receives the bruise. He regains consciousness immediately, and picks himself up as if nothing had happened. It is not uncommon to see the forehead one region of scars, as the result of repeated falls; to prevent these a pneumatic protector should be worn. This form gave rise among the ancients to the name "falling sickness," or "morbus caducens." In another form of this type the head, or the head and trunk, alone are affected. The patient does not fall, but simply drops the head forward—"nodding spasm," or "spasmus nutans"; or he drops the head and bends the trunk forward—"salaam spasm."

4. *Simple loss of consciousness with slight spasm.*—This forms a gradation from the above types to the definitely convulsive seizures. The spasm is seen as conjugate deviation of the eyes, and perhaps of the head also, or it takes the form of laryngeal and respiratory action, giving rise to a groaning noise, or may involve any part of the musculature.

5. *Local fits.*—First studied by Hughlings Jackson, these events have the name of "Jacksonian epilepsy," and this term has unfortunately become coupled with common errors that are no part of Jackson's teachings. These are (1) that some local disease invariably underlies the Jacksonian fit, and (2) that the Jacksonian fit necessarily consists of local motor convulsion. Actually, in many cases naked-eye and microscopic examination may fail to reveal any local lesion, and none such may be present. Also, the Jacksonian fit may consist of phenomena involving any possible cortical function. It may be added that local disease of the brain quite commonly evokes generalised fits indistinguishable from those of idiopathic epilepsy, and conversely that the latter form of epilepsy may express itself in the form of Jacksonian fits.

Psychic fits.—These may take the form of peculiar mental states, of instantaneous onset, remembered afterwards sometimes in exquisite detail, sometimes only in vague character. Emotional conditions of fear or horror, which may cause the patient to attempt with violence to escape from his surroundings—"cursive" epilepsy—may occur. Or, the attacks may take the form of a sudden feeling of misery, or an intense sense of personal wrongdoing, a sense of intense familiarity in surroundings which are unfamiliar, a sudden sense of strangeness, as in a patient whose fit was "suddenly seeming

to be somewhere else," a sense of euphoria or of intense mental energy, a dreamy state, often associated with smacking of the lips and champing or swallowing movements, which often has a pleasurable emotional tone. Again, the psychic fit may take the form of a highly complex and detailed hallucination.

Visual fits.—These may take the form of negative phenomena, such as dimness of vision, complete darkness or hemianopia, or of positive effects, such as flashes of light, scintillating stars or balls of fire, or of both together in the form of blindness with flashes of light. In the last case they may closely resemble the visual phenomena of migraine, and are not infrequently caused by a local lesion of the occipital region. Complex visual hallucinations may occur.

Auditory fits.—The hallucinations of sound may be of any nature—hissing, booming and elaborate musical sensations, as of bells, being common. There is usually a sense of coincident deafness or "far away" hearing, which passes off with or soon after the sound.

In one case the fits could always be produced by sounding the hallucination note upon the open diapason of an organ. No other note or sound produced the fit. (Such directly excited fits, though very rare, are well known in connection with olfactory, visual, auditory and common sensory stimulation, and have been termed "reflex epilepsy.")

Olfactory and gustatory fits.—These hallucinations are always described as of "favour," usually unpleasant. Very often, movements of the lips, tongue and jaw, or swallowing movements are present, and the dreamy state already referred to may be associated. From the location of the functions of smell and taste in the cortex of the uncinate gyri, and from the common occurrence of fits of this character in lesions of these convolutions, this type of fit is often referred to as the "uncinate fit."

Sensory fits.—These hallucinations may have their seat of commencement in any part of the body. They may remain local, but more commonly they spread from the point of origin in terms of the local representations of the body in the cerebral cortex, and usually from the periphery towards the trunk and head, but a sensory fit may spread to the extreme periphery first. For example, commencing in the fingers, it may spread up the arm to the head, or on reaching the shoulder it may invade trunk and leg before ascending to the head. It may be bilateral, confined to the anterior or posterior aspect of the body.

The sensation may be described as "numbness," "tingling," "pins and needles," "vibration," "rushing," "as if the limb were withering," much more rarely actual pain. Sometimes the sensation is indescribable. The sensory attacks have their origin in a local disturbance of the parietal region of the cortex, and may indicate the presence of an organic lesion in that region. They may be accompanied or followed by temporary loss of sensibility, in the form of astereognosis, loss of sense of position, or anæsthesia.

Another group of sensory fits for which it is impossible to give any definite cerebral localisation at present, is that of the so-called visceral auras, which are mainly referred to the distribution of the vagus nerve. Such are the very commonly occurring "epigastric" sensation, and sensations of choking, dyspnœa, nausea and cardiac sensations.

It is quite possible that the sudden feelings of malaise or of faintness

which may constitute the main feature of some epileptic attacks are expressions of the sudden lowering of blood pressure which is known to immediately precede the epileptic attack.

Disturbances in the realm of the vestibular nerve are common indications of epilepsy. Sudden giddiness may be the sole indication of epilepsy, and is a common initial event in major attacks. It may be indicative of the sudden fall of blood pressure, or the feeling of rotation may be consequent upon early spasm causing conjugate deviation of the eyes.

It must be carefully borne in mind that all the phenomena which have been described above may occur as isolated events and so constitute the epileptic attack. Often, however, the disturbance of the cortex spreads widely, involving general convulsion and loss of consciousness; but the initial phenomena are remembered by the patient as the "warning" of the attack and have from ancient times been termed "auras," when preceding general convulsion. In reality, they constitute the essential part of the attack as showing the region of the brain in which the disturbance starts, and in every patient who has such "warnings" preceding his severe attacks, the warnings occur at times by themselves without any such sequel.

Motor fits (simple paralysis).—This is the rarest of all forms of the epileptic attack. It consists in a sudden inability, relative or complete, to use a limb or one side of the body or the whole voluntary musculature, with no preceding convulsion. There are the usual signs of cerebral paralysis—at first flaccidity with a tendency for the jerks to fail; a few moments later increased jerks, with absent trunk reflexes and extensor plantar reflexes, all of which signs soon disappear. It may occur as an isolated phenomenon. More often a slight "minor" attack or a local sensory attack accompanies the onset of the paralysis. Sometimes such an attack may result from local disease of the brain. Such attacks when involving the right face or right side of the body may occasion aphasia, or the aphasia may occur alone as the attack of simple paralysis. Such attacks of simple paralysis without convulsion are well known in uræmia, hyperpiesia, metallic poisoning and general paralysis of the insane.

Local convulsion.—The common foci of onset are the angle of the mouth, the thumb and index finger, and the great toe, but the spasm may occasionally begin elsewhere. It rarely produces conjugate deviation of the eyes as a primary movement, but usually in association with, and secondary to, deviation of the head. The convulsive movements may remain confined to their place of onset throughout the fit, or may spread widely so as to involve a whole limb, one-half of the body, or the entire musculature. In fits involving the musculature of the right half of the face and tongue, speech is usually lost during the attack and returns shortly after its cessation. Spasm never affects the muscles of one eyeball alone, but the spasm is in terms of conjugate deviation of both eyeballs in one direction. The same rule applies when the neck is affected, for the head is then either rotated to one side or extended or flexed on the chest. With the other bilaterally associated muscles it is different, for the tongue is affected on one side only, as is also the face. The onset is with tonic spasm, which after a little while gives place to broken or clonic spasm, becoming more and more intermittent and finally ceasing. In some cases, but by no means in all, the convulsion

leaves varying degrees of weakness in the affected muscles—Todd's paralysis or post-epileptic paralysis, with transient signs of loss of function of the pyramidal system, such as loss of trunk reflexes, increase of jerks, and extensor plantar reflexes.

Epileptic spasm usually puts the hand in the position of extension at the interphalangeal joints, flexion and abduction at the metacarpo-phalangeal joints, flexion at wrist and elbow, and adduction at the shoulder. The feet are dropped and inturned, with extension at the knee and hip. Usually the trunk is in opisthotonos.

The sequence of tonic spasm at first, followed by clonic spasm, though usual in epilepsy, is not invariable. Purely tonic fits may occur with no clonic spasm, the tonic spasm remitting suddenly. Such fits are usually of slight severity and duration, and are almost always general and very rarely local.

On the other hand, the spasm may be clonic only. The simple jactitation already described may be taken as a simple clonic fit. Local fits, especially of the face and of the hand, may be purely clonic. Again, some of the most severe of all general epileptic convulsions are clonic throughout so far as the limb and trunk musculature is concerned, but some tonic conjugate deviation of eyes and head is usual.

Loss of consciousness in local fits.—This seems to depend upon the extent of the cortex involved. With narrowly confined fits there may be no impairment at all, as in local convulsion of the face or hand, or as in a patient who vividly described to me a slow visual fit as it was occurring. When the fit spreads, consciousness is usually impaired, and when lost, it is lost late in the fit. For example, it is usual for a convulsion which spreads to one-half of the body to cause some impairment, and if it involves both sides generally consciousness is always lost.

General convulsive fits (haut or grand mal).—There is some reason for believing that every major attack has a local commencement in some region of the brain, and that it is in reality a local fit which rapidly becomes general. When such an attack commences with a local aura there is proof positive of local commencement. When it commences with conjugate deviation of head and eyes to one side, this is certain indication that the disturbance commences in the opposite hemisphere. When the spread of the disturbance is so rapid as to cause instant loss of consciousness there is no memory to retain the initial event of the attack. The seizure may begin with any of the local manifestations above described, the epigastric aura and giddiness being two of the most frequent. Or the patient may be only aware of his attacks from the condition in which he finds himself after their occurrence. The tonic spasm commences with conjugate deviation of both eyes to one side, followed by rotation of the head to the same side. The blood pressure falls, the countenance is for a moment pallid, the eyes widely open, the pupils dilated, the cornea insensitive. The march of the tonic spasm usually causes head retraction and opisthotonos; the upper extremities are stiff in flexion and adduction, the lower extremities in extension. If standing, the patient falls usually backwards, but the conjugate deviation of head and eyes may bring his face to the ground first. The respiratory muscles and larynx, going into spasm, produce the epileptic "cry," and the respiratory movements being no longer possible the face darkens with the asphyxia, and the sphincters

may relax, with the evacuation of bowel or bladder. The protrusor spasm of the tongue and the closing spasm of the jaw may cause the tongue to be bitten. After the tonic spasm has lasted some seconds and perhaps has produced such a degree of asphyxia as seems hardly compatible with survival, it begins to break into a series of sudden shock-like, jerky movements—the clonic spasm—which continue for some seconds, becoming less regular and occurring at longer intervals until, with a final jerk, the muscles become perfectly limp. Meanwhile the relaxation of the respiratory and laryngeal spasm have allowed the respiratory movements to return and to churn up the saliva, often bloodstained, which escapes at the nose and mouth in the form of froth. At the end of the attack there is complete and unrousable loss of consciousness, the pupils are dilated and insensitive to light, the corneal reflexes absent, the knee-jerks absent, and the plantar reflexes extensor in type. In a short time the knee-jerks return, the plantar reflexes return to the normal, and consciousness returns. Usually the patient is dazed, feels ill, has marked headache, and if left to himself soon sleeps heavily for some hours. It must be noted that the general convulsive attack almost always leaves the patient face downwards, so that he has drowned in a puddle an inch deep and has been asphyxiated by his own pillow. The latter event is by very far the commonest way the epileptic meets his death from accident in a fit.

The epileptic cry.—There are two quite different sounds that may occur at the commencement of an epileptic attack. The one is a natural, conscious cry of terror at the advent, as in the patient who alternated piercing screams with “It is coming! It is coming!” before the convulsion commenced. It is curious how rarely any memory of such cries or utterances remains with the patient. The other is the epileptic cry proper—a weird, unearthly, hollow sound, produced by inspiratory spasm drawing air over the nearly closed vocal cords. This cry occurs in a minority even of severe cases, for the obvious reason that it is determined by a particular march of the spasm. If the inspiratory spasm occur before the larynx has gone into spasm or after it is in spasm, there can be no laryngeal noise, but only the commonly witnessed pharyngeal and buccal grunting and gurgling. The spasm must be so timed that the inspiratory spasm must occur as the larynx is closing, and this only obtains in a minority of the cases.

Tongue-biting.—Some patients always bite the tongue, others never, and some now and again. The tongue is always bitten at the side and some way from the tip, because it is deviated to one side in the spasm and its thicker part brought between the molar teeth. The same side is always bitten. The tongue cannot be bitten unless protrusor spasm occur either before the jaw has gone into tonic spasm or after it has broken into clonic spasm. If any other march of spasm occur, the tongue escapes. It is remarkable how little scarring occurs even from severe and repeated tongue-biting unless a piece is bitten clean out.

Incontinence.—Though common, incontinence is by no means the rule even in severe attacks. More often it is the urine alone that is evacuated, much more seldom the bowel alone, still more rarely both. A rare phenomenon during an epileptic fit is seminal emission. The occurrence of this has been denied, but the present writer has seen it occur.

Secondary events.—The degree of asphyxia during the attack may

be severe, and blood vessels may give way under the stress, with the production of surface ecchymoses or deep hæmorrhages, including cerebral hæmorrhage. The spasm is powerful and may give rise to much subsequent aching, as if the patient had been beaten all over. It may dislocate joints, rupture muscles and even break bones. A dislocation once produced in a fit always recurs with subsequent fits.

Duration of epileptic attacks.—Two minutes may be given as an outside time-limit for the duration of an individual attack, from its commencement to the end of the active phenomena, and in convulsive attacks to the end of the spasm. Usually the time is much shorter than this, and often is a few seconds only. Sometimes attacks are described as of much longer duration. When analysed, such attacks will be found to be a series of attacks with very short intervals, or slight attacks with post-epileptic functional spasm, or hysterical attacks.

Conditions after attacks.—The epileptic fit may leave no after-effects whatever, even though it be severe, but this is unusual. On the other hand, even the slightest attacks may cause conspicuous sequels. Sleep and headache are very common, especially following convulsive attacks, and they may be alternative effects, in that if sleep occur there is no headache, but if it be prevented there is severe headache. The post-epileptic paralysis of Todd has already been described, and also the aphasia which may follow right-sided attacks. The mental state is usually affected by the attack, and returns to the normal—sometimes quickly, sometimes slowly. Commonly the patient is dull and dazed, speaking at random, unreceptive, irritable, and does not fully recognise his surroundings. During this state of impaired consciousness he may pass into a condition of mental automatism, in which various acts are performed in a conscious manner but of which no recollection is afterwards retained. One patient always prepared for bed after her minor attacks, and proceeded to undress in the stalls of a theatre. The acts performed during post-epileptic automatism may have a true relation to the life and mentality of the patient. He may do spiteful and criminal acts to those he dislikes. This fact has an important bearing as regards the criminal responsibility of the epileptic. In other cases a patient after recovering from the epileptic fit passes at once into a state of hysterical convulsion. Both these post-epileptic conditions occur commonly after minor attacks, but they may also occur after major fits; they seldom occur when convulsion has been severe.

Vomiting may occur after any type of epileptic fit, but it is most often met with after a convulsive attack. As it occurs during the period of unconsciousness, there is some danger of the vomited material being drawn into the larynx. Though Gowers mentions a case in which this event proved fatal, I have not come across any accident from this cause.

MENTAL DETERIORATION AND ABERRATION IN EPILEPSY.—Many epileptics, especially those who have frequent attacks, show signs of mental deterioration, which is often progressive, and which may become severe and end in chronic insanity; while others show no such mental troubles, and some of these fulfil a long life with the highest standard of capacity.

There seems to be no correlation between the type of epilepsy and mental degeneration, though the latter is widely held to be more frequent and more severe when many minor attacks occur.

The tendency to mental failure is greatest in the cases which commence in childhood, and lessens as age increases ; while, again, in the epilepsy commencing in the degenerative period of later life, the incidence again increases. In its slighter form there is merely defect of memory, of attention and power of acquisition. In more severe degree there is greater imperfection of intellectual power, weakened capacity for attention, and often defective moral control. Mischievous restlessness and irritability may develop to vicious and criminal tendencies with advancing age. Every grade of intellectual defect may be met with, to actual imbecility. Paroxysmal outbursts of mental derangement may be met with, sometimes transient and immediately following a fit, sometimes without a fit, and sometimes lasting for weeks or months. From what has been written above upon the cause of the mental disturbance in metabolic dyscrasia, these events will be easily explicable.

PERIODICITY.—While some patients may have fits at any time and at all times, yet there is a tendency in the majority for the attacks to occur at particular epochs and not at others. Epilepsy may be strictly “nocturnal” or “diurnal.” It may occur only on rising in the morning, or solely at the menstrual epoch. The fits may come in batches of several in one day, at intervals of many months, while 7-, 14- and 28-day periods are common. A knowledge of the periodicity when present is of great value in the successful treatment of epilepsy. “Rare” fits, which occur at very long intervals, are apt to present the most severe convulsion ever witnessed.

SPECIAL VARIETIES OF EPILEPSY

EPILEPSY FROM LOCAL DISEASE OF THE BRAIN.—Almost any lesion of the cerebral hemispheres may produce symptomatic epilepsy. But not more than 5 per cent. of all such lesions do this. The convulsions which may occur in cerebral thrombosis, encephalitis and meningitis are examples of epilepsy incident with the onset of an acute lesion. Usually the epilepsy is incident when the lesion has been present some considerable time. Lesions of the brain in childhood seem to be more commonly associated with epilepsy than when occurring in adult life. Agenetic states of the brain of prenatal origin (cerebral diplegias) are associated with epilepsy in 30 per cent. of the cases, and infantile hemiplegia is followed by epilepsy in about the same proportion. Increased intracranial pressure alone seems capable of causing fits, as in hydrocephalus and subarachnoid hæmorrhage, and this may be a factor in the epilepsy of intracranial tumours and meningitis. Abscess seems very rarely to produce fits.

The fits caused by local lesions may be in almost every respect identical with and indistinguishable from the usual type of epileptic manifestation, from the slightest momentary minor fit, all through the local sensory and motor fits, to the severe general convulsion of instantaneous onset and immediate loss of consciousness. There are the same auras and the same sequels. It may perhaps be said with relative truth that the splanchnic auras (epigastric, cardiac, etc.) are uncommon, and that there is a greater tendency for consciousness to be lost late.

The minor attack is the least common fit occurring as the result of a local lesion ; the general convulsion by far the most common ; while the local fit

holds an intermediate position, and its nature is often indicative of the position of the lesion.

PKYNOLEPSY.—This is a form occurring in children, so called because of the great number of the fits which may occur daily. These are of the slight minor type, any sign of spasm being infrequent. It is rare for any major fit to occur. There is no mental impairment whatever, no deterioration of health, and no result is obtained by any form of treatment. The malady invariably ends in spontaneous cure, usually before or at the age of puberty. Its separation from minor epilepsy is of uncertain validity.

CARDIAC EPILEPSY.—This is a convenient term for the epilepsy which occurs in Adams-Stokes' disease, and in paroxysmal tachycardia, and for the fits which may occur in congenital heart disease and in some forms of cyanosis. They cannot be the equivalents of asphyxial convulsions, for they are not met with in severe chronic cyanosis, and, on the other hand, there is usually no cyanosis at all when fits occur in Adams-Stokes' disease.

VASOVAGAL ATTACKS.—Under this misleading title, Gowers described a recurrent paroxysmal symptom-complex with some or all the following components: a sensation of fullness in the epigastrium; precordial pain or discomfort; difficulty in breathing; a sense of impending death; a slowness of mental operations but without disturbance of consciousness; a sense of physical fatigue; and coldness of face and extremities. These symptoms wax and then wane gradually, and may be present for as long as 4 hours from onset to disappearance.

Gowers stated that he used the term "vasovagal" as a purely descriptive one, but without implying any theory of causation. Unfortunately, those who have adopted his terminology have overlooked its lack of foundation. Further, the various descriptions of these attacks to be found in the literature are based almost wholly upon hearsay, the attacks themselves being but rarely observed, and do not provide any evidence of vasovagal involvement. Thus, the pulse is said to be accelerated, not decreased or irregular, while the facial pallor and coldness might equally be the result of local vasoconstriction or of splanchnic dilatation. A further vagueness has been lent to the conception by the different senses in which it has been employed. Thus, Collier has used the term for attacks in which convulsions and loss of consciousness occurred, though Gowers expressly stated that consciousness was not disturbed. In short, the term has no precise meaning, no sound basis of observation, and no proper place in neurological terminology.

The alternative term recently suggested for the attacks described by Gowers, namely, "periventricular epilepsy," must also lack value until we have some precise information as to the nature of the attacks themselves. Lewis has pointed out that the term "vasovagal" may rightly be applied to the common fainting or syncopal attacks, and it is better to restrict the term to these.

MYOCLONUS EPILEPSY.—In this group are included: (1) Epilepsy of an ordinary type in which there is much simple epileptic jactitation of the muscles between the fits; (2) cases of Unverricht's myoclonus in which epilepsy is coincident.

STATUS EPILEPTICUS.—In this condition severe convulsion succeeds severe convulsion at short intervals without any return of consciousness during these intervals. It is as if convulsion recurred so soon as the body recovered sufficiently from the exhaustion produced by the last convulsion.

Meanwhile the temperature rises, and may reach a hyperpyrexia. The difficulty in feeding and watering, the severe muscular exertion and the pyrexia add the dangers of acidosis to those of exhaustion, and the patient is very apt to succumb. Status epilepticus must not be confused with frequently recurring fits in which there is some return to consciousness during the intervals, though it frequently develops from such a condition; for the latter are not accompanied by a rising temperature, are more readily subdued, and are not of nearly so severe a prognostic import. If the convulsions cannot be stopped by treatment, the patient usually dies from sudden collapse, or, the fits ceasing, he remains delirious for a while, with rapid heart and high temperature, and dies of cardiac failure. Status epilepticus may be met with in acute lesions of the brain and in chronic lesions such as general paralysis of the insane. It may occur in acute poisoning with lead, bismuth and absinth. It may develop suddenly in any type of epilepsy whatsoever, sometimes without apparent cause, sometimes as the result of over-exertion and excitement, sometimes when medicines which have been regularly administered and which have kept the fits in check are suddenly cut off.

Diagnosis.—The recognition of epilepsy requires a working acquaintance with the nature of its many manifestations and especially of the slight forms, little exteriorised, which may be easily overlooked or misinterpreted. The sudden unexpected onset, without cause, the transiency, the recurrence, and the circumstances of the moment, are useful aids.

From syncopal attacks (rapid lowering of blood pressure) epilepsy can often be distinguished by the slow onset, the gradually increasing pallor or greyness, the distancing of sound, the nausea and flatulence, the presence of an obvious cause, the length and the stillness of a fainting attack.

The hysterical attack is easily distinguished by the fact that only the convulsion of epilepsy can possibly be confused: the other manifestations of epilepsy are never simulated by hysteria. Hysterical convulsion has not the manner nor the march of epileptic spasm. It never begins with conjugate deviation of head and eyes to one side, there is not the orderly spread of convulsion, and there is never but a poor imitation of the sequence of tonic followed by clonic spasms. The movements in the hysterical fit are purposive, spectacular, violent, and are liable to be increased by restraint and are rapidly abolished by complete inattention. The functional fit never occurs except in the presence of an audience, for it would then be purposeless, and it never occurs during sleep, the tongue is never bitten, though other parts of the body and other people may be. There is no transient abolition of the tendon jerks, nor transient appearance of the Babinski plantar response. The sphincters are never relaxed. Intense converging spasm of the eyes is a common feature of the functional attack, but this sign is not met with in epilepsy. When functional manifestations follow slight and rapidly transient epileptic attacks, the distinction between these and purely hysterical attacks is often difficult and sometimes impossible, except after long observation. For the initial epileptic attack may be practically unnoticeable, and the subsequent events may be typical of hysteria and are usually amenable to the same line of treatment. Often some point in the circumstances under which the attack occurs will settle the diagnosis. Any attack having occurred during sleep, or any attack in which the patient has fallen in circumstances of serious danger, as among the traffic of a London street, or any attack

occurring when the patient cannot attract the attention of others, establishes the diagnosis of epilepsy. The best plan is to regard every hysterical fit as possibly epileptic, and every fit of doubtful type as probably epileptic, until time and circumstance bring definite conviction.

Migraine may sometimes closely simulate epilepsy when sudden paralysis, or sensory auras, or visual hallucinations occur without headache. But while the sensory phenomena of migraine may last for 5 to 30 minutes, those of minor epilepsy have a duration of seconds only.

Careful search must be made in every case for all the bodily conditions with which epilepsy may be associated. Papilloedema, headache and vomiting may reveal increased intracranial pressure from some lesion of the brain; while local paralysis, sensory loss, visual or other defect may indicate a local lesion of the brain, past or present, and this may also be suggested by the nature of a local fit. The presence of rickets, infantilism, undue adiposity, etc., may indicate the presence of some definite metabolic dyscrasia or endocrine disorder. Renal function and the condition of the blood pressure should always be examined, for even in early infancy fits may be uræmic and in the recurring epilepsy associated with small white kidney, and with cystic renal disease, the causal disease is frequently unrecognised. Where syphilis is likely, the reactions in the blood and cerebro-spinal fluid should be examined. Lastly, any evidence of chronic intoxication by metals, alcohol, absinth, etc., should be sought for.

Cysticercosis epilepsy should be thought of when the patient has lived abroad. Diagnosis depends upon the palpation of cysts in the tissues, or the shadow in radiograms of calcified cysts in the muscles, or within the skull.

Prognosis.—The outlook in epilepsy is so variable that it is difficult to indicate any but the broadest principles in prognosis. Nor can a definite forecast be made in any case until the result of treatment has been watched for some time; for cases apparently favourable may prove rebellious, and those most unfavourable may turn out brilliant successes. Speaking generally, a cheerful outlook is justified in all cases except those in which there is progressive mental deterioration, and in these the outlook is hopeless in proportion to the rapidity of the mental change. Naturally, in those cases which are associated with serious bodily disease, such as brain tumour, renal disease and hypertension, the prognosis involves that of the exciting condition.

The danger to life from the epileptic attack itself, either directly or indirectly, is not great. However severe the fit, it is extremely rare for death to occur, and when this happens it is from turning over and smothering with the wetted pillow or from choking with the aspiration of vomited material. Injury, burning and drowning may cause death, yet the number of epileptics who meet their death in this way is so infinitely small as almost to remove the danger of accident from practical perspective. In the rare status epilepticus, however, the danger to life may be very great. Spontaneous cessation of the attacks occurs in a small proportion of cases. The convulsive attacks of infancy, which continue for some years after all cause to which they can be attributed has passed away, often cease for ever at the age of 4 to 6 years. Again, after 20 years of age spontaneous cessation is met with, and it becomes more frequent as life advances. It is, in my experience, a

much more frequent event than writers upon this subject, with the exception of Gowers, have been willing to admit.

The probability of cure, arrest or amelioration by treatment may be entertained in all cases where no mental deterioration exists and where no insuperable bodily disease determines the epilepsy, in proportion as the only method of cure—the securing arrest of the attacks for a considerable time by drug treatment—can be adequately administered over a long period. It is greater when periodicity in the occurrence of fits allows these to be anticipated by drug administration. It is much greater when the following out of education, or the continuance of regular employment, allows of a fully occupied and satisfying life, and much less when education is stopped, pleasures and sports forbidden, and the patient condemned to social inferiority and ostracism, and to a gloomy, narrow life of inanition because he has a few fits. It is perhaps smallest when severe attacks occur daily or at short intervals and when both major and minor attacks occur in the same subject.

Treatment.—*General treatment.*—The general principles for the maintenance of health if good, or for its improvement if poor, should be adopted. Whenever possible, no change whatever should be made from the régime of life of a normal person. In childhood, education, discipline and pleasures and school life should be continued upon strictly normal lines, and the adult should continue with work and occupation. No advantage has accrued from the adoption of special diets, such as the abrogation of meat, the exclusion of salt or the use of purin-free foods. The production of a low grade of acidosis by a ketogenic diet is occasionally of value in the epilepsy of children. Alcohol seems to be an excitant of the epileptic attack and should be forbidden.

The forbidding of such pastimes as may be fraught with danger should a fit occur, such as swimming, boating, cycling and car driving, is necessary.

Marriage and pregnancy.—The subject of epilepsy sometimes seeks—but rarely heeds—advice as to the expediency of marriage, both in its effects upon himself (or herself) and in respect of any heritable qualities it may possess. Marriage has no necessary effect upon the course of epilepsy, and, as we have seen, direct transmission of the disease is rare. Therefore the sweeping medical prohibitions once so frequent in these circumstances are not in fact warranted by such knowledge as we possess. Every case must be considered on its merits. It has been noted that in the family history of the epileptic subject, migraine is a far more common antecedent than epilepsy, but no one would venture to advise the migrainous subject against marriage or parentage on any so-called eugenic grounds. In respect of pregnancy it is usual, though not constant, for fits to cease in the epileptic woman when pregnant, and in any event the occurrence of fits at this time constitutes no special danger and is not an indication for the artificial termination of pregnancy.

Institutional treatment.—In cases where there is low mentality, much mental degeneration or insanity, and with frequent fits, where no adequate care and occupation can be provided at home, there is every advantage in a colony, institution or asylum for epileptics. In such patients little or no good can be done by medicinal treatment, whereas regular work, discipline and interest often mitigate greatly the burden of the malady.

Surgical treatment.—There still remains a very widespread impression that local fits and fits following upon injury to the skull are likely to be benefited by decompression. There is, however, little evidence that such procedures benefit epilepsy of any kind. Cases are on record in which a cortical scar, or a meningeal adhesion to the cortex, has become an epileptogenic focus the excision of which has been followed by cessation of attacks. The presence and situation of such a scar having been previously determined by the lumbar insufflation of air and the taking of an encephalogram. In general the dictum of Pierce Clark is just, that "all operative measures upon the brain in epilepsy are allowable only when they are indicated by definite physical signs other than the fits." Thus, in a case of cerebral tumour producing epilepsy, operation is justifiable for the relief of the papilloedema, headache, etc., and with the hope of possible removal or of obsolescence of the growth following the decompression.

Medicinal treatment.—Further than the measures above described, the treatment of epilepsy is purely medicinal. There are two groups of drugs which have a remarkable effect in arresting or mitigating the occurrence of the attacks in epilepsy. They seem to have much the same effect, and may conveniently be combined or alternated in the treatment of any given case. Sometimes one group is found to suit an individual patient better than the other. No advantage seems to accrue from administering these remedies more than twice in the 24 hours, nor from using large doses. Moderate doses, such as will cause no deterioration in bodily or mental health, even if taken regularly and for years, seem to bring about the best results. The first group is that of the compounds of bromine, of which sodium bromide seems to have an advantage over the others, both as regards efficacy and toleration. The organic compounds of bromine are not so useful. Sodium bromide should not be given in larger doses than 25 grains (1·5 G.) to an adult, nor should more than 60 grains be given in the 24 hours. It is conveniently combined with arsenic (min. 2 to 3) in the form of liquor arsenicalis, since this has the effect of checking the occurrence of acne. If it be advisable to conceal the fact that bromide is being administered, Gelineau's "dragées," each of which contains 15 grains of potassium bromide, may be prescribed.

Bromism.—Even in ordinary doses, the bromides may cause some acne of the skin, especially in subjects who are prone to acne, but this is the sole derogatory effect of this remedy, which is of common occurrence. The true bromide rash, which was met with in the early days of bromide treatment when huge doses (even an ounce thrice daily) were in vogue, is highly characteristic. It is hardly ever seen in these days, but I have twice met with it from moderate doses of bromides. Mental dulling and conditions of sub-coma, which may occur from poisonous doses of the bromides, are rarely met with from appropriate medicinal administration, except in elderly subjects. The mental deterioration due to the epilepsy in certain cases is often attributed by the laity to this cause, but this occurs, and sometimes in much greater degree, in the absence of bromides.

The second group is that of the malonyl-urea compounds, of which phenobarbitone (luninal) and soluble phenobarbitone are examples. These are very powerful drugs, and must be used with care. Phenobarbitone has certainly the advantage over soluble phenobarbitone in being more pro-

longed in its action. It is conveniently prescribed in doses of $\frac{1}{2}$ grain to a child, and 1 grain, with a maximum dose of $1\frac{1}{2}$ grains, to an adult. In larger doses it is a powerful hypnotic, and in patients who have idiosyncrasy it may produce a troublesome kind of frenzy. It appears to be a more certain means of warding off attacks for many hours after its administration than is bromide.

Whatever remedy is chosen, whether it be the bromide or luminal or a combination of the two, it is essential if possible to anticipate the occurrence of the fit by the administration of the drug. Thus, if fits are nocturnal only, the remedy is given in a single dose at night, or if diurnal only, in a single dose in the early morning. Again, if, as often happens, the fits occur soon after waking, then the single nightly dose should be used. Or, if the fits occur or are more frequent at the menstrual epoch, they should be anticipated by increased dosage before and during that epoch. With fits that are diurnal and nocturnal, a night and morning dose should be used. As it is less important in patients who have employment when fits occur by night, and often most disastrous when they occur at work, for with the present Workmen's Compensation Act no company will insure a known epileptic, I prefer to give phenobarbitone as the morning remedy and bromide as the nightly remedy, since I consider phenobarbitone to be the greater safeguard against the occurrence of the attacks. The question at once arises, Why should two remedies be used? The answer is that these drugs are by no means identical in action, and that the nature of the cause of epilepsy certainly varies in individuals. Some patients do best on phenobarbitone alone, others on bromide alone, and others on a combination of the two, and the best course can only be determined after trial.

Recently, Merritt and Putnam in Boston have introduced the use of sodium diphenyl hydantoinate (dilantin, epanutin) for epilepsy. Favourable results are claimed for this in cases that have not responded well to bromide or phenobarbitone, and some confirmation of its value is being obtained in this country. Obtainable here under the names of epanutin and solantoin, it is dispensed in capsules containing $1\frac{1}{2}$ grains. For small children dosage is begun with $1\frac{1}{2}$ grains twice daily, increased to three or even four times daily until optimal results are obtained. For adults $1\frac{1}{2}$ to 3 grains twice or thrice daily may be given. Symptoms of intolerance or of overdosage are tremor and unsteadiness, and these call for reduction of dosage. It seems safe to say that for adults a dose of 3 to 4 capsules daily is without untoward consequences, or unpleasant symptoms.

When the change is being made from some other medication to epanutin, this should be gradual, one of the daily doses of bromide or of phenobarbitone being substituted in the first week, a second one in the following week, and complete substitution being achieved in the third week. It is perhaps too soon to estimate the value of this drug in comparison with its many predecessors, but in some instances it is certainly more effective in controlling fits. Its use is said to be contra-indicated in elderly persons with hypertension and in debilitated subjects.

Many other remedies have been advocated in epilepsy; a few only have stood the test of time and are still in use, both as alternatives and adjuvants to the treatment above given. These may be placed in order of merit as belladonna, digitalis and allied drugs, especially adonis vernalis, opium and borax.

Belladonna has a striking effect upon a few isolated cases. It makes some cases definitely worse. Digitalis and adonis vernalis are commonly used as adjuvants, and I think sometimes with benefit. Morphine given in careful doses is of great use in status epilepticus. Thyroid is often of value in the epilepsy associated with cerebral agenesis (cerebral diplegia), mental and bodily backwardness and infantilism. It probably acts by rendering bodily metabolism more normal and in enhancing development.

STATUS EPILEPTICUS.—The treatment of this condition, and that of rapidly repeated fits which not infrequently merges into status epilepticus, is quite different from that of epilepsy in general, for the remedies useful in the latter condition are useless and even do harm in this urgent and dangerous state. The first thing to be done is to check the convulsion, and this is best achieved by the hypodermic injection of $\frac{1}{4}$ th of a grain of morphine. (Gowers preferred hyoscine.) Another remedy is paraldehyde, in large doses (360 minims), and this has recently been successful at the National Hospital. It has the obvious advantage that it is stimulating and not depressant. The remedies formerly used such as bromide and chloral by the rectum are worse than useless. The next measure is to secure that the patient shall be provided with adequate stimulants in the form of food, water and even alcohol. To which end a nutritious liquid meal of high stimulating value and containing sugar to combat acidosis should be given by means of the nasal tube at regular intervals. An action of the bowel should be obtained as soon as possible, with a rapidly acting aperient administered with the food, and by warm water enemata. The pyrexia should be controlled by sponging repeatedly, and if high by continuous immersion, and this alone will sometimes have a dramatic effect in checking convulsion when pyrexia exists. When consciousness returns, feeding and stimulation must be carefully continued, with a gradual resumption of the routine treatment of epilepsy.

NARCOLEPSY.—In this remarkable and, as Adie has shown, by no means rare condition, two quite different kinds of attack occur. The one is the sudden onset of apparently normal sleep, which comes usually at a moment of inattention, several times a day. The sleep lasts from a few seconds to a few minutes; it is rousable, and the patient is wide awake at once and knows that he has slept, and sometimes that he has dreamed and can describe the dream. Many of the patients have a warning in the way of a feeling of intense fatigue and, thereafter, can so far repel the onset of the attack by an effort of will as to be able to get out of harm's way; but the attack is inevitable, and it is always the more severe the longer it is resisted. The second variety of attack is called the "cataplectic" attack, and this is produced mostly from a sudden emotion, which may be of any kind, but is usually an emotion which provokes laughter. There is a sudden feeling of intense weakness in the limbs, which become flaccid. The patient drops anything that he may be holding and crumbles to the ground, but often only into the sitting position. The eyelids drop and the head falls forward with the jaw dropped, and there is sometimes twitching of the muscles of the face, tongue and neck. There is complete inability to move the limbs and generally inability to speak, but consciousness is completely retained, so that the patient is afterwards able to recount every event and repeat every word spoken during the attack. In one typical case a cataplectic access was habitually induced by feelings of amusement or of anger, but

surprise was equally effective. Thus, when fly-fishing, a suddenly rising fish caused the patient's rod to droop in his suddenly weakened arm, and when shooting his gun drooped helplessly if a bird broke unexpectedly into view near him.

Usually an idiopathic malady, narcolepsy has been recorded in a few instances as a sequel of encephalitis lethargica. The malady appears never to be familial, and it does not occur before the age of puberty. Once developed, it usually continues throughout life, with variable frequency of the attacks. In most cases (amphetamine) benzedrine sulphate is specific in its action on narcolepsy. An initial dose of 1 tablet (10 mgm.) after breakfast and another after luncheon. This may be cautiously increased to 2 or even 3 tablets after these two meals, with a smaller dose after tea. Symptoms of overdosage are sleeplessness, restlessness and tremulousness. Less efficacious, but yet of distinct value in some cases, is ephedrine sulphate of doses of $\frac{1}{2}$ grain twice daily.

MIGRAINE

Synonym.—Paroxysmal headache.

Definition.—A common malady of which the only essential characteristic is recurring intense headaches, which usually develop on waking in the morning, and may be unilateral, frontal, occipital or general. The attacks usually date from childhood, but sometimes commence during later life. The headaches are often associated with vomiting, which has given rise to the designation "sick headaches" or "bilious attacks," with which is associated much vestibular disturbance as in sea sickness, and with peculiar disturbances of vision. Less common symptoms of the disease are peculiar slow sensory auræ, which occur in no other malady, attacks of hemiplegia or monoplegia or of aphasia, and attacks of ophthalmoplegia. Some of these phenomena may accompany the headaches, but others occur in attacks quite apart from the headaches, and may for that reason give rise to difficulty in diagnosis.

Ætiology.—The malady commonly makes its appearance at about the age of puberty, and tends to persist, with fluctuations in severity and frequency of attacks, until middle age, disappearing in women with the menopause. Its persistence in old age is exceptional. A history of familial incidence is common, and the subjects are commonly of an energetic and intelligent type.

Nothing is known with certainty of its essential nature of causation, and in consequence hypotheses are rife. Thus, digestive disorders, errors of refraction, of metabolism and of endocrine function have all been evoked as responsible factors, though without clear evidence in the case of any of them. Yet the correction of such errors when they obtain does not suffice to cure the malady, and it is probable that they are never more than precipitating factors in susceptible subjects. Spasm of cerebral arteries has also been suggested as a cause of the attacks, but again without adequate grounds.

The subjects of migraine are often bad sailors and bad train travellers, and mental and bodily fatigue and emotional disturbances are commonly

followed by an attack. It is important to remember that tumours of the occipital lobe and also intracranial aneurysms may be associated with attacks exactly resembling migraine.

Symptoms.—The subjects of migraine are usually otherwise quite healthy, and are often robust and strong. No peculiarities of blood pressure are noticeable. Premontory signs of the attacks are present in some cases, and these may take the form of an unusual feeling of well-being and intellectual acuity, or, on the other hand, of lassitude and depression. The attack commences most commonly on waking in the morning, when on raising his head from the pillow the patient experiences a sense of vestibular disorientation with giddiness, ocular confusion and nausea, such as is commonly felt at the commencement of sea sickness. It is at this stage of the attack, and within a few moments of its commencement, that the visual phenomena occur when these are present. Often the patient vomits at once from the vestibular disturbance, but sometimes vomiting is delayed for hours, and the vomiting may be continued as long as the giddiness persists. The visual disturbances last but a short time (from 10 to 20 minutes), but leave, as a rule, some confusion of vision and discomfort throughout the attack. The headache follows shortly upon these initial symptoms. It is cumulative, and expansile in character, and often begins constantly in a localised spot in the temple, forehead or eyeball, as a sharp boring pain which gradually spreads, and may involve the neck and arm. The pain may be unilateral, frontal, occipital or quite general. As the headache increases the face becomes pale and grey, the patient becomes much prostrated and incapable of mental or physical effort, and is unable to take food. Light, noise and movement aggravate the pain. After remaining in this condition for many hours, he falls into a heavy sleep, and awakes next morning shaken by his illness, but otherwise well. The above description covers many attacks of migraine, but many variations occur. The attacks do not always occur on waking, they may come on at any time of night or day. They may be rapidly transient, lasting but a few hours only, or they may last for days and even as long as 3 weeks, and give much anxiety in the attempts to provide nourishment and sleep for the patient. In some cases of long standing, the attacks become less severe towards middle life, and a persistent aggravating headache may develop between the attacks. When such a persistent headache is complained of alone, it is very important to inquire about preceding migraine, for the same treatment is applicable to the two conditions. One of the most characteristic features of the headache is that when once it is in full swing, no remedies will relieve it except natural sleep.

Visual phenomena.—Considering how very common migraine is, it must be clearly understood that any visual phenomena except slight confusion of vision accompanying the attacks, are rare. These may take the form of general mistiness of vision, floating spots, scotomata, bright stars and colours, hemianopia, double hemianopia with complete blindness, or psychic hallucinations of vision. In connection with scotoma and with hemianopia, the phenomenon of teichopsia may occur as follows: Upon the dark background of the scotoma or hemianopic field, a ball of light appears, which grows larger and becomes dark in the centre. This ring of light breaks at one spot, opens out and takes the form of a series of entering and retreating

angles (castellation figure) which become gloriously coloured (fortification spectrum) and which later become fragmented and fade. These visual events usually occur at the very beginning of the attack, before the headache develops, and they are rapidly evanescent, but they may occur as isolated phenomena, when no headache occurs.

Aphasic attacks may take the form of confusion of speech, word-blindness, or even of loss of speech-acceptance and exteriorisation. They accompany the headaches and occur at the commencement of the attacks. They are not of common occurrence.

Hemiplegic and monoplegic attacks usually occur quite apart from the attacks of headache. They, too, occur on waking, and consist of a transient uselessness and weakness of limbs, which lasts a few hours only. They are characterised by their occurrence in young subjects who suffer from pronounced migraine; they are rapidly transient and are not accompanied by organic signs, and almost invariably other members of the family are migrainous, and suffer with similar attacks of paralysis. In Michell Clarke's cases, 11 members of one family in three generations were so affected.

Sensory aura.—These are somewhat rare events, but they are pathognomonic of migraine, and usually occur quite apart from the headaches. The aura commences upon the periphery of a limb and is likened to that which would be produced by a multitude of cold-footed insects creeping on the skin. It travels very slowly towards the proximity, taking half an hour or more to reach from the fingers to the head, and is very alarming to the patient. It disappears rapidly without further event. It is the only aura with an exceedingly slow spread.

Ophthalmoplegia.—This is a very rare but most important event. It occurs only at the height of the headache, in severe attacks. Indeed, the patients usually say that the headache, during which the ophthalmoplegia occurred, was the very worst they had ever experienced. It is a paralysis of the oculomotor nerve trunks, most commonly of the sixth nerve alone, but sometimes of the third or fourth nerves, or of a combination of these three. It is generally unilateral, but may occur simultaneously on both sides. Severe diplopia results. It passes off in from a few days to a few weeks. When once it has occurred, it is apt to recur with subsequent attacks. In one of my patients, paralysis of the sixth nerve persisted for 18 months, gradually lessening between the attacks, and becoming complete with each fresh attack of headache. It disappeared completely, with the cessation of the headaches, when adequate treatment was adopted.

Diagnosis.—This presents little difficulty, if it be borne in mind that long installed recurring headaches on waking in the morning are surely migraine. The condition of the urine should exclude those renal cases with migraine-like headaches. Each case should be carefully examined for signs of organic nervous disease, and especially for persistent hemianopia and papilloedema, which would indicate an organic lesion of the occipital lobe. Those who are not familiar with the full range of sensory symptoms that may precede the onset of the headache, and do not realise the severe disturbance of the speech function which in some cases accompany them, are apt to take an unduly grave and erroneous view of the history given by a subject who has experienced them. Thus, a diagnosis of petit mal, or of cerebral tumour is not rarely made. It should be remembered, therefore, that the disturb-

ances which occur in a minor epileptic attack are momentary in duration and never persist, as do the migrainous symptoms in question, for several minutes. Again, consciousness is neither lost nor blunted in migraine. Although, as has been pointed out, a rare case of migraine ultimately turns out to be one of cerebral tumour, the latter diagnosis cannot readily be made unless there are supporting signs of a structural lesion. It is probable that the so-called ophthalmoplegic migraine, in many instances at least, is a symptom-complex bearing no relation to true migraine but dependent upon a gross intracranial lesion, perhaps most often an unruptured aneurysm of the internal carotid artery.

Treatment.—There are but few cases of migraine that cannot be materially benefited by treatment. Some are completely cured, while in others the attacks become milder and occur at much longer intervals. Careful attention should be paid to improvement of nutrition and general health, should these be defective. The avoidance of undue fatigue and of worrying emotions, and of any factors which are known to produce the attacks, is important. Errors of refraction should be adjusted if they are important. Medicinal treatment is by far the most useful agent. A mixture containing min. 1 of liq. trinitrinæ, min. 5 of liq. strychninæ, min. 10 of tinct. gelsemii, and 10 grs. of sodium bromide, made acid to preserve the stability of the nitro-glycerine, and administered thrice daily for many weeks or months, was advocated by Gowers; and truly there are few cases of migraine which do not derive great benefit or complete cure from this treatment. Phenobarbitone, in doses of 1 grain given every night, is most valuable, and may be used in addition to the foregoing prescription. The individual attacks are difficult to relieve. Sometimes a full dose of phenazone, acetanilide, phenacetin or aspirin given at the very commencement of the attack will ward it off, but these are useless when the headache has fully developed. The use of ergotamine tartrate—by mouth or hypodermically—has recently been recommended as an effective method of cutting short an attack of migraine. In some subjects, though by no means in all, it is useful for this purpose. Sometimes a full dose of alcohol has the same effect. It remains to keep the patient as comfortable and quiet as possible till sleep occurs. Where the attacks last over the 24 hours, and especially when they last for days, the only remedies are to induce sleep and to keep the patient nourished, the latter object being difficult to attain in long-lasting attacks. To this end hypnotics, such as barbitone or carbromal (adalim) may be used.

JAMES COLLIER.

Revised by F. M. R. WALSHE.

CHOREA

Synonyms.—St. Vitus' Dance; Sydenham's Chorea; Rheumatic Chorea.

Definition.—Chorea is an affection of the nervous system caused by rheumatic infection, and characterised by the occurrence of spontaneous involuntary movements, irregular both in time and in place of occurrence and in nature; by inco-ordination of voluntary movements; by muscular weakness, and by a variable degree of psychic disturbance.

Ætiology.—Chorea is rare among negroes, Indians and coloured races, whilst it is especially common in Jewish races. It is much more common among the poorer classes than among the well-to-do. Its incidence is upon nervous highly-strung subjects rather than upon the phlegmatic, and this is probably to be explained by the fact that the rheumatic subject is likely to be nervous and highly strung. Chorea is practically unknown during the first three years of life, and is very rare before the fifth year has passed. Common between the ages of 5 to 10 years, it reaches its maximum incidence between 10 and 15 years. After the age of 20 it is rare, except in connection with pregnancy; but a few cases have been reported up to the age of 60 years which have certainly been examples of rheumatic chorea. Females are affected twice as frequently as are males. Heredity concerns the incidence of chorea in two ways: firstly, as regards the inheritance of the rheumatic tendency, which is the most important cause of chorea; and secondly, in respect of the inheritance of the neuropathic tendency, for it is when these two are coincident that chorea is most prone to occur. As early as 1802 rheumatism was regarded as the cause of chorea, and all subsequent investigations have upheld this theory. The family history of a choreic patient generally brings to light the occurrence of acute rheumatism, of cardiac disease and of other rheumatic manifestations among other members of the family. Often the patient has suffered with rheumatic erythema, purpura, rheumatic nodules, recurrent sore throat and growing pains before the appearance of the chorea; less often an attack of acute rheumatism or cardiac disease has occurred. A large percentage of those patients who have never shown any sign of the rheumatic state before or during the attack of chorea subsequently suffer with rheumatic symptoms. The British Medical Association Collective Investigation Committee found that rheumatism preceded the chorea in 26 per cent. of the cases, and that in 46 per cent. of the remainder rheumatic signs accompanied the chorea, or appeared subsequently. If to the total of choreic patients who present rheumatic signs at some time or other, one adds those with no personal history of rheumatism, but with a family history of rheumatism, it will be found that there are but few cases of chorea in which a personal or family history of rheumatism is absent.

Psychical disturbances.—Any emotional disturbance, such as fright, anxiety, depression or overpressure in school, may sometimes act as immediate determining factors, but much more often these events simply aggravate symptoms which are already present in slight degree.

Pregnancy.—The relationship of pregnancy to chorea is very definite. It is generally met with in first pregnancies, and before the age of 25 years, and in most cases the pregnancy appears to be the only obvious cause for the chorea. The onset of the chorea is usually between the first and third months of pregnancy. It is liable to recur with subsequent pregnancies.

Pathology.—The complex nature of the involuntary movements in chorea, and their association with psychical disorders of greater or lesser degree indicate the cerebral cortex as the seat of the pathological process. The essential lesion has proved very difficult of detection by microscopical investigation, but according to Greenfield and Wolfsohn it consists in a diffuse meningo-encephalitis affecting mainly the cerebral cortex, the basal ganglia and the pia-arachnoid.

Symptoms.—The onset is usually gradual, but it is sometimes abrupt,

when emotional disturbance has been the determining cause. The appearance of choreic movements is often preceded by alterations in the mental and physical condition of the child. She becomes nervous and more impressionable than before. She is increasingly unable to apply her attention. She becomes clumsy in her movements—and lets fall objects which she is holding. Anæmia, apathy and languor and irregularity of appetite are commonly present. At this time, careful observation will discover slight involuntary movements of the face and fingers which are often unilateral in distribution. From day to day the movements become more marked and spread to the limbs and trunk. The face is constantly grimacing, and the hands and arms scarcely cease from turning about, and affection of the legs makes the walking irregular and clumsy. The child can no longer keep still, the respiration movements become irregular and spasmodic, and the chorea is fully developed. The characteristic symptoms of a well-marked case of chorea are—(1) involuntary movements; (2) weakness of voluntary movements; (3) ataxy or loss of precision of voluntary movement; (4) emotional instability and other psychic disturbances.

1. THE INVOLUNTARY MOVEMENTS are always irregular as regards time and as regards the nature of the movement. Similar movements are never repeated successively in the same part. Each movement begins rapidly, and ends suddenly, and one frequently sees the involuntary movement complicated by the addition of a voluntary movement to cover the fault. The majority of the movements are complicated, involving several muscles and often more than one joint. In the face, the more simple movements take the form of asymmetrical twitches in the lips, and about the angles of the mouth and orbits. In more severe cases, the strangest grimaces may occur. The tongue is thrust into one cheek, then projected just in time to escape the sudden snap of the open mouth. When asked to show the tongue, the child puts it out rapidly and holds it there by closing the teeth upon it. Smacking of the tongue and palate may often be heard at a distance. Lateral movement of the jaw is common. According to the severity of the case, speech may be difficult, the words being articulated slowly in slurred monosyllables. For the same reason, swallowing may be difficult or impossible in severe cases, and may necessitate nasal feeding. That the ocular muscles participate in the involuntary movements only in very severe cases.

In the upper extremities the movements appear first in the hand. The thumb is more restless than the fingers, which are spread and pressed together, flexed and extended, alternately; the wrists twist about irregularly, the forearms are constantly agitated with movements of pronation and supination, flexion and extension; while all possible movements of the shoulder occur. When the upper extremities are outstretched, the hands assume the position of flexion at the wrist and over-extension at all the finger joints in so many of the cases as to make this a characteristic feature of chorea. The lower extremities are less severely affected than is the rest of the body, and here the movements are best seen when the child is lying down. The gait tends to be clumsy and insecure, and in severe cases walking becomes impossible. Alteration of the rhythm of the respiratory movements is conspicuous and is highly characteristic of chorea. The breath is often taken rapidly and held for some time, then let go with a loud sigh. The trunk is often involved, and movements of a writhing nature are characteristic.

So far as the limbs are concerned, the movements may be confined to one side, more commonly the left side, and the condition is then called hemichorea; but the involvement of the face and trunk is always bilateral and is generally equal upon the two sides. In hemichorea, the movements are always of slight severity. Severe chorea is never confined to one side. Choreic movements cease during sleep, and, except in severe cases, can be controlled more or less by voluntary effort; the attempt to write, for example, will generally cause cessation of the movements in the right arm for the time being. They are generally increased by observation, emotion and self-consciousness, but in a few cases it will be found they are worse when the child is alone and unobserved. The violence of the movements of the limbs may cause the skin over the prominences to ulcerate from friction against the clothing, and the head and limbs may be badly bruised from contact with adjacent objects, and unless the patient be properly protected, wounds may occur, which are liable to infection with such grave consequences as abscess, erysipelas and pyæmia.

2. LOSS OF POWER is shown in the mild cases by incapacity for exertion and undue fatigue. More severe degrees of paresis may accompany or succeed the appearance of the movements. It may be observed that in one limb, or upon one side of the body, the choreic movements are becoming less marked, and that the limbs are becoming progressively weaker. Soon the arm hangs loosely by the side, and the leg is dragged in walking. The degree of choreic paralysis bears no relation to the severity of the movements, for the former may be severe, when the latter are slight and vice versa. Choreic paresis is apt to return with successive attacks of chorea, but not always in the same region.

Limp Choreia (chorea mollis).—This is a more severe degree of choreic paralysis affecting the whole musculature. It may be preceded by the usual symptoms of chorea. More often the paralysis is the first noticeable symptoms, and this develops rapidly in from 24 to 48 hours. The paralysis is characterised by complete flaccidity of the limbs; the child lies upon its back and does not move, and if one of the limbs be raised from the bed and then released, it falls limp and lifeless. The head is no longer held in a natural position, but falls round on to the ear. Careful investigation, however, rarely fails to reveal some slight choreic movements, either in the face or in the fingers. Paretic chorea and chorea mollis run a benign course, and recovery is said to be almost invariable.

3. INCO-ORDINATION OF VOLUNTARY MOVEMENT may be the first symptom of chorea to draw attention, and it may precede the appearance of the choreic movements. It may be very obvious when the movements are slight, and it is most noticeable in those of the hand and forearm, which lack precision, and in those of articulation, deglutition and respiration. The involuntary movements that have been described are superimposed upon voluntary movements which they render inco-ordinate, at times interrupting them abruptly and at other times tending to prolong them.

4. PSYCHICAL DISTURBANCES are common, some degree of emotional instability, failure of attention and depression being present in most cases, and, generally, in proportion to the severity of the affection. Delirium may occur in acute and grave cases. It is usually violent and loquacious, and resembles other forms of toxic delirium, and it is of serious prognostic import.

Mania is quite exceptional in children, but it is not an uncommon complication in adolescents and adults. The form of the aberration may be acute mania, melancholia or delusional insanity. The psychical disorders, slight or severe, usually disappear with the chorea, and in all cases the prognosis as regards permanent mental recovery is good.

OCULAR PHENOMENA.—The pupils are frequently dilated and may be unequal and excentric, and hippus may be present.

Sensibility is not impaired. The sphincters are not affected. The skin reflexes are normal. The deep reflexes are also normal in a large proportion of cases, but often the knee-jerk shows an alteration which is peculiar to chorea. On tapping the patellar tendon, the resulting contraction of the quadriceps is unduly sustained, and the leg remains in a position of extension at the top of its excursion for several tenths of a second. This is not invariably present even in a series of knee-jerks taken successively from a single subject, and the occasional appearance of an apparently sustained jerk is due to the coincidence of reflex contraction and choreiform movement. In severe cases, the deep reflexes may be diminished and rarely may be absent for months.

RHEUMATIC MANIFESTATIONS.—Cardio-vascular changes are common in chorea. In nearly all the cases, careful and repeated examination of the heart will reveal slight dilatation and reduplication of the second sound, often with reduplication of the first sound, and increased rapidity of the pulse. Doubtless these are signs of a slight myocardial weakness, resulting from the rheumatic infection. Irregularity of the pulse is probably dependent upon the altered rhythm of respiration. Systolic murmurs are common, and these may be hæmic in nature, or may be the expression of cardiac dilatation, but in the majority of cases they are indicative of endocarditis. Endocarditis is present in 90 per cent. of the fatal cases. At least one-half of all cases present cardiac murmurs, which are suggestive of the presence of endocarditis, while some cases with no cardiac murmur during life are found post mortem to have endocarditis. The mitral valve is commonly affected, lesions of the aortic valve being quite rare. Pericarditis is a frequent associate of endocarditis; only in rare instances does it occur alone. The valvular affections which are met with in chorea may be the result of antecedent rheumatism, or they may develop in the course of the chorea; or while no signs of endocarditis are present during the attack, the patient may shortly afterwards present the signs of organic valvular disease. Cutaneous affections which occur in rheumatism are met with also in chorea, namely, erythema, purpura and subcutaneous nodules. Acute articular rheumatism is comparatively rare, and when it occurs it is usually accompanied by a cessation of the choreic movements. When rheumatic phenomena are present and in the acute mania of chorea, pyrexia is usually present, but uncomplicated chorea is an apyrexial disease.

RECURRENCE.—One-third of the subjects of chorea have more than one attack. Females are more prone to a recurrence than males in about the same proportion as they are more liable to original attacks. The average interval between the attacks is one year. If, therefore, a patient has remained well for 2 years, it is improbable that a recurrence will take place. The greater the number of choreic attacks, the more likely is the heart to be found affected, and, therefore, cardiac complications are more often met with

in recurrences. In a recurrence of chorea the symptoms are usually less severe and their duration shorter than in the original attack.

Course and Prognosis.—The disease tends to a spontaneous termination after a variable time, which is usually from 6 weeks to 6 months. The duration rarely falls short of the earlier period. The average duration of cases treated in hospital has been found to be 10 weeks. Cases which last for more than 12 months are not rare, and slight cases with remissions may last several years. The course of the malady is that after a gradual development of symptoms, there is a stationary period during which symptoms are well marked, followed by a period of gradual diminution. In some of the more severe cases of chorea where deglutition is difficult the patient is likely to be insufficiently fed; and this constitutes a grave danger, since in the condition of semi-starvation so induced, the chorea develops apace. Articulation and swallowing become impossible, and the movements become ceaseless, so that both rest and sleep become impossible; the patient wastes rapidly, and is in danger of death from exhaustion unless prompt measures for restoring the depleted nutrition are taken. This is the condition known as "chorea gravis."

The proportion of fatal cases occurring in chorea is less than 2 per cent. Death is most often met with in first attacks, occurring about the age of puberty, and it is very uncommon in young children and in recurrences of chorea.

Diagnosis.—The nature of the involuntary movements of chorea is usually so characteristic as to make diagnosis easy, and to avoid any confusion with other maladies which present conspicuous involuntary movements. Nevertheless, occasionally a case of multiple tics in a child does present difficulties, for the movements are not—as is so commonly stated—invariably repetitive. In chorea the involuntary movements may lead to the dropping of objects from the hands. This does not happen in the case of tics. Again, when the choreic subject gives the observer a firm and sustained handclasp, the irregular waxing and waning of the muscular contraction may be felt throughout by the observer. In a case of tics, the contraction is steadily maintained as in the normal subject. In myoclonus, the movements are short and shock-like, while in athetosis they are slow and rhythmic.

Treatment.—It is all-important in the treatment of chorea, from the mildest to the most severe cases, that physical and mental tranquillity should be secured. It is well to commence treatment in every case with several days' absolute rest in bed, provided that such treatment can be carried out without entailing the fretting which enforced imprisonment may produce. A bright room, an interesting companion, and varied amusements during the period of rest, are desirable, and isolation from other children is advantageous. It is, however, better to abandon enforced rest than to allow it to become irksome to the patient, and result in mental depression and emotional upsets—conditions above all things to be avoided.

When absolute rest is considered inadvisable, or after it has been carried out, the ordinary periods of rest should be prolonged. The child should be well clad in woollen garments, especially at night, since the spasmodic movements are liable to leave her uncovered. Improvement in the condition of bodily nutrition is to be aimed at in all cases. Choreic children are mostly

ill-nourished and thin, and the effect of a liberal supply of food and nutritious food upon the course of the disease is striking. When swallowing is difficult, it is best to resort at once to nasal feeding, which rarely causes as much discomfort as the ineffectual and exhausting endeavours to take food with the spoon. A china feeding-cup must never be used, since the spout may be broken off; an enamelled metal cup is safe. It has been pointed out above that chorea gravis is dependent upon a condition of relative starvation, and here nasal feeding should be employed, and the meal should consist of strong beef-tea, Bengel's food, lactose and milk; it should not measure more than three-quarters of a pint for a child of 8 years old. Severe cases, in which the movements are violent, call for skilled attention, and a trained nurse is required night and day. The patient should lie upon a water mattress, placed upon a large guarded bed, the sides of which are everywhere protected by pillows, which must be fixed. When a cot is used, it is easy to pad all the ironwork with cotton-wool, over which bandages are wound. If the limbs are injured, they should be wrapped in cotton-wool applied with a light bandage.

When the patient is improving, measures calculated to enhance control of the limbs, such as exercises under supervision and simple drill, are very useful in hastening the disappearance of the movements. Warm and tepid baths and douches applied regularly and in such a way as to be grateful to the patient, and to produce no fright, are very useful adjuncts. The compounds of salicylic acid are of great value, and of these aspirin is the most useful. It should be given thrice daily after meals in doses of 10 grains for a child between the ages of 6 and 14 years, and 15 grains for an adult, and it should be continued until convalescence is complete. It is well borne and has no deleterious effect, and even more frequent doses may be given. A larger dose given at night is the best remedy for sleeplessness. In very severe cases, the administration of hyoscine is sometimes very useful. It should be given in doses of $\frac{1}{100}$ th grain thrice daily. The administration is followed immediately by wide dilatation of the pupil and slight flushing, and by peaceful sleep. The bromides have little or no value as sedatives. In addition to the above remedies, tonics such as iron, glycerophosphates, hypophosphites, strychnine, cod-liver oil and malt are often valuable, especially during convalescence.

HUNTINGTON'S CHOREA

Synonym.—Hereditary chorea of adults.

This is a somewhat rare disease, in which symptoms almost identical with those of rheumatic chorea, namely, involuntary spontaneous movements, ataxy, paresis and slow and slurring articulation, gradually appear in adult life, and usually about the age of 40 years, and are accompanied by progressive mental failure, with delusions and suicidal tendency. The choreic movements are never severe, but the inco-ordination may be well marked. Maniacal outbursts are not uncommon. The disease always progresses slowly to a fatal termination in from 5 to 30 years, and treatment is entirely unavailing. It is a familial disease, and the transmission is direct from parent to child; but if a generation escape the malady, it seems not to reappear subsequently. Sporadic cases, in which no heredity can be traced,

do, however, occur. The sexes are equally affected. Further than the heredity no causal factors are known. The morbid anatomy consists in a slow progressive degeneration of the nerve-cells of the cerebral cortex and basal ganglia, with consecutive atrophy of the convolution, neuroglial overgrowth and meningeal thickening.

SENILE CHOREA

A malady in which typical choreic movements constitute the chief feature is met with in elderly people, and is possibly due to a progressive neuronc degeneration in that region affected in the other forms of chorea. It differs from Huntington's chorea in the late onset, the absence of heredity, and in the absence of mental changes.

MYOCLONUS

Synonym.—Paramyoclonus multiplex.

The characteristic symptom of this condition is the occurrence of sudden shock-like contractions of the muscles, which may vary in intensity from simple fibrillary twitching to contraction which causes a violent movement of a limb. The movements are often symmetrical, and are especially incident in the proximal muscles of the limbs.

Ætiology.—The malady appears in children usually between the ages of 5 and 15 years, while in adults it commences between the ages of 25 and 40 years. Both sexes are liable to the affection. Many instances, in which several children of the same parents have been affected, have been recorded, and in a few the malady has been transmitted through several generations. Nothing further is known as to the causation.

It is probable that the seat of the morbid process is in the cells of the cerebral hemispheres, since myoclonus is further associated with epilepsy and with progressive mental failure.

Symptoms.—The movements of myoclonus are simple sudden movements, and may exactly resemble the movement resulting from a single faradic stimulus. Each movement commonly involves a single muscle only, and it may concern no more than a few fibres, resembling then the fibrillary twitching common in progressive muscular atrophy. In other cases, many muscles may be implicated in the shock-like spasms, which may be of so violent a nature as to throw the patient to the ground. The distribution of the contraction is never determined by that of the nerve supply, nor do the muscles contract according to their synergic association. Myoclonic movements are irregular as regards rhythm and range of successive movements. The upper limbs are more affected than the lower, and the proximal parts more than the distal, while the periphery, the hand and foot, often escape. Voluntary muscular effort usually checks the myoclonic movements, but in rare instances it excites or augments the spasm. The electrical excitability of the muscles is unaltered, and there is no muscular wasting, but the mechanical excitability of the muscles is increased, and percussion of a muscle may evoke the spasms. The sphincters are unaffected. The reflexes, both superficial and deep, are normal. Sensory phenomena are

absent. Speech may be seriously interfered with when the muscles of jaw, tongue, palate and larynx are implicated, and spontaneous laryngeal and pharyngeal noises may occur. The ocular muscles seem never to be the seat of the movements. Epileptiform convulsions are present in some cases, and for these the term "epileptic myoclonus" has been used.

Diagnosis.—This is not difficult when the simple shock-like movements in symmetrical muscles, without any resemblance to volitional movements and entirely destitute of rhythm, occur in this disease alone.

Course, Duration and Prognosis.—Myoclonus, as a rule, is a slowly progressive affection up to a certain stage, and when this is reached it may remain stationary for years, having little tendency to shorten life, death ultimately occurring from some other disease, without any period of freedom from the spasms. Rarely the disease has ended fatally within a few months of the onset, with progressive mental failure and coma.

Recovery may take place spontaneously, or as a result of treatment, but the affection is very prone to recur.

Treatment.—Every available measure should be used to improve the general bodily condition so as to bring about a more stable condition of the nervous elements, by improving their nutrition. The only drug which influences the disease is arsenic. It must be borne in mind that the malady is an intractable one in proportion to the time the symptoms have persisted, and that some cases recover spontaneously.

SPASMODIC TORTICOLLIS

Definition.—A disease of the nervous system, characterised by tonic and clonic contraction of the superficial and deep muscles of the neck, causing the head to assume either a position in which it is turned to one side and upwards, or a position of marked retraction (retrocollic spasm). It is more correctly to be regarded as a disturbance of movements than of muscles, and perhaps, physiologically considered, it may be spoken of as a disorder in the carriage of the head. This carriage is a more complex and highly co-ordinated function in the erect posture than in the quadrupedal posture; it is a function peculiar to man, and in this sense is of recent evolutionary development. We may perhaps see in this a factor determining its frequent derangement, as in spasmodic torticollis.

Ætiology.—The disease is most frequently met with in middle-aged adults, but it may occur at any age from puberty onwards. It is twice as frequent in females as in males. The causation is most obscure. Not infrequently neuropathic heredity, such as epilepsy and insanity, exists, and the patients are often of highly-strung, nervous, irritable dispositions. Nervous shock, prolonged anxiety, and general ill-health have frequently preceded the onset of symptoms. Less often local strain, or injury and exposure to cold, have been the presumably exciting causes. In a few cases it appears to develop from an occupation neurosis; it developed, for instance, in a tailor who in drawing each stitch had the habit of making a short jerking movement of the head to one side. It occasionally occurs as a symptom of hysteria; but such cases should be carefully separated from those in which there is no hysterical manifestation, as being more susceptible

to treatment and having no tendency to recur when once cured. A torticollis movement may occur as a variety of tic. In one case under our care typical torticollis was the end-result of lethargic encephalitis.

Pathology.—No morbid anatomical changes have been found. On account of the involvement of several muscles, effecting special movements, in this disease (as is well instanced by the over-action of the frontalis in retro collic spasm, for retraction of the head is always normally associated with raising of the eyebrows in the act of looking up), it is probable that torticollis is due to disorder of those centres which direct such associated movements of the affected muscles.

Symptoms.—The onset is usually insidious, but in rare cases may be quite sudden, as in the case of a man aged 40 years, who, when walking along a London street, suddenly turned his head at the sound of an accident which shocked him severely; he was unable to turn his head back without using his hands to do so, and he subsequently developed the most severe torticollis. The initial symptom is always spasm, which may be either tonic or clonic, and frequently both forms of spasm are combined in the same case. In the tonic form, the head is retracted and the face turned to one side, usually the left, and owing to the retraction of the head the face is turned upwards. The shoulder on the side to which the head is inclined is usually raised. In severe cases all the muscles of the upper extremity, the scaleni and the face muscles, may become involved. The spasm, except in the earliest stages, always involved muscles of both sides of the neck. Where the bilateral involvement is general and equal, the rotation of the head does not occur, but it becomes strongly retracted, and the condition is then known as retrocollic spasm. Such retrocollic spasm is always accompanied by marked over-action of the frontales, the skin of the forehead being thrown into transverse wrinkles. In the clonic variety there is jerking movement of the same muscles, usually associated with some degree of tonic spasm. The eyes do not follow the movements of the head in the jerking. The muscle primarily involved is the sterno-mastoid, the action of which is to incline the head forwards and towards the shoulder of the same side, and rotate the face to the opposite side. The next muscle involved is the splenius of the opposite side, which inclines the head backwards and rotates the face towards its own side, its rotatory action thus coinciding with that of the opposite sterno-mastoid. When the splenii of both sides act together, the head is strongly retracted. Next to be affected are the upper part of the trapezii, the trachelo-mastoids and other deep neck muscles, and with further spread of the spasm, any neighbouring muscles of the shoulder and upper extremity may be affected. Sleep causes cessation of the clonic spasm, but not always of the tonic spasm when the case is severe. The spasm is always increased by fatigue and excitement. There is no wasting of the muscles involved, but on the other hand, they may be even hypertrophied if the spasm has existed for long, and their electrical excitability may be increased. The amount of pain associated with the spasm varies greatly. There may be a slight feeling of cramp only, but usually there is a great deal of aching pain, which may radiate down the arm and into the side of the head, and make life unbearable to the patient. More rarely, sharp neuralgic pains are present.

The course of the disease, which has no tendency to shorten life, is chronic

exacerbations and remissions under treatment being common, and recurrence, after temporary cure, frequent.

Diagnosis.—This is usually quite simple. Fixed positions of the head associated with spasm occur in disease of the cervical spine, especially in spinal caries, and are also associated with enlarged lymphatic glands in the neck. The local signs of these conditions, however, are characteristic.

Treatment.—Spasmodic torticollis is a most intractable condition, and in many cases temporary alleviation is all that can be secured. It is usually best to begin treatment by rest in bed, the patient lying supine with the head low and between sandbags or pillows. The regular administration of phenobarbitone, of chlorbutol, or of chloral and bromide may then be tried. Many years ago Bastian claimed good and permanent results from a continuous narcosis lasting 3 weeks and induced by chloral hydrate. Probably a combination of rest as above described, together with massage and resistance exercises is the most useful line of treatment. In some cases the application of a plaster mould, fixing head and shoulders, and worn for one or more months, or a more easily removed and lighter metal splint will give complete respite from muscular spasm while it is worn, and very occasionally permanent respite after removal. In severe and disabling cases this is well worth trial. Surgical measures (tenotomy, excision of the sternomastoid, posterior root section) have all proved disappointing and are not to be recommended.

There is a *congenital form of torticollis* which is of a very different nature. The disease is prenatal and analogous to congenital talipes, the sternomastoid alone is affected, and nearly always that of the right side. Such a muscle is frequently ruptured during birth, and this has given rise to the opinion that the birth injury and subsequent hæmatoma of the muscle were responsible for the torticollis. In many of these cases there is marked facial asymmetry, the face being smaller on the side of the affected sterno-mastoid. This association points strongly to some defect in the nerve centres of the medulla.

Treatment.—This consists in tenotomy of the contracted muscle.

THE TICS

Synonym.—Habit spasm.

Definition.—A group of maladies characterised by the occurrence of either—(1) sudden, rapid, twitch-like, involuntary co-ordinated movements, always of the same nature and in the same region; or of (2) sudden psychical phenomena, imperative ideas and explosive utterances; or (3) of a train of deliberate highly co-ordinated actions produced by an imperative idea. Any combination of these phenomena may occur.

The tics are both ætiologically and clinically related to spasmodic torticollis, into which some of the motor tics graduate. A torticollis movement may occur as a tic, and it may in rare cases pass over into an established torticollis.

The tics may be conveniently divided for clinical purposes into the following groups, between which any combinations may occur:

1. The clinical picture is made up by the occurrence of sudden twitch-like co-ordinated movements, which resemble reflex or defence movements. The

movement is always of the same nature and occurs in the same region, though several different tics may occur in the same patient. The usual region affected is the face, with the pharynx and larynx, the neck and upper extremity. This form occurs chiefly in children, and usually runs a favourable course—Simple Tic.

2. The spasms are more severe and complicated than in simple tic, and imperative ideas and explosive utterances are common and important symptoms. The condition is met with soon after puberty, and more commonly in males—Convulsive Tic.

3. There is no spasm or other motor manifestation, but the psychic tic is expressed by uncontrollable imperative ideas, explosive utterances, arithmomania, etc.—Psychical Tic.

4. Under conditions of mental stress and embarrassment, and in conditions of boredom, the patient performs some highly complicated and co-ordinated act which relieves his nervous tension and fascinates him—Co-ordinated Tic.

The tics are expressions of unrest and of physiological embarrassment in consciousness in a nervous system which is highly sensitive and not too stable. There is always the desire to relieve the embarrassment by the occurrence of the tic, and a feeling of relief when it has occurred, coupled often with disappointment at the failure of its suppression.

While the more simple forms of motor tic from their pattern suggest strongly that they are caused by some peripheral irritation from the conjunctiva in the case of a blinking tic, from the nose in a case of snuffling tic, and from the larynx in a case of laryngeal tic, and that constant irritation from these regions has set up a habit, yet it cannot be too strongly pointed out that no such peripheral irritation precedes the onset of tic, for the irritation and cause come from within the nervous system. Severe peripheral irritation does not cause tic, nor does the correction of errors of refraction, the removal of tonsils and adenoids and of teeth, or circumcision aid in the cure of the malady, though it is only too common to see cases in which these procedures have been inflicted upon the tiqueur one after the other, to the detriment of his tic and of his general health.

1. SIMPLE TIC

Synonym.—Habit spasm.

This is a common disorder of late childhood, the majority of the cases occurring between the fifth and the tenth year. Either sex is prone to the disease. The onset may be preceded by deterioration of health from any cause, and sometimes fright and emotion bring on the tic. Often the malady arises in perfectly healthy children without assignable cause. The children are usually highly strung and intelligent. It is a rare event to see a dull and backward child with a tic.

Symptoms.—The recurring tic appears somewhat suddenly, and may reach its height in a few days. The movements are of the nature of a simple *act*. They occur suddenly and without warning, and are executed rapidly. Usually the movement is of one kind only; but sometimes several movements coexist. The common site of the spasm is the head, face and neck. Blinking, winking, alternate elevation and depression of the eyebrows, side to

side movements of the mouth, tossing the chin in the air, sudden movements of the tongue, palate or larynx, accompanied by an unpleasant fidgeting sound, are of frequent occurrence, while any movement of the head upon the shoulder, torticollic movements, shrugging of the shoulder, and any movements of the arm may be met with. Respiratory movements are often associated with those occurring in the tongue and larynx. Tic affecting the legs is much less common. The movements cease during sleep. Generally a variable time of some length separates the individual movements, but in severe cases these may follow one another almost unceasingly. They are increased by excitement and by observation, and can usually be controlled by the will, but only for a limited time.

Diagnosis.—The movement of tic is so peculiar that it cannot be confused with any other spontaneous, involuntary movement. It is the same movement, repeated with very rapid execution, in the same place. It is short and sharp, like a twitch. In chorea the movements are slow compared with those of tic, and are irregular in nature, in time and in place.

Prognosis.—Most cases of simple tic recover, whether they are treated or not. They recover much more quickly under treatment, and two or three months suffices in most cases to see the end of them. The longer a tic lasts, the more difficult it is to cure. In the rarest cases only does a tic of this nature persist or merge into one of the more severe forms.

Treatment.—A scrutiny of the general health should be made, and any defects attended to. Matters of hygiene, diet, education, exercise and pleasure should be correct and normal. Observation and remarks upon the child's defects, and anything tending to increase self-consciousness should be avoided. The confidence of the child should be gained if possible, and any source of mental worry, or grief, or annoyance should be ascertained and comforted. Restraint and discipline should be kindly taught, and an orderly life followed in which the child is happy, and in which his time is fully and congenially employed. In severe cases only is it necessary to interdict all physical and mental exertion and excitement, and enjoin rest in bed, and these measures should only be employed for a short time. Aspirin in 10-grain doses 3 times a day is a most valuable remedy, hardly to be dispensed with in any case. Tonics are often useful.

2. CONVULSIVE TIC

In this malady, which was first described by Gilles de la Tourette, and which bears his name, the same movements as are met with in simple tic occur; but these are more severe and more widely spread, and they may involve the whole body in spasm at one time. In addition, there are psychic tics, which cause irresistible impulses, among which are explosive utterances, repetition of words, sounds and gestures, and also imperative ideas.

Ætiology.—The stigmata of physical and mental degeneracy are rarely absent, neuropathic and sometimes direct heredity is often present. The malady is said to be more common in males, and is met with more often in France than in England—where it is a rare disease. The symptoms appear usually between the ages of 10 and 15 years, and commonly follow physical or mental shocks or acute illness of any kind.

Symptoms.—The spasmodic movements resemble at first those of simple tic in their nature and rapidity, and favour the same sites ; but they are not restricted to the repetition of the same movement, but successive movements may vary widely in position and extent and sometimes involve the whole musculature of the body. The great variety of facial grimaces, head jerking, grotesque attitudes and ridiculous pantomime which may occur in this affection lead commonly to the belief that the patient is shamming. The tic is not continual as in the simple form. It occurs in the form of bouts in which the same pantomime is reproduced. These are often excited by observation and emotion. They can often be controlled, but with much fatiguing effort on the part of the patient, who becomes so worn out with half successful efforts to control them that he ceases to make the attempt. Between the attacks the patient seems quite normal. The psychic phenomena are the same as in psychical tic, about to be described, and the treatment of the two conditions is identical.

3. PSYCHICAL TIC

In this condition there is no muscular spasm ; but the sudden event takes the form of explosive utterances, imperative ideas and impulsive acts. This condition often occurs as a part of convulsive tic. The exclamatory tic consists of some sound or word or group of either, which is habitually uttered, with complete irrelevancy of time, place or sense. Sometimes the words are of an obscene nature and cause the greatest distress to the patient, who, often of innocent mind, is never safe from putting himself to shame. The utterances may be single, or may be repeated over and over in rapid succession. Echolalia, which is an uncontrollable impulse to repeat sounds heard, or to repeat words which the patient or others have just spoken, may be met with. The great characteristic of the condition is that though the patient desires above all other things to prevent their occurrence he cannot do so by any effort of will. Other symptoms that are commonly met with in this condition are imperative ideas and impulsive acts of all sorts, and in addition insanity of doubt, agoraphobia, acrophobia, mysophobia, etc., and arithmomania. In severe cases grave signs of mental deterioration slowly supervene, judgment and memory fail, will power and attention are lost, and the patient becomes incoherent and insane.

Diagnosis.—Both in the convulsive and psychical tics the diagnosis is placed beyond doubt, both by the nature of the movements and by the peculiarity of the psychic disturbance.

Prognosis.—Permanent recovery has occurred from both these conditions ; but such an event is rare. Most of the cases follow a downward course despite treatment, and many end in suicide or insanity.

Treatment.—General tonic treatment, with change of circumstance and kindly moral and physical discipline, with healthy pursuits and congenial intellectual and physical occupation are the most likely to benefit. When fixed and imperative ideas are present the patient must be guarded, as one of unsound mind.

4. CO-ORDINATED TIC

In this condition complicated co-ordinated movements are habitually repeated without apparent cause or purpose—especially in conditions of

mental stress. It can be best illustrated by the account of an individual case under our care : A brilliant scholar at a public school was noticed to absent himself for no apparent reason, and when sought for, on the occasions on which he could be found, was always discovered in some secluded place rapidly revolving the two index fingers round the inside of a loop of string. He explained to me that this act had always given him relief from mental stress and anxiety, and that he was ashamed of it, and did his best to overcome it, and often succeeded, but that sometimes the desire for relief from stress overcame him. He was treated, and had no return of the tic for some years. He had become head boy of the school and captain of the football team, and on the occasion of the match of the year with another great school with much anxiety and responsibility upon his shoulders, at the moment of the commencement of the match the captain was not to be found. He was recovered from the act of revolving his index fingers round the inside of a loop of string in a secluded place, in time to perform brilliantly and win for his side.

CRAFT PALSY

Synonyms.—Occupation Palsy ; Occupation Cramp.

Definition.—A peculiar malady determined by the habitual use of one set of muscles for the constant repetition of an act of short range, to the exclusion of acts of wider range and acts involving a different set of muscles. The symptoms are: (1) local pain in the muscles concerned ; (2) local spasm of the muscles ; (3) loss of volitional control of the range and nature of the movements ; and (4) weakness of the movements. These symptoms may occur separately or together.

Ætiology.—This disease may be occasioned by any occupation which requires the constant repetition of movements of small range, and which necessitates the holding of the limb rigidly for the fine co-ordination, to the exclusion of free and wide range movements. Consequently it is almost confined to those employments involving finely co-ordinated movements of the fingers, hand and upper limb. As far as is known, a neuropathic inheritance is neither an essential nor a common factor in the production of this disability. Yet at the same time, the fact that young persons are apt to break down while in training, while others follow the occupation of scrivener or telegraphist for years with impunity, it has been suggested that some inherent weakness in the nervous system may underlie the development of the cramp. But, however, it is not necessary to invoke these obscure factors, when faulty motor habits are known to play an important rôle and are adequate to account for the disability.

In the case of writing there are two motor components, pen holding and pen moving. The former is of no importance in the production of writer's cramp, but defects in the latter are of primary importance. The pen may be moved solely by the muscles which hold it, the hand being fixed and the digits alone moving. Less faulty is the habit when writing is done largely by movement at the wrist. Best is the method in which writing is a free movement involving all the limb muscles. Of note in this connection is the fact that subjects of writer's cramp may be able when standing to write on a blackboard. It is those who confine the muscular action most rigidly to

the small hand muscles and the flexors of the index and middle fingers who are most likely to develop cramp. In the use of the Morse key such restriction of activity to small muscles groups is more or less inevitable, but the more modern machines which do not involve this, and have come into greater use, have not this fault and thus telegraphist's cramp is less common now than formerly.

It has been reported that focal lesions in the subthalamie region have resulted in a disability resembling writer's cramp, but the observation is not necessarily relevant to the ordinary example of this condition.

The following is a list of some of the occupations in order of frequency in which this malady occurs: writers with the pen, telegraphists, seamstresses, violinists, machinists, cigar and cigarette rollers, dairy milkers, pianists and typists. It has been said that shorthand writers never suffer from this complaint. In most instances the disability in the use of the hand is confined to the particular movement which first gave rise to it, but in some severe cases other movements may ultimately come to be difficult: *e.g.* shaving, sewing, knitting. It should be borne in mind, however, that when at an early stage of what seems to be writer's or telegraphist's cramp other movements become deranged the presence of some organic nervous affection should be suspected. This point is further dealt with under the heading of diagnosis. The very general use of the typewriting machine, with its free and multitudinous movements required for its manipulation, has made writer's cramp a very rare disease compared with years ago; but it must be borne in mind that when the malady is once installed it is likely to follow the sufferer from one occupation to another. As an example, a telegraphist developed cramp in manipulating the Baudot instrument. He was rested and transferred to lighter duties involving the use of the Morse instrument, where he after a time failed. He was then transferred to counter duties, involving the use of the pen, and became incapacitated with writer's cramp. He was then used to close envelopes, and developed cramp over that act, and ended his service as a messenger. Several of the occupations above mentioned are scheduled in an Act of Parliament as dangerous trades, owing to the liability to cramp, and the employer is bound to compensate for such disability arising in his employ.

Symptoms.—These are of two orders, namely: subjective, consisting of discomfort, pain and the sense of fatigue; and objective, comprising muscular spasm and the abnormalities of movement arising from it and from the effort to avoid both pain and spasm. In some subjects pain, in others spasm predominates.

The onset is gradual, the movements of the pen become inexplicably difficult and tend to be irregular, the strokes extending too high or too low. The subject then finds himself grasping the pen with excessive force, and the correct adjustment of the finger ends becomes hard and apt to fail, the index slipping off the penholder. This he tries to correct by a still firmer grasp. The hand then begins to ache, and feels heavy and tired. With the passage of time all these symptoms increase, and the writing becomes more irregular and the nib is driven more firmly into the paper which it penetrates, the ink spluttering over the sheet. Some tremor may develop in the limb. As the condition grows worse, the cramp appears more and more readily when writing is started, so that even taking the pen in the hand

may evoke cramp. At the same time, other fine and repetitive movements of the hand may be performed with normal ease and facility. The pain which in varying degree accompanies the cramp tends as the affection grows worse to spread from the small hand muscles up the limb until the whole arm and shoulder ache.

TELEGRAPHIST'S CRAMP

There are four instruments commonly used for sending. The Morse instrument is a hard contact key provided with a spring and knob, and the manipulation is performed by making the contact against the spring pressure with the knob held loosely in the palm of the hand. The movements involved are flexion and extension of the wrist only. The arm is supported from the shoulder only. This is a bad cramp-producing instrument, but it has the advantage that it can be manipulated with either hand. If telegraphists are taught to be ambidextrous from the first the incidence of cramp falls to a very low level.

The Baudot instrument consists of a piano-board with five keys, which are manipulated by three fingers of one hand and two fingers of the other, and with the permutations and combinations of these five keys all the signals are made. The movements have to be synchronised to the beats of the commutator. The forearms and wrists rest upon the table. This is the worst cramp-producing instrument that ingenuity could devise, for the movement is of the fingers only, and it is utterly restricted both in space and in time. The Hughes instrument is a piano keyboard with many more keys, and its use is not often productive of cramp. The Gell instrument is a typewriter keyboard, and the movements are free. It is the best instrument in so far as it is not a producer of cramp. Two points of interest stand out, especially in connection with cramp in telegraphy. The one is that, notwithstanding the discomfort, spasm and obvious disability, the subject is rarely reported for faulty sending, so great is his aptitude for dodging his disability. The second point is that so many subjects will work for years, and often to the end of their service, with very obvious cramp, which never proceeds to incapacity. On the whole, the incidence of telegraphist's cramp is now small.

Diagnosis.—From what has been said of the character of the symptoms in these forms of cramp, of the mode of their production by a particular movement-complex, and of their occurrence in the absence of signs of organic nervous disease, it seems reasonable to state that errors of diagnosis should not occur.

Nevertheless, errors are not infrequent and consist in the diagnosing of writer's or of telegraphist's cramp when in fact some organic affection is present. Paralysis agitans, with little or no tremor, and post-encephalitic Parkinsonism provide fruitful sources of error. In the clinical picture thus presented, the initial symptoms may involve the right arm and hand, and at first consist in a difficulty in the normally rapid and free performance of fine movements. Not unnaturally the handwriting may be affected early. It becomes slow in performance, spidery and progressively smaller, and the effort to continue writing may be irksome and even painful. The total clinical picture in such a case is made up of such small deviations from the normal that the inexperienced or careless observer may miss them and may note no more than the patient himself has noted ; namely, that it has become

difficult and uncomfortable to write. Amongst other organic conditions which may be encountered under the erroneous diagnosis of writer's or telegraphist's cramp may be included cervical rib, any organic nervous affection which impairs fine hand movements, arthritis, and painful affections of muscles. The general principle which underlies accuracy of diagnosis here as elsewhere is careful and systematic clinical examination.

Course and Prognosis.—In a young subject, who shows signs of the malady during training or soon thereafter, the outlook is hopeless with regard to continuance of the occupation, and the progress is from bad to worse. In older subjects the course varies greatly. Some cases recover completely and permanently, even though they continue with the occupation. In others—and this class is much larger than is usually supposed—the condition of cramp becomes stationary, and persists though not in disabling fashion. In a third and numerous group it progresses to incapacity, and tends to reappear with every change of occupation. In a few cases the patients become incapacitated for all the finer movements of both hands. The prognosis is usually serious; but a correct forecast can only be made from the history and progress of each individual case.

Treatment.—The responsibility and costliness which the Compensation Act entails upon employers are slowly enough but surely leading to the abandonment of those instruments, the manipulation of which may produce cramp. Good teaching of unconstrained methods of manipulation and encouragement of ambidexterity in all the occupations concerned are important prophylactic measures. Long hours and the speeding-up of work should be avoided. After long absence from work, the work should be gradually resumed and not recommenced at full pressure. When the malady appears, rest and change of work afterwards are absolutely essential. Long-continued rest, be it remembered, cuts both ways for, as has been pointed out above, resumption after long rest is actually a cause of cramp, for long unemployment decreases the stability and the aptitude of the mechanism. General hygienic and tonic treatment are important. It is doubtful whether local treatment, in the way of massage, electricity, etc., can do any good, except to satisfy the patient. Sporting exercise of any and every kind is most useful. The Post Office authorities adopt the very admirable plan of re-training cramp subjects by daily practice with the instruments for a few minutes, the time of practice being gradually lengthened as capacity increases.

CRAFT ATROPHIES

Under this title are described a medley of conditions in which local atrophy of muscles, pain, numbness and sensory loss occur in connection with regions which are habitually over-exerted. These conditions have been met with in platers, filers, file-makers, locksmiths, rowers, glassworkers, etc., and seem to be really examples of local traumatic fibrositis, involving the nerves, and produced in some cases by the continued pressure of the tools.

Many of the cases recover with rest and treatment appropriate for a local interstitial neuritis.

JAMES COLLIER.

W. J. ADIE.

Revised by F. M. R. WALSHIE.

COMPRESSION OF THE SPINAL CORD

In compression the lumen of the spinal canal is reduced in a small part of its vertical extent, and the spinal cord is injured at this point, either directly by pressure, or indirectly by interference with its vascular supply. With the exception of acute inflammation of the membranes, all the extramedullary lesions of the spinal cord come under this heading. The characteristic clinical feature of compression is the combination of two sets of phenomena: local or root symptoms in those regions supplied by the roots arising from the cord at the level of the lesion, and remote or cord symptoms due to interruption of the conducting paths in the white matter. It is convenient to divide this subject into two parts—slow compression and compression of rapid onset.

1. SLOW COMPRESSION

The commonest causes are tuberculous spinal caries, vertebral tumours, meningeal tumours and cysts; rarer causes are aneurysm, gumma, leukaemia, Hodgkin's disease, Paget's disease, syphilitic caries, spondylitis deformans, and other chronic inflammations of the bones and joints of the spine.

Ætiology and Pathology.—1. *Tuberculous spinal caries (Pott's disease).*—Spinal caries is the most frequent cause of slow compression. It occurs most often in children, but is common in adults, and may begin late in life. Signs of injury to the cord develop in about 1 case in 20, and are usually preceded by obvious deformity of the spine; but in many cases they appear before disease of the bone is suspected. Rarely paralysis comes on for the first time in an adult who has had a curvature since childhood.

The cord may be damaged by direct pressure of displaced bone, or by an abscess beneath the periosteum of the diseased vertebræ; but in almost all cases the injury is indirect, and results from œdema of the cord, arising from interference with its blood supply by tuberculous granulation tissue, which forms on the outer surface of the dura mater and fills the epidural space (pachymeningitis externa). The functions of the cord may be temporarily deranged for long periods by this œdema, without permanent damage to the nervous tissues; hence, when the disease is cured, the œdema subsides and the cord recovers. In cases of greater severity necrosis of the nervous structures follow thrombosis of the vessels, or prolonged pressure causes atrophy of nerve roots, and complete recovery is impossible.

2. *Tumours of the vertebral column.*—Vertebral tumours are about twice as common as all the other forms of extramedullary tumours together, and almost all of them are malignant. Carcinoma is always secondary, and is a frequent and distressing complication of cancer elsewhere. A very small primary carcinoma, e.g. of the breast, thyroid or prostate, may produce extensive disease of the vertebræ, and signs of compression may appear before the existence of the primary growth is suspected. On the other hand, they may appear several years after complete removal of the primary growth, and may be the first evidence of a recurrence. Sarcoma, the commonest form of primary growth, begins in the bone or periosteum of the bodies or laminae, often in several at once, or simultaneously at different levels. Secondary

sarcoma arises by metastasis from sarcoma elsewhere, or by direct extension from a growth in neighbouring soft parts, *e.g.* of tumours in the mediastinal and retro-peritoneal spaces.

The growth of vertebral tumours is usually rapid, and extensive portions of the spinal column may be completely destroyed. The cord is compressed by the growth itself, by displaced bone, or by a process of the growth which invades the spinal canal through an intervertebral foramen. As a rule, the dura mater sets bounds to its inward extension. Benign tumours of the spine are rare. They usually grow forwards, but occasionally an osteoma, a chondroma, or an exostosis produces signs of compression.

3. *Meningeal tumours.*—These are divided into two groups—intradural and extradural. The first are twice as common as the latter, and more than half of them are simple, encapsuled, and easily removable. Myxoma, fibroma, sarcoma, endothelioma, and psammoma are common. Other forms are rare. In most cases they lie posterior or postero-lateral to the cord, and are seen when the cord is exposed by laminectomy; but in a few cases they lie in front and may escape detection.

Sarcoma is the commonest extradural tumour. It is sometimes encapsuled, but more often it is a diffuse growth difficult to remove.

These tumours usually grow very slowly, and several years may elapse between the onset of the first symptom and the time when an accurate diagnosis can be made. They do not invade the substance of the cord, nor penetrate the dura, nor give rise to metastases.

4. *Cysts.*—Cysts, parasitic and non-parasitic, may compress the cord and produce symptoms indistinguishable from those of solid tumours. In some countries hydatid cysts form a high proportion of all spinal tumours. They may invade the spinal canal from adjacent soft parts or from the vertebræ, or arise primarily in the membranes. They are often multiple, and are nearly always extradural. Cysticercus cysts, which are very rare, are usually single and intradural. Non-parasitic cysts are collections of fluid contained within slightly thickened adherent membranes. They are among the most frequent of spinal tumours. They are most likely a result of circumscribed inflammation of the pia-arachnoid, and have been known to follow an injury; but their ætiology is obscure. The cyst is often opened inadvertently during operation, and the only evidence of its former presence is flattening of the cord and atrophy of the nerve roots at the point where a solid tumour was expected. Sometimes the position of the cyst can be inferred at operation, from the absence of normal pulsation below it. On puncturing the membranes in this position fluid escapes under pressure, and the pulsations reappear. The name *meningitis serosa circumscripta* is applied to this condition.

5. *Aneurysm of the aorta* is a rare but well-known cause of spinal compression. The dorsal region is most often affected, three or four vertebral bodies being slowly eroded until the dura mater is exposed. Rupture into the spinal canal has been observed. Berry aneurysms upon the surface vessels of the cord occurring in connection with occult coarctation of the aorta have been many times recorded. The pathognomonic sign of this cause of compression paraplegia is the high arterial blood pressure in the arm as contrasted with the low pressure in the leg.

6. *Syphilitic caries.*—Gummata and caseous masses of syphilitic origin

in the bones of the spine may cause a condition not unlike that of tuberculous caries. It is very rare, and usually occurs in the cervical region where the cord may be compressed.

EXTRADURAL COMPRESSION

Symptoms.—*Local or root symptoms.*—Pain in parts supplied by the sensory roots arising from the cord at the level of the lesion is often the first symptom. It may be a dull ache, a feeling of constriction, a sharp cutting pain, or pain so severe as to be almost unbearable. It is often brought on or greatly increased by movement of the spine or by coughing. The skin in the painful area is sometimes hypersensitive at first, but very soon its sensibility is diminished, while the pains persist (*anæsthesia dolorosa*). The nerve trunks are not tender as in ordinary neuralgia. Severe pains are rarely absent in cases of vertebral tumour. In spinal caries they are usually absent or slight. Injury to the motor cells or anterior roots leads to weakness, wasting and loss of tone in the corresponding muscles. In some cases root symptoms are absent throughout the course of the disease, and the first effects of compression are referable to interruption of the conducting paths in the cord.

Remote or cord symptoms.—Although all the tracts are submitted to the same degree of compression, their functions are not impaired at the same time. While variations are common, the symptoms usually arise in the following order: first weakness and spasticity in the lower limbs, then impairment of sensation, position and passive movement, temperature, pain and touch being affected in this order. Defective sphincter control often precedes and sometimes follows sensory loss.

Motor symptoms.—Interruption of the pyramidal tracts produces spastic paraplegia in parts below the lesion. The clinical features are—(1) diminution of voluntary power; (2) alterations in the amount and distribution of muscle tone and in the attitude of the limbs; (3) changes in the tendon and skin reflexes; (4) the occurrence of certain involuntary and reflex movements.

The phenomena of spastic paraplegia have been analysed by Walshe as follows:—It is essential to remember that the muscles of the lower limb are divided into two distinct groups—the flexors and the extensors—and that the muscles which dorsiflex the foot and toes are physiologically flexors, while the corresponding plantar flexors are extensors. In all that follows these important muscles will be grouped according to this nomenclature.

1. Loss of voluntary power varies from slight weakness of one group of muscles to complete paralysis of both limbs, and depends on the degree of damage to the pyramidal tracts. It usually begins in the distal segments of the limb, and is greater in the flexors than in the extensors. Dorsiflexion is the earliest and remains the most severely impaired movement.

2. The tone in all the muscles increases early, and is greatest in the extensors. Hence an early symptom is generally stiffness of the limbs, especially a difficulty in flexing them. If the limbs are handled passively, the resistance to flexion is found to be greater than to extension. As power diminishes spasticity increases, until at length the limbs are held constantly in an attitude of complete extension. This combination of weakness and spasticity with extended lower limbs is known as “paraplegia in extension.”

3. Exaggeration of the tendon reflexes is a constant early sign. The abdominal reflexes below the level of the lesion and the cremasteric reflexes are lost early. The normal plantar reflex is also lost, and is replaced by a different kind of reflex—Babinski's sign, the "extensor" plantar response.

As the damage to the cord increases, and when certain extra-pyramidal motor tracts are affected, the extensor muscles gradually lose their tone, for which connections with the brain-stem through these extra-pyramidal tracts are essential, while the tone in the flexor muscles, which depends on a reflex arc which is purely spinal, is retained. The result is that the knee- and ankle-jerks, which indicate tone in extensor muscles, are lost while the reflexes from flexor muscles (hamstring-jerks) persist. At the same time, in some cases, the limbs are gradually drawn up by the unopposed action of the flexors. This combination of weakness and spasticity with flexed lower limbs is known as "paraplegia in flexion." At first the flexed position is occasional—flexor spasms—later it becomes constant, but is still due entirely to excess of tone in the flexors. Finally, contractures occur in the muscles, and the deformity becomes permanent. In many cases of compression the stage of paraplegia in extension gradually merges into one of complete flaccidity of all the muscles, without the occurrence of paraplegia in flexion, and all the tendon reflexes are lost.

4. While the limbs are still rigid in extension, the commonest involuntary movement is a spontaneous clonus of the extensor muscles, in which the whole limb trembles as it does when ankle clonus is elicited in a case with marked spasticity. In the later stages, where the extensor muscles are beginning to lose their tone, a new kind of movement appears, in which the limbs are drawn up suddenly from time to time by an involuntary contraction of the flexor muscles—flexor spasms. Further, by appropriate stimulation may reflex movements can be produced in the paralysed limbs. The most important of these is the "flexor reflex of the lower limb." It is elicited most easily by stimulating the outer border of the sole by firm pressure or a pin-prick, and in its complete form consists in flexion of the hip and knee, dorsiflexion of the foot, and an upward movement—so-called extension but physiological flexion—of the great toe. When the damage to the motor tracts is slight, when the limbs are rigid in extension and the movement of flexion is prevented by the hypertonus of the extensors, or when almost all reflex activity has disappeared, the reflex appears in its minimal form. A part of this minimal response is an "extension" of the great toe. The normal plantar response is obtained from the sole alone. The pathological reflex, of which the "extensor" response is a part, may be obtained not only from the sole, but when well developed by stimulating the skin and deeper structures on any part of the lower limb. In the light of this the nature of many reflexes which have been described as isolated signs of pyramidal tract disease, *e.g.* the "extensor" plantar response, Oppenheim's and Gordon's signs, and many well others, becomes clear. In all of them a stimulus is applied to some part of the lower limb, and the response is a flexion reflex, whose most obvious component is "extension" of the great toe. It is unfortunate that the term "extensor response" is commonly used to describe a movement which is physiologically one of flexion.

Sensory symptoms.—Sensory loss may appear first in the area supplied by the roots arising from the cord at the level of the lesion, or in parts below.

As a rule motor disturbance is severe before any remote sensory loss is found. In some cases, especially of spinal caries, the limbs are completely paralysed before sensation is affected. The reverse condition, severe sensory loss with slight motor disturbance, does not occur in compression. Remote sensory loss appears first in one of two positions. In most cases the soles first become less sensitive, then the legs, and later the thighs. In a smaller number the loss appears first over the lower sacral segments, and extends upwards in segmental progression. When both sides are equally compressed all forms of sensation may suffer equally; but in most cases position and passive movement, temperature, pain and touch are impaired in this order.

If one side of the cord is more affected than the other the signs are those of a modified Brown-Séquard syndrome with the superficial sensory loss greatest in the limb in which most power is retained, and loss of position and passive movement greatest in the weaker limb. Ultimately, whatever the order of loss at the beginning, as the compression increases, sensation of all kinds is diminished or lost in all parts below the lesion. In exceptional cases the skin in the distribution of the lowest sacral segments retains its sensibility when the loss in all other parts below the lesion is severe. Such a distribution of sensory loss suggests a lesion damaging the more mesial fibres of the sensory path. Occasionally the onset of symptoms in extramedullary compression is rapid, and severe paraplegia develops in a few days. This is seen most often in cases of sarcoma of the mediastinal or retro-peritoneal spaces where the growth, which has infiltrated one or more vertebral bodies, surrounds the dura and produces œdema of the cord. In cases of rapid onset the limbs are flaccid from the beginning, and the tendon jerks are diminished or lost.

INTRADURAL COMPRESSION

When the pressure is equal on both sides of the cord, the symptoms are the same as in extradural compression. If one side is affected before the other, as by tumours growing from a nerve root, the symptoms are at first unilateral, and in many cases spasticity and weakness are confined for a long time to the lower limb on the same side as the tumour. Occasionally alterations in subjective sensation (*paræsthesiæ*) in the limb of the opposite side precede motor symptoms by a considerable interval, and form the grounds for the patient's first complaint.

At a later stage the following characteristic syndrome appears:

1. A band of sensory loss, on the side of the tumour in an area corresponding to the distribution of the sensory roots arising from the cord at the level of the lesion, often with root pains in the same area.
2. Weakness and spasticity confined to or greatest in the limb on the same side (pressure on the pyramidal tract).
3. Diminished sensibility to temperature, pain, and touch on the opposite side (pressure on the crossed sensory tracts).
4. Impairment of the sense of position and passive movement in the weaker limb (pressure on uncrossed sensory tracts in the posterior columns).

As the compression increases both limbs become weak, spastic, and insensitive, and the symptoms progress as in extradural compression. Examination of the cerebro-spinal fluid often reveals a condition which is practically

pathognomonic of compression--*the loculation syndrome* of Froin. This consists, in its complete form, in an increase in the amount of albumin with absence of or slight increase in the number of cells, and a yellow colour (xanthochromia) in the fluid. The normal amount of albumin is about 0.025 per cent. In compression it is often increased a hundredfold or more, and readings above 1 per cent. are very common. A low cell count and 0.1 per cent. of albumin are very strong evidences of compression, and as the amount increases the diagnosis becomes more certain. Xanthochromia is common; but it occurs in other conditions, and its value as a sign is slight. These changes are found in the fluid only below the site of compression.

Diagnosis.—A complete diagnosis establishes: (1) the existence of compression; (2) its situation; (3) its pathological nature.

1. When signs of injury to the cord or nerve roots are found associated with disease of the spine at a compatible level the diagnosis is obvious. What root pains are the only symptom the diagnosis is difficult. They are often falsely interpreted as referred pains, or as indications of disease in the painful part itself. Thus angina pectoris, gall-stones, pleurisy, renal colic, hip-joint disease and other painful conditions have been diagnosed, and unnecessary operations have been performed. Pain of root distribution should always arouse suspicion, and provoke a careful examination of the spine and of the nervous system.

If the signs are those of spastic paraplegia, spinal syphilis, disseminated sclerosis, syringomyelia and amyotrophic lateral sclerosis must be excluded. *In many cases this can be done by examination of the cerebro-spinal fluid.* Patients with removable spinal tumours are still allowed to develop incurable paralysis because this examination is not made. Such an omission in a case of paraplegia of doubtful origin amounts to neglect. The loculation syndrome in the fluid is almost pathognomonic. It is never absent in chronic cases, and is never found in any of the focal or system diseases, for which compression might be mistaken.

Spinal syphilis is detected by examination of the blood and cerebro-spinal fluid. Valuable time may be lost, however, or an irretrievable error made, when the blood of a patient with a removable tumour happens to react positively to Wassermann's test. In a series of cases operated upon by Sargent at the National Hospital, the reaction was positive in four patients from whom a non-syphilitic tumour was removed.

Disseminated sclerosis may present the picture of progressive spastic paraplegia with considerable sensory loss, and confusion between it and compression by slowly growing tumours is common, each disease being mistaken for the other. The more serious error is to mistake the curable for the incurable disease. It is advisable to feel dissatisfied with the diagnosis of disseminated sclerosis, so long as the symptoms are purely spinal, and to re-examine the patient at intervals in the hope of finding evidence of a simple tumour. The transient nature of the early symptoms and the presence of signs of cranial nerve troubles serve to distinguish disseminated sclerosis in most cases. Slight nystagmus must not be accepted as decisive evidence against tumour, as it may be present in compression, especially of the cervical region.

Amyotrophic lateral sclerosis simulates compression in the cervical region, in that wasting in the muscles of the hands and arms is associated with

signs of spastic paraplegia in the lower limbs; but is distinguished by exaggeration of the tendon reflexes in the wasted arms, by fibrillary tremors in the muscles, and by the absence of objective sensory disturbances.

Syringomyelia is easily recognised by the characteristic sensory changes. Dissociation of sensation to the degree which is common in this disease is never seen in extra-medullary lesions.

2. SEGMENTAL DIAGNOSIS.—As the motor sensory and reflex functions of each segment of the cord are known, the level of the lesion can be deduced by noting the highest point at which these functions are impaired.

Motor localisation.—Each segment of the cord contains nuclei for several muscles, and most muscles receive nerve fibres from more than one root; but as each muscle seems to have one main root of supply, the weakness, wasting and loss of tone vary in distribution with the segment affected. The muscles which suffer most when the corresponding segment is damaged are named hereunder:

*C*₄. Supraspinatus, infraspinatus. *C*₅. Biceps, deltoid, brachialis anticus, supinator longus. *C*₆. Pronators of forearm. *C*₇. Triceps, extensors of wrist and fingers. *C*₈. Flexors of wrist and fingers. *D*₁. Small muscles of the hand. *D*₂₋₁₀. Intercostal muscles. *D*₁₁₋₁₂. Muscles of abdominal wall. *D*₁₁-*L*₄. Ilio-psoas (mainly *L*₅). *L*₅. Adductors of thigh. *L*₄. Abductors of thigh, extensors of knee. *L*₅. Hamstrings. *S*₁. Glutei—calf muscles. *S*₁. Anterior tibial muscles—peronei—small muscles of foot.

Wasting of the muscles in an intercostal space is a valuable guide, as the muscles of each space are innervated from one segment alone. If the lesion is at the level of the ninth dorsal segment the rectus abdominis is paralysed below a point about an inch above the umbilicus. In such a case, when an attempt is made to raise the head against the resistance of a hand placed on the forehead when in the supine position, the upper part contracts and the umbilicus is drawn upwards (excursion of the umbilicus). If the lesion is at the twelfth dorsal segment the entire rectus contracts, but the iliac regions bulge, owing to paralysis of the lower part of the oblique muscles.

Localisation by changes in the reflexes.—Above the lesion the reflexes are normal, at its level they are diminished or lost, below it the skin reflexes are diminished or lost, and the tendon reflexes are exaggerated. The segments on which important reflexes depend are:

*C*₅. Biceps- and supinator-jerks. *C*₆. Pronator-jerks. *C*₇. Triceps-jerks. *D*₇₋₁₂. Abdominal reflexes. *L*₂. Cremaster reflexes. *L*₅. Knee-jerks. *S*₁. Ankle-jerks. *S*₁. Plantar reflexes.

In lesions involving the fifth cervical segment of the cord, such as may be found in syringomyelia and in injuries associated with dislocation of the cervical spine, Babinski has recorded that the supinator jerk may be abolished and replaced by finger flexion when the lower end of the radius is tapped. This is known as "inversion of the radial reflex," and is a useful localising sign in lesions of the segment in question.

Sensory localisation.—The sensory areas supplied by each segment of the cord are shown in the diagram on the opposite page. Root pains in the distribution of one or more of these areas form a sure guide to the affected segment. When they are absent, the level of the lesion is determined by ascertaining the highest point at which sensation is impaired. Very often when

the two sides of the cord are compressed unequally the anæsthesia is confined to one side, or extends higher on one side than on the other. In these cases, when the segmental diagnosis is made from the sensory signs alone, the lesion, a tumour for example, is found several segments higher than the point indicated by the signs. The uppermost limit of sensory loss in these circumstances is a result of interruption of the sensory paths in the cord, and the discrepancy in the signs is due to the oblique course taken by the sensory fibres in crossing the cord. In the mid-dorsal region the decussation for pain and temperature is complete one segment above the point of entry of the root conveying these impressions to the cord, that for touch in two segments. At higher levels, crossing takes place more slowly, until in the upper cervical region impulses which enter together in one root ascend on the same side of the cord for five or six segments before all of them reach the opposite side. At all levels pain crosses soonest, then heat, then heat, and touch slowest of all.

It follows that in unilateral lesions the upper level of the anæsthesia on the opposite side of the body, caused by injury to sensory paths in the cord, is below the segmental level of the injury. Also that the level is higher for one form than another. The fibres which cross slowly escape by ascending beyond the lesion on the uninjured side before they cross, while those which cross quickly are caught after crossing. Hence the level of sensory loss is highest for pain and lowest for touch, with temperature intermediate. Occasionally the tumour is found below the level predicted. In these cases the functions of the segments above the lesion are impaired by œdema.

Surface anatomy.—If the cord is to be exposed at the level of the affected segments their relation to the spinous processes of the vertebræ must be known. This is obtained as follows: In the cervical region to the number of the spine add 1—the fifth cervical spine lies over the sixth cervical segment; to the number of the upper five dorsal spines add 2—the fourth dorsal spine lies over the sixth dorsal segment; down to the tenth dorsal spine add 3—the tenth dorsal spine covers the first lumbar segment. The eleventh dorsal spine corresponds to the third lumbar segment, and the twelfth to the first sacral. The cord terminates just above the level of the first lumbar spine.

Intrathecal injection of lipiodol.—The upper level of a lesion which narrows or obliterates the lumen of the spinal canal, or simply blocks the spinal subarachnoid space, can be ascertained by injecting lipiodol through the occipito-atlantoid ligament into the subarachnoid space, the lipiodol falls rapidly to the point of constriction, where it is arrested and can be seen clearly by X-rays. If there is no constriction it falls to the bottom of the thecal space, where it remains indefinitely and does no harm. This procedure is of great practical value when the existence of a compressive lesion or its exact site is in doubt.

3. **DIAGNOSIS OF THE CAUSE OF COMPRESSION.**—When spastic paraplegia develops in a patient who is known to suffer from *spinal caries*, the cause is obvious; but when it precedes the appearance of signs of bone disease the diagnosis is difficult. In all cases of compression the spine must be examined repeatedly for deformity, tenderness and limitation of movement. If tenderness is found constantly in the same place, and the nervous symptoms are compatible with disease of the underlying segments, disease of the bones is

almost certain. In young persons disease of the spine is usually caries, and in adults caries is also the commonest cause; but tumours of the spine and aneurysm must be excluded. Severe root pains are rare in caries but are the rule in vertebral new-growths. An aneurysm would present other signs.

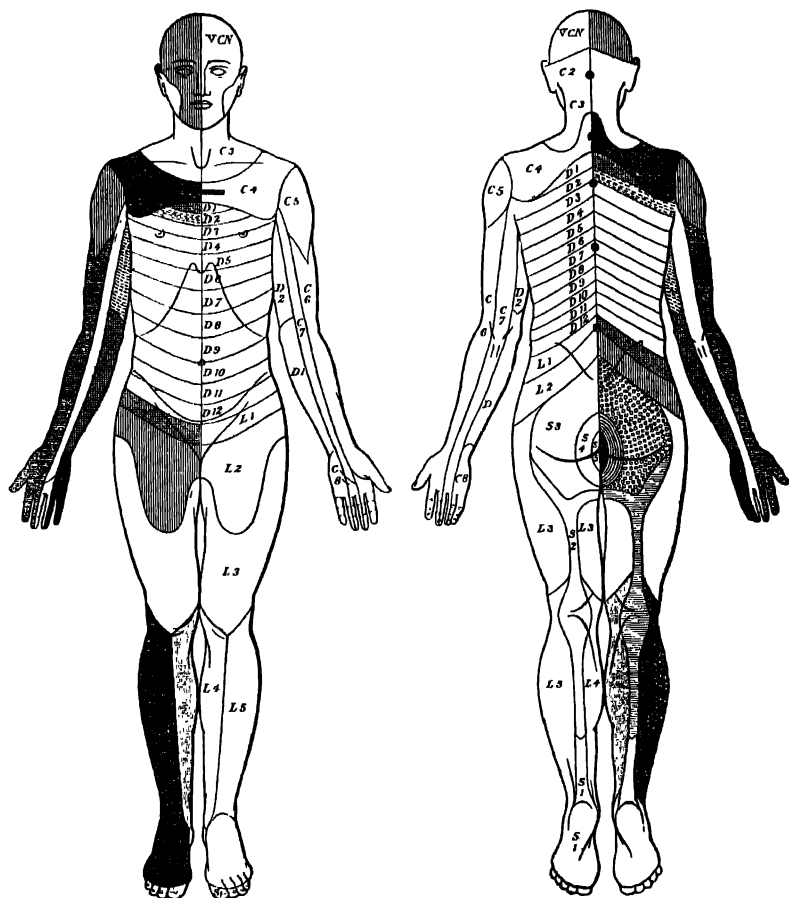


FIG. 99.—Diagram of cutaneous areas of posterior nerve roots (after Collier and Purves Stewart).

An X-ray picture will usually demonstrate the presence and nature of the bone disease.

Vertebral tumours.—When root pains occur in a patient with malignant disease, or from whom a malignant growth has been removed, the diagnosis is clear. Mistakes are easily made when pains are the first symptom, as their

root origin is not recognised. Diminished sensibility in the painful area indicates the nature of the pain, and this directs attention to the spine, where tenderness or deformity is discovered. As most vertebral tumours are secondary, the next step is to examine the parts where carcinoma is common, remembering that a small primary growth, *e.g.* in the breast, thyroid or prostate, may give rise to widespread metastases in the bones. In the absence of a history or signs of new-growth in other parts, the diagnosis is founded on the combination of local tenderness or deformity and rigidity of the spine with root or cord symptoms. The severity of the root pains, and their great aggravation by movement, are characteristic.

Meningeal tumours.—Severe pain of root distribution is present in many cases, and when this is followed after some time by spastic paralysis of slow onset and steady uninterrupted progress, affecting first one leg and then the other, the combination and especially the course of the symptoms are almost pathognomonic. In caries the root pains are rarely severe, signs of bone disease are rarely absent, the paralysis is usually bilateral from the beginning, and is severe before any sensory loss is found. The distinction from vertebral new-growth may be impossible when the latter occurs without bone symptoms or X-ray signs. Practically the diagnosis between meningeal and intramedullary tumours is impossible. If the signs and symptoms in any case are those of compression, and an approximate indication of the level of the lesion can be given to guide the surgeon, then, if other obvious causes such as caries, aneurysm and vertebral tumour have been excluded, an operation must be performed. Until the lesion can be seen the diagnosis is in doubt.

Course and Prognosis.—*Spinal caries.*—The course of the bone disease does not always run parallel with the paralysis, and either may alter in severity independently; but if the caries undergoes cure the paralysis usually diminishes. Considering the severity of the paralysis, the prognosis is favourable and astonishing recoveries occur. The outlook is best in young people with disease in the dorsal region. Many recover completely, but more often, especially in adults, recovery, though considerable, is partial. So long as the lower limbs remain spastic in the extended position with exaggerated tendon reflexes the prognosis for complete recovery of power is good; but if the limbs become flexed, if they become flaccid, if the knee- and ankle-jerks are lost, if sensory loss is severe, or if there is wasting in the limbs following damage to lower motor neurones, the outlook is very bad. Many patients live for years with severe paralysis; but life is constantly endangered by sepsis from bed-sores, ascending infections of the urinary tract, chest complications, and tuberculous disease in other parts.

Vertebral tumours.—When sarcoma or carcinoma spreads to the vertebræ from surrounding parts the duration of the disease is measured in weeks or months, and death is due to the primary condition. In primary sarcoma, and in some cases of carcinoma of the vertebræ, life may be prolonged for a year or two, and death is due rather to complications of the cord disease—bedsores, cystitis, etc.

The course of *tumours of the meninges* is often extremely slow. Root symptoms may precede paralysis by months or even years, and the weakness may increase gradually for several years before walking becomes impossible. Malignant growths are fatal, and simple growths equally so, if not removed. Most patients with simple tumours come to operation during the second

year after the onset of the first symptom. The mortality after operation for the removal of simple tumours is very low in skilled hands. The prognosis for recovery of power depends in part on the duration of the weakness in the lower limbs. Complete recovery may be expected if it has not lasted more than a year, and if sphincter control has not been lost. When the paralysis is of longer duration recovery, though gratifying, is rarely complete. Nevertheless, full return of power has been seen after three years of severe paralysis.

Treatment.—*Spinal caries.*—This is to be directed to curing the bone disease in the hope that cure of the paralysis will follow. Complete rest on the back and fixation of the spine for many months is the routine treatment. The general condition of the patient is to be improved by fresh air, a liberal diet and cod-liver oil, iron and arsenic, and great care is to be taken to prevent bedsores, cystitis and deformities of the limbs. For adults, especially if they are bread-winners, a more rapid cure is desirable. This is provided by surgery in Albee's operation, or a modification thereof, in which a bone graft from the tibia is wired into a cleft made in the spines of the affected region. In this way fixation is effected, further deformity is prevented, and the time lost is reduced from a year or more to a few months. Adults sometimes recover after a short rest in bed, if a suitable jacket is worn to immobilise the spine.

Operation must be considered—(1) when a sudden increase of deformity or severe root pains and great increase of paralysis come on together, and suggest pressure by displaced bone; (2) when an abscess forms; (3) when paralysis persists long after the bone disease is cured, or when in an adult there is no improvement after 6 months' rest; (4) when life is endangered by respiratory paralysis.

Vertebral tumours.—In slowly growing primary growths of the vertebræ laminectomy is indicated to relieve pressure, or to prevent pain by cutting sensory roots or dividing the antero-lateral columns of the cord. The operation is merely palliative, but is often followed by considerable temporary recovery.

Meningeal tumours.—If the symptoms suggest an intraspinal tumour, and a segmental diagnosis has been made, an exploratory laminectomy should be done in the hope of finding a removable tumour.

2. COMPRESSION OF RAPID ONSET

Ætiology.—The commonest causes of rapid compression are fracture or dislocation of the spine.

Dislocations occur most often between the atlas and axis, or between the fifth and sixth cervical vertebræ. They are sometimes incomplete, and thus may cause compression of slow onset. More often they are complete, and the cord is compressed between the laminae of the dislocated vertebra, which is displaced forwards, and the body of the underlying vertebra. Fractures are commonest in the lower dorsal and upper lumbar regions, and follow most often a fall from a height on to the feet or buttocks. They also occur apparently spontaneously in spinal caries and in vertebral tumours. A blow on the back may fracture the vertebral arches, and cause compression of the cord. The degree of damage to the cord varies greatly.

Symptoms.—Sudden severe compression produces a complete interruption of conduction through the damaged segments; that is, a physiological transection of the cord. There is total flaccid paralysis, total sensory loss, abolition of tendon reflexes, and retention of urine. For a few days there may be no plantar response, but a Babinski response then makes its appearance. Bed-sores often develop with great rapidity, and many patients succumb within a few weeks. But in those that survive, retention of urine gives place to overflow incontinence, the tendon jerks may return, and the condition described above (p. 1718) as “paraplegia-in-flexion” may ensue. In this case, any stimulus applied below the level of the lesion produces violent flexion spasms in the legs, contraction of the abdominal wall, and the expulsion from the bladder of some—but not of all—its contents. Head and Riddoch have applied the term “mass reflex” to this phenomena.

Dislocations between the fifth and sixth cervical vertebræ may produce severe damage to the cord as described above, with the addition that the arms are paralysed also, the sympathetic pathways in the cord are interrupted, and there is narrowing of the palpebral fissures, and sometimes priapism. Less well-recognized are the various clinical pictures of minor injury to the cord as the result of vertebral injury. These include slight weakness, with ensuing wasting of the hands and forearms, slight spastic paresis of the legs, with the corresponding changes in the reflexes, narrowing of one or both palpebral fissures, and inversion of the radial reflex on one or both sides. There may be no sensory loss after the first two or three weeks. The slow progress of the wasting in the arms for some time after the injury may lead to a diagnosis of amyotrophic lateral sclerosis, but care in examination and history-taking should prevent this error.

Prognosis.—The prognosis is always extremely grave in severe cases, death resulting in high cervical lesions from paralysis of all the muscles of respiration, in lesions at lower levels from sepsis following bedsores, or from infection of the urinary tract.

Treatment.—When the signs are those of complete division of the cord, treatment by operation is directly contra-indicated. If some voluntary power is retained, or if sensory loss is not absolute, operation may be considered, especially if the compression is caused by fracture of the vertebral arches. The cord should be exposed in every case where the level of the fracture points to injury of the cauda equina. These roots being peripheral nerves have a chance of regenerating, and this may be enhanced by freeing them from compression by displaced fragments of bone. With skilled nursing, patients with complete division of the cord may survive and live for many years.

GENERAL MANAGEMENT OF PARAPLEGIA.—In all cases of severe paraplegia from spinal cord lesion where sensory and sphincter functions are also impaired or lost, whatever the nature of the lesion, there are certain general principles of treatment. The patient should be nursed on a fracture bed with an air, water, or rubber mattress. The back should be attended to four hourly, first washed with soap and water, then carefully dried, rubbed with surgical spirit or eau-de-Cologne, and powdered. These measures harden the skin and make it less likely to break down under the constant pressure of the body weight. The patient's position should be changed from time to time to prevent the development of sacral bedsores. As far as possible

care should be taken to prevent the skin from becoming wet and sodden when there is incontinence of urine, and the toilet of the anus after defæcation should be careful and thorough. There are various remedies for the sacral or trochanter bed sore when it develops. Separation may be hastened by wet dressings of eusol, sometimes by fomentations—though the latter should be used with caution. The ulcer is packed with eusol gauze, or with an ointment of zinc oxide and castor oil. When it is clean and healing begins, it may sometimes be hastened by dressings of gauze soaked in red lotion. The heels should also be carefully watched for the appearance of the hæmorrhagic blisters which herald the development of a sore. Rings for the heels may avert them, and air rings for the sacrum may also be needed.

SYRINGOMYELIA

Synonym.—Slatu Dysraphicus.

Definition.—Syringomyelia is a very chronic and irregularly progressive disease of the spinal cord and brain stem, dependent upon a peculiar lesion of the grey matter, glial increase and the formation of irregular cavities being the most conspicuous features of this lesion. Clinically, the malady is characterised by a deep loss of sensibility to pain and to temperature, other forms of sensibility remaining relatively unaltered, and by muscular atrophy and weakness of varying distribution in the upper extremities, and further by spastic weakness of the lower extremities, owing to involvement of the pyramidal tracts at the level of the lesion.

Ætiology.—Both sexes may be affected, and males are more prone to suffer than females. Heredity plays no part in its causation. Age is most important, in that this disease appears either to be congenitally installed, or to commence during the period of growth. It has been diagnosed with accuracy as early as the sixth year of childhood, and rarely if ever do the symptoms commence later than the age of 30 years.

Pathology.—The primary lesions of syringomyelia are always found in that region of the spinal cord which was originally occupied by the central canal, or in close connection with the ventricular system of the brain stem; and it is certain, therefore, that syringomyelia is referable to a pathological process affecting the central canal and its surrounding glia, and that this pathological process, in many cases at least, is installed before the completion of the development of the central canal of the nervous system. Two essential lesions and four other commonly occurring lesions make up the morbid process of this disease:

Essential lesions.—(1) Cavitation of the posterior part of the grey matter; (2) gliosis, with liquefactive degeneration of the abundant glia—*other lesions commonly but not invariably present*; (3) degeneration of lower motor and vasomotor neurones; (4) degeneration of lower sensory neurones; (5) distension of cavities producing pressure effects; (6) secondary ascending and descending degenerations.

The seat of commencement of the disease is invariably in the dorsal grey matter of the lower half of the brain stem and upper half of the spinal cord, and most commonly of all, in the lower three cervical and upper three dorsal segments. The cavitation occurs primarily always in that part of the grey

matter which held the original central canal, namely, the region of the posterior commissure, posterior median septum and posterior horns.

Degeneration of the lower motor neurones occurs in at least half the cases. It is most marked in the cervical and upper dorsal regions of the cord, and may be very local or extensive. It is commonly explained as caused by the extension of the gliosis and cavitation into the anterior horns of the grey matter, and by the distension of the cavities exerting pressure upon these regions, and causing atrophy of the cells. To the pressure exerted by such distended cavities are also attributed the ascending and descending degenerations which are commonly found in the pyramidal tracts below the level of the lesion, and in the posterior columns above.

Tumour formation is not uncommon in cases of syringomyelia. Massive growth may be found in the pons and in the spinal cord.

Symptoms.—*Disturbances of sensibility.*—By far the most constant and characteristic feature of syringomyelia is a sensory loss of a peculiar kind which was named by Charcot "the dissociated sensory loss." This is a loss of sensibility to painful impressions and to thermal stimuli, while sensibility to touch, to vibration, to position, to passive movement and to the appreciation of location upon the skin, remain relatively or entirely intact. In other words, those forms of sensibility which travel by a path crossing in the commissures of the spinal cord are lost, because the lesion of syringomyelia destroys especially the region of the commissures, while these forms of sensibility which travel by paths which are uncrossed in the spinal cord and do not traverse the region especially affected by syringomyelia, but are conducted by the posterior columns, are not affected. Further, the lateral region of the dorsal reticular formation of the medulla, mesial to the restiform body and ascending root of the fifth nerve, and a little ventral to these structures, is especially prone to the lesion of syringomyelia, and it is this region which contains the whole path for pain and temperature sensibility from the opposite half of the body, and a lesion in this situation will produce hemianalgesia and hemithermanæsthesia, while the paths for other forms of sensibility, situated mesially on either side of the *raphé* of the medulla, escape. Again, the lesion excavating the ventral horn in any part of the cervical region may extend so as to interrupt the spinothalamic tract which lies immediately dorso-lateral to the ventral horn, and so cause loss of pain and temperature sense on the opposite side everywhere below the level of the lesion.

The destruction of the commissures in the lower cervical and upper dorsal regions produces the dissociated sensory loss symmetrically over the thorax, upper extremities, neck and face, the distribution varying with the extent of the lesion. The sensory loss over the face is explained in that the sensory root of the trigeminal nerve has its ending in the upper three segments of the cervical spinal cord, and the pain and temperature sensibility of the face is interfered with if the posterior grey commissure in the region of these segments is damaged. Only rarely does the symmetrical sensory loss extend below the thorax, for the reason that the spinal lesion does not often extend below the mid-dorsal region. The sensory loss will vary in depth, extent and symmetry of distribution according to the completeness, extent and symmetry of the lesion. Thus, in early and slight cases, the sensory disturbance may not amount to more than a relative loss of pain and temperature confined to the

hands and ulnar borders of the forearms, while in an advanced case there is usually complete inability to appreciate painful and thermal stimuli over an area which would be covered by a sleeved jacket, and this area often extends over the neck and the face. Combinations of the "sleeved jacket" sensory loss with hemianalgesia and hemithermanæsthesia often occur in cases where both the spinal lesion and the medullary lesion are present. The dissociated sensory loss makes its advent insidiously, and is often unnoticed by the patient and discovered for the first time on medical examination. Or it may appeal to the patient, who on bathing finds that he appreciates heat and cold upon some parts of the skin and not on others. Not infrequently he finds that he injures himself or burns himself without noticing it at the time.

Subjective sensibility is not often affected, and for the most syringomyelia may be described as a painless disease; but there are very notable exceptions. Sensations of heat and cold, dull fixed pains, lasting neuralgic pains, and lightning pains in no way differing from those of tabes, may occur. These pains are confined to the regions which are the seat of the other symptoms. Especially important in this connection are those cases in which the distension of the cervical spinal cord is so great as to cause that structure to press upon the bones of the spinal canal. Here constant and often intolerable aching pain in the neck, upper extremities and thorax may result, with rigidity of the neck, and this may render life so insupportable as to necessitate surgical interference for the relief of the pressure.

Muscular atrophy.—This common clinical feature of syringomyelia is met with in considerably more than half the cases. As may be gathered from the nature of the lesions, though usually bilateral, it is often not symmetrical, and may be entirely confined to one side. The intrinsic muscles of the hands and the muscles of the ulnar side of the forearms are first and most affected in the ordinary run of cases. The atrophy is often here confined, but it may extend up the arm; but it is unusual for the whole upper limb to be affected. Sometimes the shoulder muscles are first affected, and again the scapulo-thoracic and humero-thoracic muscles may be early involved. The upper intercostals, and that section of the muscles which supports the spine, supplied from the upper six dorsal segments suffer, but the scalenes seem generally to escape. The muscular atrophy is strictly limited, and is apt to become complete in the muscles affected. The lesions of the medulla may involve the motor nuclei of the cranial nerves. Atrophic paralysis of the muscles supplied by the vago-accessory nerve is far from uncommon, and the discovery of this paralysis in a young subject should always arouse suspicions of the presence of syringomyelia. The paralysis is unilateral and involves palate, pharynx and all the muscles of the larynx upon the affected side. Similarly but in much rarer cases, atrophic paralysis of the face, of the trigeminal muscles, of the sternomastoid and trapezius or of the hypoglossal muscles may occur from a unilateral involvement of the corresponding motor nuclei. Fibrillation in the affected muscles is said by most writers to be of common occurrence. It has been conspicuous by its absence in most of the large number of cases which have come under our observation. One would expect it to be confined in syringomyelia to such times as the muscular atrophy is progressing.

Contractures resulting from the muscular atrophy are commonly seen in the hands, and the deformity resulting tends towards the "griffin's paw"

type, but hardly reaches the degree seen in ulnar nerve paralysis, and is often much modified by trophic and vasomotor changes, and by the results of injuries and whitlows.

The lower extremities escape so far as atrophy of muscles is concerned. Spinal curvature is present in many cases. It consists essentially in a kyphosis or kypho-scoliosis of the upper dorsal region, with a compensatory lordosis and lateral curve in the lumbar region. The upper convexity is to the left from the major use of the right hand. It is dependent upon paralysis of the trunk muscles, from involvement of the anterior horns in the upper dorsal region, and, in addition, dystrophic changes in the bones may be factors in its production. It is more marked the earlier it commences during the period of growth, and where heavy manual occupation has been followed.

Trophic and vasomotor disturbances.—Thickening of the bones or a condition of osteoporosis and brittleness may be met with. More often Charcot's arthropathy occurs. It differs in no way from the similar condition in tabes dorsalis, and is confined to the joints of the analgesic region, and affects the joints of the lower extremity only when there is a hemianalgesia from a lesion of the spinothalamic tract either in the cord or in the medulla. In syringomyelia Charcot's joints are seen chiefly in workmen who are engaged in occupations which constantly expose the analgesic joints to jarring and bruising.

The most characteristic of the trophic changes consists in thickening of the subcutaneous tissue and of the skin itself, which is seen in the hands. The fingers become thick and swollen and lose their natural outline, the tips become blunted, and the knuckle-folds thick and coarse, and some vasomotor paralysis renders them unduly red, or even blue. They have been termed "sausage-like" fingers, and often stand out in contrast to the wasting of the intrinsic muscles of the hand. A similar condition affecting the whole hand is common, and was termed by Charcot the "fleshy hand" or "main succulente." The analgesic condition of the hands and the thermanæsthesia present expose them unduly to injuries and, since these injuries are likely to be unnoticed or disregarded, septic infection arises easily, and the results of injuries, burns and whitlows are frequently seen, giving rise to further deformity from scars, loss of the terminal phalanges, from whitlows and contractures, and from sepsis extending to the tendons.

The lower extremities usually present a slight spasticity, with the signs of involvement of the crossed pyramidal tracts. This does not often produce much disability in the use of the lower limbs. In cases, however, where the lesions involve the lateral regions of the cord, either by direct extension or by the pressure of distended cavities, severe spastic paraplegia may result. And again, in very rare cases, such pressure may lead to total evascularisation and total transverse lesion of the spinal cord with the appearance of a complete flaccid paraplegia with incontinence, total sensory loss and absent deep reflexes.

Sphincter trouble is usually absent, or slight and occasional; but in cases where paraplegia is severe any degree may occur.

The skin reflexes of the trunk are diminished or absent, and the plantar reflexes are of the extensor type, according to the degree of pyramidal involvement. Some degree of pes cavus is often present. The knee-jerks and ankle-jerks are increased, and foot-clonus, etc., is present.

Considering that the efferent neurones of the cervical sympathetic system have their origin in the brain stem, and their exit from the spinal cord in the lower cervical and upper dorsal segments, thus traversing the whole of the region usually affected by the lesion of syringomyelia, the frequency with which paralysis of the cervical sympathetic occurs is easily understood. It may be complete or incomplete, unilateral or bilateral, and is recognised by smallness of the pupil, narrowing of the palpebral aperture (sympathetic ptosis), and a peculiar flatness of expression on the side of the face affected, with decrease or loss of sweating. These signs are much more obvious when unilateral than when bilateral, for, in the absence of the contrast which a normal side of the face gives, they are often overlooked when bilateral.

Ophthalmoplegia is very rare, but it may occur, since the syringomyelia lesion may be found as high as the region of the third nucleus. Nystagmus is an almost constant feature of syringomyelia, as it is also of most lesions of the cervical spinal cord.

MORVAN'S DISEASE.—This variety of syringomyelia is so peculiar in its clinical aspect as to need especial description. In addition to the lesion of the spinal cord characteristic of syringomyelia, there are intense changes in the periphery of the nerve trunks of the limbs. Instead of the usual loss of pain and temperature sensibility, distributed in jacket form upon the upper limbs and trunk, there is absolute loss of all forms of sensibility in the hands, wrist high, and in many cases also in the feet, ankle high. Progressive atrophy of the intrinsic muscles of the hands and feet occurs. Severe vasomotor paralysis brings about permanent cyanosis of the hands and feet, with much thickening of the skin and subcutaneous tissues, to which are added the effects of injury and septic processes in insentient regions, in the form of whitlows, necrosis and loss of digits. Another peculiarity of this malady is that the extremities are exceedingly painful in the early stages and until the sensory loss becomes deep. Morvan's disease resembles Raynaud's disease in the cyanosis and tendency to necrosis of the fingers and toes, but it is easily distinguished by the complete absence of intermitting vascular spasm and by the peculiar loss of sensibility. Anæsthetic leprosy may be distinguished from Morvan's disease by the characteristic skin lesions in other parts of the body, by the palpable thickening of the nerve trunks, and by the less definitely limited areas of sensory loss. Every transition between typical syringomyelia and Morvan's disease has been described.

Course and Duration.—The malady, commencing insidiously, progresses very slowly, and often ceases to progress for periods which may amount to many years. The tendency to the destruction of life is not great; but when rapid extension of the physical signs, and especially of paralysis and muscular atrophy of the upper extremities and respiratory muscles, occurs, the end is likely to come quickly. Signs of great distension of the cavities, such as pain and rigidity of the neck, and also severe and increasing paraplegia, with sensory loss of all forms of sensibility below the level of the lesion, point to a rapidly fatal termination.

It is not unusual to meet with well-marked cases in which the signs develop and increase during late childhood and early adult life, and then remain more or less in a stationary condition, allowing an occupation to be followed until well after middle life has been reached; but with the advent of the degenerative period of life, from the age of 45 years onwards, there is

always a slow increase of the disability which puts an end to useful capacity. Many of the cases become incapacitated in early life, after which the disease becomes arrested, and the patients live on for many years, sometimes in a bedridden condition. Few reach the age of 60 years. Rapid extension of the physical signs leads to death from involvement of respiratory muscles. Otherwise the patients succumb to intercurrent disease. Sudden and unexpected death sometimes occurs, and it is especially to be remembered that this is likely to occur after the administration of anæsthetics, and as a result of surgical procedures.

Diagnosis.—Syringomyelia has to be differentiated, in its early stages, from those diseases which cause slowly progressive muscular atrophy in the upper extremities, and, in its later stages, from other lesions of the central region of the spinal cord. Those cases in which the lesions are chiefly in the ponto-medullary region must be distinguished from other slowly oncoming lesions of the brain stem.

The age of onset, during the later years of childhood and the earlier years of adult life, is important, and during this period slowly developing paralysis, with or without muscular atrophy and with sensory loss, should always suggest the possibility of syringomyelia. Other causes, which may produce this symptom group, and which may be confused with syringomyelia, are local lesions of the peripheral nerves, local lesions of the brachial plexus, and, especially, the lesion produced by the presence of cervical ribs, root lesions, lesions of the central grey matter of the spinal cord, especially central tumours of the spinal cord, hæmatomyelia, and lastly certain general diseases of the nervous system, progressive muscular atrophy, peroneal atrophy and myotonia atrophica. That the sensory changes of syringomyelia of peculiar nature are usually the first signs of that disease is important; but unfortunately is not without many exceptions, both as to the nature of the sensory changes and as to their time of appearance.

Local lesions of the peripheral nerves produce signs which are confined to the distribution of the nerve involved; the sensory loss is to all forms of sensibility, and the condition is commonly unilateral. In syringomyelia, however, the lesion in the early stages may be confined to one side of the cord, and to one posterior horn so far as the production of sensory loss is concerned, and the muscular atrophy may be so narrowly confined to the distribution of the ulnar nerve as to cause close resemblance between the two conditions. Any sensory loss over the trunk, or signs outside the distribution of the peripheral nerve, will, if present, clearly divide the two conditions.

Cervical ribs may produce slowly progressive atrophy of muscles, pains and sensory loss, very difficult to distinguish from those resulting from syringomyelia. The diagnosis in these cases is beset with peculiar difficulties, for so frequently do cervical ribs produce no nervous symptoms at all that their presence, when demonstrated, does not argue that they are the cause of the symptoms. Again, cervical ribs are among the commonest of the developmental peculiarities which are so frequently seen in the subjects of syringomyelia. Slow muscular atrophy and slowly oncoming sensory loss and perhaps pain characterise both syringomyelia and cervical rib paralysis, and the distribution may be unilateral or bilateral in either condition; but it is only when the signs and symptoms are strictly confined to the upper

extremities and neck that difficulty arises. The slightest definite physical sign outside of this region at once turns the diagnosis in favour of syringomyelia, and of these signs cervical sympathetic paralysis, sensory loss on the trunk, and alteration of the abdominal and plantar reflexes are most important. A very careful search must be made for any such signs, and the patient observed over a considerable time before a certain diagnosis is made.

Lesions of the nerve roots, either from inflammatory conditions, bone disease, pachymeningitis or neoplasms give rise to more severe pain than does syringomyelia, and the development of the symptoms is much more rapid. Lesions of the central grey matter of the spinal cord may produce a symptom complex, closely resembling that of syringomyelia. Central tumours of the spinal cord, when of slow growth, are hardly distinguishable, inasmuch as the lesion of syringomyelia is in reality a central tumour of the cord. The majority of central tumours, however, are of more rapid development, and speedily produce severe paraplegia. The presence of Froin's syndrome (hyperalbuminosis in the cerebro-spinal fluid) is much in favour of tumour.

Progressive muscular atrophy in its early stages may cause difficulty in diagnosis, since the muscular atrophy in syringomyelia may in rare cases precede the appearance of any sensory loss or may be well marked when the sensory loss is slight. In this connection widely distributed fibrillation is of great importance in indicating a diagnosis of progressive muscular atrophy, particularly if it be seen in muscles not conspicuously wasted. In peroneal atrophy the atrophy of the intrinsic hand muscles is always preceded by a more extensive atrophy of the muscles below the knee, which are never atrophied in syringomyelia.

Syringomyelia of the brain stem may be distinguished from other lesions of this region by its insidious onset and the special tendency to the involvement of the lateral region of the medulla containing the vago-accessory nucleus and the central pain and temperature path, so giving rise to a unilateral paralysis of palate, pharynx and larynx with hemianalgesia and hemithermanæsthesia on the opposite half of the body. Often some signs of cervical syringomyelia coexist; but the medullary lesion may exist alone, and it cannot be too prominently borne in mind that any very slowly progressive lesion of the brain stem of insidious onset may be of the nature of syringomyelia.

Prognosis.—Recovery never occurs; but arrest of the disease for long periods is frequent. Those disabilities, which are the result of pressure or distension, may abate spontaneously or as the result of treatment, and in arrested cases training may bring about lessening of the disability. Increasing symptoms, especially if the increase be rapid, are always a cause for anxiety, and increasing involvement of the respiratory muscles is the gravest of events.

Treatment.—Some authorities believe that mercury and iodide of potassium have a definite effect in benefiting the disease when the symptoms are progressing. Application of deep X-rays to the cervical and upper dorsal regions of the spinal cord has been followed by arrest of the progress of the disease and, rarely, improvement of symptoms. Pains are to be relieved with the common analgesics. Massage, exercises and training are all likely to make some improvement in the disability in arrested cases.

HÆMATOMYELIA

Ætiology and Pathology.—Hæmatomyelia, or hæmorrhage into the spinal cord, has in the past figured in neurological literature to a much greater extent than its frequency warrants. It has been commonly supposed, for example, to be an almost invariable consequence of serious local injuries of the cord, whereas in fact in these circumstances hæmorrhage plays a very minor rôle. G. Jefferson has in fact expressed the view that it is doubtful whether such a state as hæmatomyelia actually exists. It is true that small punctate hæmorrhages may be seen in the damaged areas, but as Holmes, from his study of the spinal injuries of warfare, has pointed out, the essential lesion in these circumstances is œdema, followed by degenerative changes in the cells of the grey matter, and in severe cases by central necrosis. Large central hæmorrhages are not found. Similarly, certain inflammatory affections of the cord, such for example as acute poliomyelitis, may show minute hæmorrhages, but here again nothing occurs to which the term hæmatomyelia can reasonably be applied. In short, hæmatomyelia is an exceedingly rare form of spinal cord lesion.

A recent study by C. Richardson made on material at the National Hospital, Queen Square, has lent a necessary sense of proportion to our views on this matter. By the term hæmatomyelia is now usually meant a large central hæmorrhage, with a tendency to spread longitudinally over several segments. The central region of the cord and the dorsal horns are the situations in which this hæmorrhage is commonly observed in the rare instances in which it is to be found. If we agree that traumatic hæmatomyelia is an extremely rare condition, we are left with what has been called "spontaneous hæmatomyelia" and with secondary hæmatomyelia.

SPONTANEOUS HÆMATOMYELIA.—This rare condition appears to arise only when there is some abnormality of the spinal vessels. Such abnormalities are angioma and other malformations, and—excessively rarely—syphilitic and arterio-sclerotic disease of spinal arteries; in other words, the so-called spontaneous hæmatomyelia is really secondary hæmatomyelia.

Symptoms.—From what has been said it is apparent that the clinical picture usually given of hæmatomyelia is that of local trauma of the cord, and since such local crushing is commonly associated with fracture, fracture-dislocation, or transient dislocation of the cervical spine between the fifth and sixth vertebræ, this picture is that of a quadriplegia of sudden onset with slow recovery of variable degree. True hæmatomyelia may indeed occur in the cervical region of the cord, when it is apt to prove fatal. Richardson's analysis indicates that the characteristic clinical picture is that of a sudden paraplegia, accompanied by pain and sensory changes, subjective and objective. It appears that all forms of sensation are lost at first.

In the non-fatal cases a variable degree of recovery is possible. In a few reported cases, it appears that a syringomyelic type of dissociation of sensation may ensue.

Diagnosis.—The diagnosis of primary hæmatomyelia rests upon the sudden onset, the rapid development of symptoms which soon come to a standstill, and the physical signs of a central lesion of the spinal cord. The

distinction has to be made from acute myelitis. Acute myelitis, though rapid in onset, does not show the sudden development of symptoms seen in hæmatomyelia. Prodromata often precede the onset.

Treatment.—The general treatment is that of any severe spinal cord lesion with paraplegia. When there is evidence that a syphilitic lesion of the spinal arteries is in question, the treatment is that of spinal syphilis. Angioma and other malformations of the spinal vessels are not amenable to surgical intervention.

MYELOMALACIA

Synonym.—Softening of the Spinal Cord.

The term "myelomalacia," which implies softening of the Spinal Cord, has been applied by some authors to those conditions of local destruction of the spinal cord consequent upon the cessation of blood supply, and especially upon thrombosis of its blood vessels, as apart from the extensive local destructions which may result from inflammatory conditions. Such a distinction does not rest upon any logical, pathological or clinical basis, for thrombosis and ischæmia make up a part of the pathological process of all traumatic, inflammatory and pressure lesions of the spinal cord, and may occur as terminal events in certain diseases of the spinal cord where vascular lesions are otherwise conspicuous by their absence. Therefore, since softening of the spinal cord may be the result of widely different pathological processes, and since it does not constitute a definite clinical entity, it will suffice here to refer to those maladies in which it is chiefly observed.

TRAUMATIC CONDITIONS.—As a result of high explosives bursting in the neighbourhood of the spinal column, even without signs of external injury or signs of damage to the bones, the spinal cord may be found to be completely diffuent over several segments. The same result may be met with from the passage of a high velocity bullet through the spinal canal, whether the spinal cord be touched by the bullet or not; and again, the same condition occurs from the vibration of an impact when a missile hits and lodges in the surrounding bone, without directly involving the spinal canal or cord. A slighter degree of the same condition may be seen in fracture dislocation. When, as the result of injury to the spinal column, the spinal cord is torn across, the distal segment may soften completely.

PRESSURE LESIONS.—Pressure upon the spinal cord abrogates function chiefly by producing ischæmia and, if the pressure be prolonged or severe, necrotic softening occurs, and the more readily, if there be strangling of the segmental vessels which supply the cord and accompany each nerve root.

INFLAMMATORY CONDITIONS.—In acute spreading myelitis, in which the spinal cord is infected with micro-organisms secondarily to a general blood infection, as may occur in small-pox, gonorrhœa, dysentery, etc., the cord softens and may become diffuent. In acute transverse myelitis, softening depends upon the severity of the initial œdema and its duration, the degree of obliterative arteritis, and the consequent thrombosis that may occur. It may be largely avoided by the energetic and early application of anti-syphilitic treatment.

SENILE PARAPLEGIA.—This condition, which is not very rare, and in which

spasticity of the lower extremities with weakness comes on gradually in later life, and does not, as a rule, reach a severe degree, has been attributed to ischæmia and even to softening of the spinal cord from arterial disease and the failing circulation of old age, by Moxon, who first described it. The pathology of these cases seems by no means certain, and there are few records of the anatomy. It seems certain that no appreciable softening can occur, on account of the slightness of the paraplegia and the absence of any sensory loss. Gowers doubted whether they were spinal in origin at all, and attributed some to the occurrence of cortical changes in the brain, while others he placed in the category of paralysis agitans. From the occurrence of definite mental failure in some of the cases, a cerebral site for the lesion is likely. Dr. Greenfield has recently examined for us a very typical case and found no changes in the spinal cord, but extensive degeneration of the pyramidal cells of the motor cortex.

SUBACUTE COMBINED DEGENERATION

Synonym.—The Anæmic Spinal Disease.

Definition.—Subacute combined degeneration is a disease most common in the second half of adult life, of which the onset is usually insidious and the course progressive. The lesions in the nervous system consist of a primary demyelination, commencing in the centre of the white columns and affecting the long fibres first and most, and the short intersegmental fibres which lie close to the grey matter last and least. Neuroglial condensation follows very slowly upon the demyelination. The posterior and lateral columns of the spinal cord are early affected, and it is to the affection of both these columns that the term "combined degeneration" alludes. The clinical features are usually strikingly distinct, in that subjective sensations, such as tingling, numbness and burning, occurring usually at the periphery of the limbs, are early, obtrusive and persistent, and are accompanied or followed by the development of a paraplegia which may be of a spastic, or a flaccid and ataxic, or of a mixed type, according to the degree of affection of the lateral and of the posterior columns in each case, and the degree of involvement of the peripheral nerves. We owe to Risien Russell, Batten and Collier the first complete pathological and clinical account of this disease.

In the late stages of the malady, the paraplegia tends to become complete and of the flaccid type, with loss of the deep reflexes. Anæmia accompanied by a peculiar "buscuit-like" discoloration of the skin is present in all cases at some period of the disease, with the exception of some few of the cases of short duration. This anæmia tends in every case, if life is prolonged, to develop into a pernicious anæmia which is typical, both clinically and pathologically.

Ætiology.—First met with in the third decade of life, the malady becomes increasingly frequent until a maximum incidence occurs in the sixth decade, while cases commencing in the seventh decade are not uncommon. The sexes are equally affected. Familial incidence in this disease, as also in pernicious anæmia, has been recorded by Hurst, Piney and others.

Pathology.—The essential lesion is demyelination of the axis cylinders, and subsequent degeneration of the latter, in the posterior and lateral columns of the cord. It has also long been known that some degeneration of peri-

pheral nerves occurs in this disease, and recently Carmichael and Greenfield have confirmed that the same process of demyelination seen in the cord is to be found in the peripheral nerves. At first the myelin sheath swells and later disintegrates. This change first occurs in the lower dorsal region of the cord, and is first seen in the centre of both posterior columns, and soon afterwards in the centre of either lateral column, as small areas of a darker and more translucent appearance than the normal white matter. It is only at an early stage of the disease that the anatomical picture is strictly one of postero-lateral degenerations, for soon after, spots of degeneration appear on either side of the anterior median fissure and in other parts of the antero-lateral columns. The degenerated areas increase in size centrifugally, coalesce with one another, reach the surface of the cord and eventually involve the whole of the white matter of the cord as seen in transverse section, with the exception of the narrow zone of short internuncial fibres which everywhere clothe the grey matter. This "annular or ferrule-like" degeneration in the lower dorsal region is highly characteristic, and occurs in no other disease.

From its starting-point in the lower dorsal region the degeneration spreads upwards and downwards in the white columns of the spinal cord, and for this reason the term "funicular myelitis" was applied to it by Henneberg. This extension depends upon the occurrence of small isolated spots of degeneration in the posterior, lateral and antero-lateral columns, which increase in size and thus join the area previously degenerated. The degeneration tends to extend upwards indefinitely, and in severe and advanced cases has been found as high as the internal capsule in the pyramidal tract.

The lesions of the white columns entail the usual secondary degenerations, both ascending and descending; but these occur late, and are often much less obvious than might be expected from the severity of the local lesions. The destruction of the axons by the local lesions also causes a series of retrograde changes in the corresponding nerve-cells, and tigrolysis, vacuolation, shrinking and neurophagy may be conspicuous, especially in the cells of Clarke's column and in the cells of Betz, which gave origin to the pyramidal fibres. Occasionally the disease is entirely confined to the posterior columns of the spinal cord. The muscles are conspicuously wasted in the later stages, and the muscle fibres show great diminution in size and poor striation. There is not any considerable increase of the muscle nuclei, and little or no fibrosis occurs.

Blood.—In a few instances, anæmia has been absent throughout, the hæmoglobin content and the cytology being normal; this has occurred chiefly in cases which have run an acute and fatal course in a few months. Usually the blood shows an anæmia of varying severity; the hæmoglobin ranges from 35 to 75 per cent., the lower of these figures being common; the colour index is usually above the normal, and may be as high as 1.6. Macrocytosis is present. Anisocytosis, poikilocytosis and polychromatophilia are common. Normoblasts are often numerous and megaloblasts may be found in numbers. A relative lymphocytosis is almost always present, and may reach as much as 55 per cent. This change occurs early, and is helpful in the confirmation of the diagnosis of the nervous disease.

A careful investigation of the blood-changes at various stages of the disease and of the post-mortem findings in a large series of cases has proved beyond any possible doubt that the blood-changes in every case are identical with

those met with in the various stages of pernicious anæmia, and that a typical post-mortem picture of pernicious anæmia occurs frequently in subacute combined degeneration. The cerebro-spinal fluid presents no abnormalities either as regards albumin, sugar or cells.

The early writers believed that the anæmia was the essential part of the disease, and that the degenerations in the nervous system were the result of vascular changes consequent upon the anæmia. This view is negatived by the facts that some cases progress to a fatal issue without any evidence of anæmia, and that in others the nervous manifestations may become severe long before any anæmia is evident; and, most importantly, no case has been recorded in the literature, nor has one occurred in the very large series examined by the writers, in which the nervous manifestations developed in a patient already under observation for anæmia.

The experimental evidence and the clinical and pathological features of the disease suggest, therefore, that the anæmia and cachexia and the degeneration of the nervous system are not dependent the one upon the other, but that they are the concomitant but not necessarily synchronous results of one and the same cause, which is deprivation from a product of gastric digestion in the presence of hydrochloric acid which is subsequently stored in the liver, and which is essential to the normal formation of the erythrocytes. Hurst and others have shown that achlorhydria is present in nearly all the cases.

Symptoms.—In a large majority of instances the symptoms appear insidiously and without any exciting cause. Sometimes the onset is more rapid, and may be preceded by severe gastro-intestinal symptoms such as vomiting, diarrhœa, jaundice, malaise and pyrexia. In a few cases the onset has been so rapid as to suggest the diagnosis of acute myelitis, and in one of these which was under our observation and pathologically verified, two attacks of temporary paraplegia has preceded the onset by 8 and by 4 months respectively.

The cardinal signs may be summarised as follows: peripheral subjective sensations, which occur early and are remarkably obtrusive, are complained of in the periphery of the limbs in most cases, but may occur in the perineum, neck and back of the head and in the tongue. Sensory loss is found, which commences upon the limbs with peripheral "stocking and glove" distribution, and reaching on to the trunk ascends in segmental distribution. Astereognosis occurs in the upper extremities. Paraplegia may be (a) flaccid from the first, with loss of deep reflexes; (b) spastic, remaining spastic throughout (rare); (c) spastic, changing to flaccid paralysis with loss of the deep reflexes. The first of these three clinical types is the commonest, and it provides almost all—if not all—the cases which respond favourably to treatment. Both forms of paraplegia are accompanied by marked ataxia. Girdle sensations, lightning pains, fixed pains, gastric crises, exaggeration of superficial reflexes, are all encountered. Sphincter paralysis is late. Loss of sexual power is early. There are muscular wasting and lowering of electrical excitability of general distribution in the paraplegia region. Anæmia, which may be absent throughout or may become apparent at any period in the course of the disease, is conspicuous at the time of the onset of the nervous symptoms in about one-half of all cases.

Peripheral subjective sensations are so constantly the earliest symptom, discomforting to the patient and so persistent, as to form a most distinctive

feature of the disease. These sensations are variously described, but tingling and numbness are the most common. Creeping sensations, smarting, burning, icy coldness, tightness and pain are all common. They are usually felt first upon the tips of the fingers and toes, and subsequently spread up the limbs. A girdle sensation is the rule, and it is sometimes painful.

Sensory loss first appears at the periphery, and spreads up the limbs like the sensory changes of a polyneuritis, waning in severity as the base of the limb is approached. In time it reaches the abdominal wall, but rarely extends as high as the thorax. Earliest to diminish, and first to disappear, are the postural modes of sensibility and vibration sense; tactile sensibility may also be severely affected; but thermal and cutaneous pain sense are usually least affected. A marked and almost constant feature of the disease is the tenderness of the calf and plantar muscles to pressure. Such tenderness is not usually found in association with a spinal cord lesion, and its presence is one of the reasons for regarding the peripheral nerve changes already referred to as of importance in determining the symptomatology.

In the common *flaccid type* of paraplegia, weakness and unsteadiness of gait are commonly preceded by the paræsthesiæ and sensory changes described above. There is at first a ready fatigue on exertion, a dragging of the feet and an ataxy of gait when tired, and also aching pains in the muscles of the legs. Examination reveals some weakness, especially of flexion and dorsiflexion in the legs, the tenderness already mentioned, and diminution or loss of the knee-jerks, and loss of the ankle-jerks. Sooner or later an extensor plantar response betrays the development of lesions in the lateral columns of the cord; but at first the plantar response may be of the flexor type, and when this is the case, the nervous clinical picture is not readily distinguishable from that of polyneuritis. In some cases it is doubtful if the distinction could be made were it not for the accompanying abnormalities in the blood and gastric secretions.

In this clinical type, of course, some of the signs may be the expression of a posterior column lesion, and it is known that in a few cases the spinal cord lesion is confined to this column. In the less frequently seen *spastic type*, the clinical picture is one of a predominating lateral column lesion in the cord. The case may pass through the stage of paraplegia-in-extension to the final one of paraplegia-in-flexion, with extremely painful flexion spasms, loss of sphincter control, and the development of bedsores. In some such cases, the limbs may become flaccid, and the tendon-jerks disappear before contracture ensues. But whether this change from spasticity to flaccidity occur or not the clinical type is one which progresses ruthlessly and does not respond to treatment.

Sudden exacerbations of the symptoms may occur at any time, and these are commonly associated with malaise, pyrexia, vomiting or other signs of gastro-intestinal disturbance and by an increase in the anæmia, as if there had been a sudden increase in the condition, which is responsible both for the anæmia and for the spinal degeneration. As the disease advances, the paraplegia involves more and more of the trunk, progressing upwards. In some cases the upper extremities are affected early, and may even be the first regions to show signs of the disease. In the course of time the paraplegia becomes complete, with great wasting of the muscles and reduction of their faradic excitability.

The paraplegia does not, as a rule, reach the upper limits of the region supplied by the cervical enlargement of the spinal cord, and even in the most severe cases the condition of the upper extremities is one of partial paralysis, most marked in the periphery and associated with considerable wasting of the muscles of the hands and forearms. In addition to the muscular wasting, there is usually conspicuous wasting of the subcutaneous fat. In late stages of the disease the general bodily wasting becomes extreme.

Dysuria generally appears when the paraplegia becomes pronounced. It does not often occur in the early stages of the malady, and sometimes its appearance is delayed until remarkably late. When once established, it does not show any tendency to improve with treatment. Finally, the control of the rectum and bladder becomes completely lost.

Soft translucent œdema of the extremities and trunk is frequent, especially when the anæmia is severe, and is dependent upon the anæmia and upon the impaired innervation of the paraplegic region.

General mental deterioration, mild delirium, drowsiness and torpor frequently occur at any stage of the disease, and are referable to the anæmia and the metabolic disturbance, and possibly also to widely spread cell changes in the cerebrum. General convulsions have been reported in a few cases.

Dimness of vision is common when anæmia and debility are severe. Papill-œdema of slight degree is sometimes met with, and doubtless in relation to the anæmia. Optic atrophy has been reported in a good many cases. Small retinal hæmorrhages are not uncommon. A minor degree of nystagmus is the rule, and may depend upon the involvement of the cervical spinal cord, all lesions of which seem to be regularly associated with nystagmus, or this may be attributed to affection of the cerebellum, for changes in the Purkinje cells of this organ have been repeatedly found. Herpes is not infrequent. It may occur anywhere, and has several times affected the distribution of the trigeminal nerve. A hæmorrhagic lesion of the sensory ganglion has been found.

Although anæmia is one of the most characteristic features of subacute combined degeneration, since it occurs in every case of long duration at some time or other, and since it is sufficiently striking as at once to suggest the diagnosis in at least two-thirds of all the patients when they first come under observation for nervous symptoms, yet it may be absent throughout the course of the disease in a rapid case, and its appearance may be delayed until several years after the disease of the nervous system is manifest. The anæmia in almost every case is identical in every respect with pernicious anæmia. Of those cases in which the blood picture is not typical, nearly all show megalocytosis, with a relative lymphocytosis and a high hæmoglobin index, as do early cases of pernicious anæmia, and it may be said with certainty that the longer the patient survives, the greater the likelihood of typical pernicious anæmia developing. The spleen has been enlarged in many cases, and the marrow of the bones is typical of pernicious anæmia, as may be also the iron reaction in the liver and the changes in the myocardium and other muscles. As in pernicious anæmia, the tongue is clean, and this occurs so regularly that any appearance of furring of the tongue may justly be said to exclude the diagnosis of this disease. Fractional test meals show an absolute achlorhydria, or a relative achlorhydria, in the same proportions

as do cases of pernicious anæmia. The colour of the skin is often peculiar and striking, even when anæmia is not severe, and is best described as "biscuit-coloured." A bright malar flush upon this yellowish biscuit-coloured background gives a characteristic and vivid facial aspect in the earlier stages of many of the cases. The symptoms and signs common to all anæmic states, breathlessness, headache, cardiac and venous murmurs and œdema, are commonly present, but hæmorrhages are uncommon. Syncopal attacks may occur. Irregular pyrexia is almost invariably present at some period in the course of the disease, and this quite apart from fever-producing complications, such as cystitis and bedsores. In the later stages progressive emaciation is constant, and if life be prolonged it becomes extreme.

Diagnosis.—In the earliest stage, and before the appearance of any definite sign of organic spinal disease, there may be such disability as to suggest the diagnosis of functional paraplegia. When organic signs appear, it is especially from disseminated sclerosis, spinal tumour and tabes dorsalis that the diagnosis has to be made. The preponderance of the peripheral subjective sensations, and the presence of a florid complexion with anæmia, should always suggest the diagnosis. Slight spastic ataxy is the common clinical picture of subacute combined degeneration, of disseminated sclerosis and of spinal tumour. Peripheral sensations and peripheral numbness are not features of disseminated sclerosis, and the presence of peripheral sensory loss should always challenge that diagnosis, whereas diplopia, nystagmus, transient amblyopia and intention tremor are not early symptoms of subacute combined degeneration. Spinal tumour is especially distinguished by a sharp line of sensory loss, transverse to the axis of the body, which does not spread up from below in slow fashion.

When subacute combined degeneration commences with flaccid ataxy and loss of the deep reflexes, the distinction must be made from tabes dorsalis. The extensor plantar reflex, which is almost always present in the former disease and which is rare in early tabes, the entirely different distribution of the sensory loss in the two diseases, the loss of power in subacute combined degeneration, and the results of the examination of the blood and cerebro-spinal fluid for syphilitic reactions and of the latter fluid for lymphocytosis, are important aids in the differential diagnosis.

In the well-developed stages of the disease, its recognition presents no great difficulty. Attention is quickly attracted by the conspicuous anæmia and biscuit-coloured skin. Following a period of slight paraplegia, often lengthy, the steadily increasing paralysis of the lower extremities, with perhaps sudden exacerbations, producing complete and lasting helplessness, the characteristic distribution of the sensory loss which spreads upwards towards the cervical region, the severe lightning pains, the irregular pyrexia, the anæmia and the relatively late onset of sphincter trouble serve to separate this disease from other forms of paraplegia. The change from the spastic to the flaccid type of paraplegia with loss of the deep reflexes and persistence of the extensor response, which occurs in some of the cases in the late stages, is highly characteristic.

It is also necessary to bear in mind the strikingly close resemblance which the disease we are considering may bear to polyneuritis. The differentiation may in the early stages depend chiefly, if not wholly, upon the examination

of the blood and the result of a fractional test meal. But, sooner or later, the appearance of an extensor plantar response will indicate the presence of a cord lesion. On the other hand, in the spastic type, the presence of muscular tenderness in the legs is a strong indication in favour of subacute combined degeneration.

Course and Prognosis.—The duration of the disease varies within wide limits, but the rapid downhill progress, ending in death within a few weeks or months which was formerly common, is now exceptional. Conflicting claims are made as to the possibility of cure, and it is probable that some at least of the difference of opinion depends upon a failure to appreciate that in the common flaccid type of the disease some at least of the signs and disability may be due to a peripheral nerve lesion and not to degeneration within the cord. This is certainly the clinical type which responds most favourably to liver, stomach, iron and thyroid therapy; while all but the very slightest cases of the spastic type—in which the important lesion is plainly in the lateral columns of the cord—fail to respond to the most intensive therapy.

In the flaccid type, appropriate treatment—if given early enough—will effect considerable restoration of muscular power, of co-ordination and of sensation. Usually, however, paræsthesiæ in the legs and feet, and sometimes in the fingers, remain. The restoration of lost knee-jerks is rare, and that of lost ankle-jerks still rarer. Vibration sense may be partially restored, and very occasionally an extensor plantar response may disappear. It is yet too early to say how enduring these improvements may be. It is clear that for most sufferers, the prognosis of subacute combined degeneration is far better now than it was formerly, but the enthusiasm that would speak of cure as within reach, or prevention of nervous lesions in cases of pernicious anæmia when treated sufficiently early, as certainly possible, has still to justify itself. It is not shared by the neurologist.

Treatment.—Whatever the degree of anæmia present, intensive liver treatment is essential. Daily parenteral injections of the chosen preparation is necessary until the red cell count reaches the five million figure, or as near it as possible. Even this may be insufficient to secure improvement in the nervous symptoms. Indeed these require more and longer continued liver treatment than does the anæmia. The spastic type of case responds badly, but the more numerous flaccid cases may make remarkable recoveries if taken in hand before the malady is too advanced. Previous to the introduction of liver feeding we found that thyroid extract had a remarkable effect in removing the anæmia, and that it could be tolerated by patients suffering from subacute combined degeneration in very large doses, even as much as sixty grains a day. The more advanced the stage of the disease is, the less result may be expected from any form of treatment. Any suppurative condition of the body should be energetically treated. Every care should be taken to delay the advent of bedsores and cystitis. When present, these are often amenable to treatment in the early stages of the disease and in less acute cases, but in the more acute cases and in the later stages they are inevitable and the bodily vitality is too low for any reparative process to take place. Lightning pains and other pains are relieved by such analgesics as aspirin, acetanilide, amidopyrine, phenazone, etc. Reflexor spasms are among the most troublesome of the symptoms, since their frequent occurrence

denies sleep to the patient, and they are most important factors in the occurrence of bedsores. The remedy which seems to have most effect in checking these spasms is barbitone.

MOTOR NEURONE DISEASE

Synonyms.—Progressive Muscular Atrophy; Amyotrophic Lateral Sclerosis; Chronic Bulbar Palsy.

Definition.—A disease of gradual onset which may develop at any age from puberty onwards, and in which the anatomical findings consist invariably, whatever be the clinical picture, of three orders—(1) a progressive degeneration, shrinkage and disappearance, cell by cell, of the upper motor neurones or cells of Betz in the ascending frontal convolution, with consequent degeneration of the corresponding fibres in the pyramidal tracts; (2) a similar atrophy, cell by cell, in the lower motor neurones with corresponding degeneration of motor fibres in the peripheral nerves and atrophic degeneration of the muscles innervated by the affected cells; (3) a diffuse atrophy of the white matter of the spinal cord, the posterior columns conspicuously excepted.

A most mysterious feature of the disease is the non-correspondence between the anatomical findings and the symptomatology. In the first place, though the upper motor neurone lesion is constant, many cases run their course without the slightest external evidence that the pyramidal system is involved.

The clinical picture is one of gradually oncoming weakness and disability, due either to atrophy of the muscles from the lower motor neurone lesion, in which case the paralysis is flaccid and atrophic, or to spastic paralysis of the muscles from the upper motor neurone lesion, in which case the paralysis is spastic without atrophy, or to the combined lesion of both upper and lower motor neurones, in which case the paralysis is both spastic and atrophic, and the muscular atrophy never becomes complete. Fibrillary twitchings of the muscles are always present, and form an important diagnostic feature. Any of the skeletal muscles may be affected from the ocular muscles to those of the feet.

The clinical aspect varies greatly according as the incidence of the palsy is upon the muscles supplied by the brain stem, or upon the muscles of the trunk and limbs, and again, according as the atrophic element or the spastic element is present alone, or as both coexist in the same region or in different regions of the body.

The following are the usual clinical types, but it must be borne in mind that every transition between these types may be met with:

(A) With incidence upon the muscles supplied from the brain stem; Progressive bulbar paralysis: 1. Pure atrophic bulbar paralysis. 2. Spastic atrophic bulbar paralysis. 3. Pure spastic bulbar paralysis.

(B) With incidence upon the muscles of trunk and limbs: 1. Pure atrophic type—(a) local and slowly progressive; (b) general and rapidly progressive. 2. Spastic atrophic type; amyotrophic lateral sclerosis—(a) the spasticity and atrophy are coincident in the same muscles; (b) the atrophy affects the upper limb and the spasticity the lower limb. 3. Pure spastic type. This is more commonly seen as an early stage of amyotrophic

lateral sclerosis, where the spasticity of the lower extremities precedes the atrophy of the upper extremities by some months or years.

(C) Mixed bulbar and spinal forms.

Ætiology.—The earliest age incidence has been at 12 years, and several cases have been recorded which developed the disease at that age. As age advances the incidence of the malady becomes more frequent, until it attains a maximum between the ages of 30 and 40 years, after which there is a slow decline. It does not commonly commence in advanced age, but one case has come under the writers' observation which commenced at the age of 77 years. Males are affected three times as frequently as females, but in the cases occurring before the age of 25 years, the females predominate. Heredity only rarely influences the disease. The question of the relation of trauma to the causation of this disease admits of no decisive answer. In any given case it is impossible to establish a relationship, but some observers have recorded examples of a close sequence of injury and onset of the disease, and they regard the two as in the relation of cause and effect. We know of no pathological process by which a peripheral injury may set up within the central nervous system a selective neurone degeneration. If such there be, it has yet to be discovered. A series of cases of injury to the cervical spine associated with the signs of relatively minor injury to the fourth and fifth segments of the cervical cord which have been recorded by Walshe and Ross, raise the possibility that some cases of so-called progressive muscular atrophy are traumatic in origin, but are not at the outset or in their subsequent development genuine cases of motor neurone disease (cf. p. 1725, Section on Compression of Rapid Onset). Syphilis seems to be in definite causal relation with some of the cases. A positive Wassermann reaction, both in the blood and in the cerebro-spinal fluid, is found in a much larger proportion of the cases than give any history of syphilitic infection. Further, quite a number of instances of the supervention of a typical progressive muscular atrophy in cases of tabes has been observed and recorded at the National Hospital, London. It has been freely stated that progressive muscular atrophy of syphilitic origin differs from the non-syphilitic forms in its lack of symmetry and in its course; but this certainly does not hold good for very many of the cases which show a positive Wassermann reaction. In a large majority of all cases of progressive muscular atrophy, no causal factors whatever can be discovered.

Pathology.—To the naked eye, a cross-section of the spinal cord may show some diminution in size of the ventral horns. The essential lesion is a primary degeneration of the cells of the ventral horns of the spinal cord and in the homologous motor nuclei of the brain stem, namely, the hypoglossal, facial, trigeminal and oculo-motor nuclei. Coupled with the degeneration of the lower motor neurone, is a degeneration of the upper motor neurones of the pyramidal system. In the ventral horn cells the degeneration is evidenced by a gradual shrinking in size of the cells, which lose their dendrites and become oval or spherical in shape. The Nissl bodies slowly disappear, and only in rare and rapid cases is definite chromatolysis seen. The nuclei dwindle and become irregular and distorted.

The dorsal and lateral horns are almost invariably intact, but degenerative changes are sometimes seen in the cells of Clarke's column. The affection of the motor nuclei of the brain stem in the bulbar cases is in every way similar

to that of the ventral horns. The degeneration of the motor nerves which take origin from the degenerate ventral horn cells, often proceeds *pari passu* with the degeneration of the cells. But in some cases this is conspicuously and very mysteriously not the case.

The affected muscles are soft and toneless, and the muscle fibres are found irregularly degenerated, bundles of normal and of degenerating fibres, until the atrophy is complete, being found side by side. The characteristic change is shrinkage of the affected fibre to a calibre much less than normal. As is usual in all slow tissue degenerations, fibrosis and local arterial disease accompany the atrophy of the muscle fibres.

The pyramidal neurones (cells of Betz), which characterise the precentral cortex, undergo a degeneration very similar to that of the ventral horn cells, but with this difference, that the earliest structural changes are found in the most distal part of the pyramidal fibres, and that subsequently these fibres die back towards their cells of origin in the cerebral cortex. The degeneration of the upper motor neurones never proceeds to the complete destruction of anything like all the pyramidal fibres.

The pathological nature, therefore, of progressive muscular atrophy is a widely scattered degeneration of nervous elements not even confined to the motor systems, though these are in the main affected, since the afferent spino-cerebellar tracts are constantly found degenerated, from some unknown cause.

Symptoms.—The following description of the clinical features is based upon an analysis of 500 cases which have come under observation at the National Hospital, London. The onset is in most cases very gradual, but it may be more rapid, and severe incapacity may result in the course of a few months. In rarer cases, a severe degree of paralysis may develop in the course of a few days, and in such cases it is not uncommon to see the most remarkable temporary improvement. The nature of the onset, as a rule, indicates the course which the malady will pursue. A very slow onset is followed by a very slowly-advancing disease, often interrupted by long stationary periods, whereas the more rapid the commencement, the quicker will be the advance and the sooner will a fatal issue occur. Accompanying and sometimes preceding the onset, and not infrequently conspicuous during the early states of the disease, are certain sensory symptoms which, from the confusion in diagnosis they may cause and from the scant attention which has been paid them in descriptions of the malady hitherto, deserve emphasis. These symptoms are confined to the regions where the wasting first appears, and consist in a subjective feeling of stiffness and uselessness, much increased when the limb or the body is cold. Or there may be dull aching pains, intermittent neuralgic pains which may be severe, or a sensation of coldness or numbness which may be intense. Painful cramp in the muscles which are about to be affected is comparatively common.

The *muscular wasting*, which constitutes the most characteristic feature of the disease, may commence in any group of the skeletal muscles whatsoever. It may be first manifest in such rare situations as the facial muscles, intercostal muscles, muscles of the back and abdominal muscles. The commonest situation is in the muscles of the upper limb, where the distal (intrinsic muscles of the hand) or the proximal muscles (deltoids, spinati, etc.) are first affected in about an equal number of cases. In the hand, the muscles

of the thenar eminence are the first to waste, and this is followed by atrophy of the hypothenars, of the lumbricals and of the interossei with the usual flattening of the palm, exposure of the flexor tendons in the palm from loss of the bulk of the lumbricals, hollowing of the interosseal spaces and a tendency to the "griffin's paw" attitude of the hand. The *main en griffe* is never so marked in this disease as in paralysis of the ulnar nerve, syringomyelia, etc., because the wasting soon affects the long flexors of the fingers, and further contractures of the affected muscles are not well marked in progressive muscular atrophy. As the wasting spreads to the muscles of the forearm, the flexors are usually affected before the extensors.

When the upper arm is primarily affected the wasting is first seen most often in the deltoids, whence it spreads upwards, involving the spinati and the muscles attaching arm to scapula, and arm and scapula to trunk. Among these muscles some tend to escape the atrophy relatively, or to be affected much later than others, and these are the triceps, the latissimus dorsi, the lower half of the pectoralis major, the levator anguli scapulæ and especially the upper half of the trapezius, which for this reason was called "*ultimum moriens*" by Duchenne. In the limbs the wasting always commences in one limb, but soon spreads to the corresponding limb of the opposite side and tends ultimately to become symmetrical. The attention of the patient may be first drawn to his malady by the altered appearance produced by the atrophy, and this is more common when the commencement is in the hands, where the subcutaneous tissue is thin and the region constantly in view. Or the disability consequent upon the weakness may be noticed first, and this is always the case where the commencement is in the bulbar muscles, and usually also where the muscles of the legs, proximal muscles of the arms and trunk muscles are first involved. Lastly, the fibrillation may be so marked as first to attract notice.

The *loss of power*, which accompanies the muscular wasting, is, as a rule, commensurate with the wasting, and does not become absolute until the atrophy is complete. To this rule, however, there are two very important exceptions. In the first place, when the affected muscles are both tonic from the upper motor neurone lesion and atrophic from the ventral horn-cell lesion—the tonic atrophy of Gowers—the loss of power is always much greater than can be accounted for by the degree of wasting present. It is a remarkable and entirely unexplained fact that when this tonic atrophy is present the muscles never completely waste, whereas in flaccid atrophy they waste completely, if the patient survives sufficiently long. When the disease commences with initial flaccid paralysis without wasting, it is usually rapid in its course, any temporary improvements notwithstanding. This initial flaccid paralysis without wasting, especially if it improves temporarily, may give rise to great difficulty in diagnosis, for it generally occurs in one limb only, and its rapid development, and in some cases a conspicuous improvement, may give rise to the impression of a gross organic lesion of the ventral horn or ventral roots, and to hopes of recovery which are falsified later.

The disability which progressive muscular atrophy produces in the limbs is always much more marked when the limbs are cold, and conversely. There may be an appearance of vasomotor paralysis, redness, blueness and some swelling of the periphery, but this seems to occur much more as the result of the continual pendent position of the hands, when the muscles, which

flex the elbow and which raise the shoulder, are affected, than as the result of any definite vasomotor palsy. In the regions where the muscular atrophy is apparent, the fat and subcutaneous tissues also waste slowly and progressively, and in all but the rapidly progressive cases this wasting is conspicuous.

Next in order of frequency to initial wasting in the upper extremities comes the incidence of the disease upon the muscles concerned in facial expression, articulation, mastication and deglutition, and in lesser degree upon the muscles of phonation; and the disease may be confined to these muscles throughout the whole of its course. From the widely different clinical picture resulting, and from the fact that all these muscles are supplied from the brain stem and upper two segments of the spinal cord, this form of the disease has borne the name of "progressive bulbar paralysis," or "labio-glossopharyngeal paralysis." Here the wasting commences in the intrinsic muscles of the tongue and spreads thence to the orbicularis oris, to the extrinsic muscles of the tongue, pharynx and larynx, to the muscles of mastication and, eventually, but in less degree, to the facial muscles generally; but only in rare cases are the oculo-motor muscles affected.

The intrinsic muscles of the palate, the constrictors of the pharynx, the intrinsic muscles of the larynx, and the muscle of the oesophagus are little affected. This seems at first an anomalous and astonishing fact, considering how great and important are the troubles with deglutition in bulbar paralysis. But the anomaly disappears at once when one considers that the muscles which are concerned with buccal deglutition are the muscles of the tongue, those forming the floor of the mouth, including the mylohyoid and the digastric, the muscles which raise and lower the jaw, and those of the lips. Further, the muscles which are most important in pharyngeal deglutition are those which raise and lower the hyoid bone and larynx as a whole, and these are the stylohyoid and stylopharyngeus, the palatoglossus and palatopharyngeus, the geniohyoid, thyrohyoid, sternohyoid, sternothyroid and omohyoid. All these muscles are early and severely affected in bulbar paralysis; and when they fail, the intrinsic muscles of the palate are unable to shut off the naso-pharynx, the constrictors of the pharynx are entirely unable to perform the act of deglutition, and the intrinsic muscles of the larynx—though phonation is never lost—are unable, since the larynx is unfixed by the extrinsic muscles, to modulate the tone of the voice. The very active pharyngeal reflex and the well-known great difficulty in using the laryngoscope on account of spasm of the pharynx in the subjects of this disease, are very good clinical evidence that the pharyngeal constrictors are not affected.

The earliest physical sign of bulbar paralysis is the loss of the finer movements which are essential for correct articulation, and consequently a slurring dysarthria develops and increases, and the consonants become less and less distinct until they are inaudible. The failure of the palate to close upon the posterior pharyngeal wall begets a nasal element in the voice. Later, the patient becomes unable to interrupt his blast at any of the stop positions, and his utterance becomes a long, moaning, monotonous, inarticulate sound. His phonation remains, but he cannot alter its pitch nor divide it into parts of speech, except by taking a fresh breath. The orbicularis oris is early affected, and the lips lose their firmness and become thin,

and as they weaken, the unopposed retractors of the angles produce a wide, straight mouth, both at rest and in emotional action. Whistling and pursing up the lips become impossible, and ultimately there is much dribbling of saliva, for this can neither be retained by the lips nor swallowed. The tongue shows fine fibrillation, and as it wastes its point, becomes rounded, and is protruded with difficulty. Its surface becomes dimpled and faceted, and in the end consists solely of the covering mucous membrane, the glands and the fibrous tissue, and lies motionless in the floor of the mouth, resembling a crinkled mushroom. The muscles of mastication all become affected. The bite becomes feeble and the mouth cannot be opened against resistance. In the late stages the jaw drops and the mouth is constantly open. The combined weakness of tongue and buccinators makes it very difficult for the patient to keep his food between his teeth in mastication, and often he aids his disability by digital pressure upon the cheeks. Nasal regurgitation is not uncommon. The difficulty in swallowing is greatest with fluids, for these require quick action, and is next greatest with lumpy solids, for these necessitate powerful action. It is least with food of a porridge-like consistency, and this should be carefully borne in mind in feeding the patients.

The other muscles of the face are affected later and to a much less severe degree than is the orbicularis oris. It is as if there were a physiological selection on the part of the disease for the nervous mechanism subserving mastication and deglutition. Still in the majority of cases there are bilateral general facial weakness and wasting which, with the peculiar mouth and dropping jaw, produce a characteristic facies which can be instantly recognised. If the upper facial muscles are tested by raising the eyelid with the finger against resistance, invariably they will be found to be weak. Only in very rare cases does the atrophy extend to the oculo-motor muscles. As in the paralysis of the limbs, so also in bulbar paralysis, concomitant signs of both upper motor neurone and of lower motor neurone lesion may exist. When such tonic atrophy of the bulbar muscles is present, the symptomatology and clinical appearance are the same as have been above described for the simple atrophic form, with the exception that the jaw-jerk and the other muscle-jerks of the bulbar region, which are absent in the latter condition, are brisk in the tonic-atrophic form. And, further, it must be remembered that the additional element of spastic paralysis adds greatly to the degree of the paralysis as a whole.

In less common cases of progressive bulbar paralysis the upper motor neurone lesion alone is in evidence, and the bulbar paralysis is purely spastic. Here the symptomatology as regards articulation, deglutition, etc., is the same, and the facial aspect identical with that of the simple atrophic and tonic-atrophic forms. The muscle-jerks are brisk. The appearance of the tongue, however, is quite different; it is smooth, narrow, stiff and drawn into a narrow compass by the spasm of the muscles composing it. It appears too small for so large a mouth. There is no fibrillation, and the muscles are nowhere wasted.

The muscles of the back of the neck, the splenius, complexus, etc., are not uncommonly the first muscles to be affected with the wasting of progressive muscular atrophy. There is increasing difficulty in extending the head, which drops forward, causing a characteristic attitude, which is associ-

ated with a constant overaction of the frontales which raise the brows to clear the line of vision when the head is dropped forward, so giving rise to a permanently furrowed brow. The loss of substance in the muscles of the back of the neck, together with the dropping forward of the head, causes the lower cervical and upper dorsal spines to stand out in undue prominence, and to give an appearance approximating to that of an angular curvature.

Primary affection of the lower extremities is much less common than that of the upper extremities, bulbar region or neck muscles. The anterior tibial and peroneal muscles are usually attacked first, and less commonly the quadriceps. The clinical type is that of flaccid atrophy in most of the cases. Tonic atrophy, which is so common in the upper limbs and in the bulbar region, is rare in the legs. Spasticity without atrophy from the upper motor neurone lesion alone is very common in the lower extremities. It forms a characteristic part of the frequently occurring clinical type of amyotrophic lateral sclerosis, in which the upper extremities or bulbar region are affected with atrophic paralysis, and the legs with spastic paralysis. In this common combination the atrophic paralysis is usually of the tonic and much less frequently of the simple flaccid type. Spasticity from the upper motor neurone lesion may develop in the lower extremities long before there are any signs of atrophic paralysis elsewhere from the lower motor neurone lesion, and such cases present the physical signs of a primary lateral sclerosis. Therefore, it cannot be too strongly borne in mind that any case presenting the features of a primary lateral sclerosis in an adult may eventually prove to be one of progressive muscular atrophy.

Wherever the site of commencement of progressive muscular atrophy may be, it invariably spreads to other regions, sometimes slowly and with periods of arrest which may last for years, sometimes with remarkable rapidity. The manner of spread is usually in terms of the contiguity of the affected elements in the nervous system; but it is sometimes in terms of the physiological association of the muscles, as is commonly seen in the bulbar forms of the malady. When the disease is definitely installed the appearance of fibrillation, in any muscles otherwise unaffected, is a sure sign that atrophy will shortly commence in those muscles.

According to the method of advance shown by the disease, cases of progressive muscular atrophy fall into two groups which it is important to distinguish. In the first group, the atrophy spreads locally and slowly and remains confined to one region of the anatomy during most of the course of the malady. These cases are always of the simple atrophic type, and they usually survive a long time. Such cases, however, tend to become general just before the end. In contrast with the local type of the affection is the group in which the manifestations, commencing locally, spread within a comparatively short time to many parts of the anatomy, or even become universal. The spread may be very rapid, and the end may occur in a few months, or it may be slower; but it is unusual for any of the cases forming this group to survive for more than eighteen months. This group comprises (1) the generalised cases of simple flaccid atrophy; (2) all the cases of amyotrophic lateral sclerosis; and (3) most of the bulbar cases.

Fibrillation is a most important symptom of the disease, and is an associate of the muscular atrophy. It precedes the wasting of the fibres, and is a sure

herald of the advent of wasting in this disease. It ceases to occur when the muscle is completely wasted, and is not seen when the atrophy is not progressing. On account of the importance of fibrillation as a diagnostic sign of progressive muscular atrophy it is important here to consider those other conditions in which it is met with clinically. It occurs in syringomyelia and in peroneal atrophy, but only when the muscular atrophy is progressing; and, therefore, it is only an occasional symptom in either disease. It is often very marked in cases of interstitial neuritis (sciatica, etc.). It occurs in a most magnified and conspicuous form in certain conditions of gastro-enteritis, and is presumably due to an intoxication, and to this form of fibrillation the term "myokimia" has been applied. It is not met with in polyneuritis, poliomyelitis, myopathy, nor in the common gross lesions of nerve trunks, nerve roots or spinal cord.

The *electrical reactions* of the affected muscles vary according to the degree of degeneration. Since normal and degenerate fibres are stimulated side by side in the affected muscle, there will be some lowering of the response to faradism with a tendency to a polar change. This is known as the "mixed reaction," and it is common to all diseases in which muscle degenerates fibre by fibre. Faradic excitability lessens as more of the muscle fibres degenerate, and when degeneration is complete all electrical excitability is lost. The excitability of the affected muscles to direct mechanical stimuli, such as percussion, is increased so long as any living muscle remains.

Contractures are conspicuous by their absence in this disease, which is thus strongly contrasted with peroneal atrophy and some other muscular atrophies. If the atrophy becomes complete in a whole limb the end-result is that the limb is flail-like and without contracture.

Mental alterations are constantly present in the cases in which the bulbar region is affected. Emotional instability and hyperexcitability are the usual change. The patient is easily excited to tears or to laughter by trivial causes, and when so excited cannot control his expression of emotion. He himself feels little joy or grief during the paroxysms of laughing or crying.

Sphincters.—In the majority of the cases these are not affected, but every now and then dysuria in any of its forms occurs, and it may occur early in the course of the malady, and it may be severe. Loss of sexual power is very common.

Reflexes.—The superficial reflexes are modified in this disease, on the one hand by spasticity, when this is present, and, on the other, by the muscular atrophy which may prevent response in the affected muscles. The pharyngeal reflex in bulbar cases is usually brisk, notwithstanding the statement to the contrary, which most antecedent writers upon this subject have recorded; but the response is not the normal response, involving all the muscles concerned in deglutition, for these are atrophied and paralysed; it is confined to the constrictors of the pharynx and the muscles of the palate, with the feeble co-operation of such of the somatic bulbar muscles as are still able to act. The plantar reflexes are usually of the extensor type when the legs are spastic; but this does not always obtain, for there may be definite rigidity of the legs with brisk knee-jerks and foot-clonus with a persistent flexor response. Similarly, the abdominal reflexes do not disappear so constantly or so early as is the case in disseminated sclerosis, for example, and they may persist when the legs are markedly spastic and extensor plantar

responses have appeared. The muscle-jerks disappear from the affected region in simple atrophic cases *pari passu* with the wasting of the muscles. In cases of tonic atrophy they are everywhere increased, even in regions where the atrophy is severe, and in this type of the malady they never disappear. The same increase of the muscle-jerks occurs in the purely spastic cases.

Diagnosis.—The malady has to be distinguished from the many conditions in which progressive weakness and wasting of the muscles occur, from those in which muscular wasting and spasticity are conspicuous clinical features, and lastly from other diseases, in which bulbar symptoms are early evidenced. Peroneal muscular atrophy very closely resembles progressive muscular atrophy, in that slow wasting and fibrillation of the muscles are the chief clinical features. The points which distinguish the two conditions are that peroneal atrophy is often a familial disease, and is apt to commence in childhood, when it is unusual for progressive muscular atrophy to begin. The location of the atrophy is peculiar, and when well marked in the periphery of all four limbs, as is common in this disease, cannot be confused with progressive muscular atrophy since the latter disease never has this distribution. Syringomyelia is easily distinguishable by the early and striking loss of pain and temperature sensibility. Cervical rib not uncommonly produces atrophy of the intrinsic muscles of the hand, and, though this is usually confined to one hand, it may be bilateral. Further, it is exceptional for the atrophy to involve all the small hand muscles simultaneously, or equally. It picks out the *opponens pollicis* first and most severely, and is not uniform for all the hand muscles, as in progressive muscular atrophy. Pain in the distribution of the eighth cervical and first dorsal roots, and some loss of sensibility, may be present. The atrophy remains local, and is never accompanied by fibrillation. The abnormal rib is easily discoverable on radiographic examination. It must be borne in mind that cervical ribs are not uncommon, and that their presence does not necessarily prove the cause of atrophy of the hand muscles, for cervical ribs may be present in progressive muscular atrophy, in syringomyelia, and in any other disease.

Arthritic muscular atrophy occurs in the regions of joints which show easily recognisable disease. Fibrillation does not occur, nor are there alterations in the electrical excitability of the wasted muscles. Dystrophia myotonica is at once separated from progressive muscular atrophy by the myotonus, when this latter symptom is present. When myotonus is absent, the characteristic wasting of the sternomastoids, and of the muscles of the thighs, the age of the subject, and sometimes the presence of cataract should suggest the diagnosis.

Lesions of peripheral nerve trunks may be diagnosed by the history of a local cause, by the discovery of a palpable local lesion upon the course of the nerve, and by the confinement of the atrophy to the distribution of one particular nerve, while open pain and sensory loss occur in that same distribution.

Lesions of the nerve roots, and especially those produced by pachymeningitis and by neoplasm in the vertebræ may cause signs and symptoms so closely resembling those of the more rapid forms of progressive muscular atrophy, as to render correct diagnosis very difficult. Such a lesion in the cervical region, for example, may give rise to wasting of the hand and fore-

arm muscles, and a spastic condition of the legs, resembling exactly a condition of amyotrophic lateral sclerosis, without deformity or rigidity of the spine, and without pain or sensory loss. In such cases of difficulty the course of a little time will bring the advent of the conclusive symptoms of a local pressure lesion. It is important in this connection to remember that pressure upon the spinal cord results in hyperalbuminosis of the cerebro-spinal fluid, and if the lesion causing the pressure is syphilitic, there is likely also to be lymphocytosis in that fluid, neither of which conditions is found in progressive muscular atrophy.

Diagnosis is most difficult in those cases where spasticity in the limbs is the first sign of progressive muscular atrophy to appear, and where such spasticity precedes the appearance of any muscular atrophy by a long time. If it be clearly borne in mind that spastic paralysis may be the earliest, and for a time the only sign of progressive muscular atrophy, and that among the many diseases of the nervous system, which commence with the same clinical picture of spastic paralysis, a certain diagnosis cannot be made until further distinguishing signs appear, error will be avoided. The importance of the examination of the cerebro-spinal fluid in doubtful cases cannot be too strongly emphasised.

Course and Prognosis.—The nature of the disease is to progress, and to extend its area of invasion until a fatal issue is reached. The progress may be rapid, and the end may be reached in a few months, or it may be slow, and many years may elapse before death occurs. The local types of slow onset are the most gradual in their development, and these are often characterised by periods of arrest in the progress of the disease. The generalised simple atrophic type of the disease is the most rapid, especially when it commences with severe initial flaccid paralysis without atrophy.

In the bulbar types of the disease, and in amyotrophic lateral sclerosis, the course is for the most steadily progressive. Every type will show, however, upon occasion, exacerbations and remissions, and the exacerbations are the most important, and in the bulbar types may bring about the end in a few hours. Of particular interest are rapid extensions of a flaccid paralysis, which may occur in a few hours, and which resemble, and indeed are identical with, onset of the disease with initial flaccid paralysis without atrophy, which has been already described. Whatever type of the disease be present, it tends in the end to spread and to become general.

Involvement of the respiratory muscles or severe bulbar symptoms, and the pulmonary complications which may accompany either condition, may bring about the fatal issue. It is usual, however, for death to occur in a manner which is common to so many degenerative nervous diseases, a rapid increase of the paralysis is associated with an increasing lethargy, which soon deepens into a rapidly fatal coma. It is uncommon for death to occur from intercurrent maladies. The average tenure of existence after definite signs are present is under 1 year in the generalised flaccid type, and it may be as short as 2 months. Bulbar symptoms are not generally survived for more than 12 months. Localised cases of simple atrophy may live for many years. Some of the patients in whom a positive Wassermann reaction is found improve, and the disease is sometimes arrested by antisiphilitic treatment.

The progressive character of the disease renders the prognosis grave in

every case. There are some cases occurring in middle life, which are presumably cases of progressive muscular atrophy of local distribution and slow onset and course, which become finally arrested or even improve ; but in the absence of pathological verification the true nature of such cases is open to doubt.

In amyotrophic lateral sclerosis the average duration of life is not more than 2 years from the onset. When bulbar symptoms are present the average duration is under 2 years. In the generalised cases the average duration is under 1 year. Widely spread fibrillation in muscles, which are neither weak nor wasted, is the constant herald of generalisation, and renders the immediate prognosis serious. In cases where syphilis is present the prognosis is more favourable, and there is even a possibility of arrest and improvement if energetic treatment of the associated condition is provided. Rapid extension of the weakness, the advent of bulbar symptoms, involvement of all the respiratory muscles, and especially general asthenia and drowsiness are the signs which usher in the fatal result.

Complications.—By far the most common complication which is met with in cases of progressive muscular atrophy is the presence of some syphilitic lesion of the nervous system, and this may be of any nature, both local or general. *Tabes dorsalis*, associated with progressive muscular atrophy, is not uncommon. General paralysis of the insane has been noted in a few cases, as has also paralysis agitans.

Treatment.—For the most this malady seems to be entirely uninfluenced by any treatment that has hitherto been adopted. Even where syphilis is a factor in the causation, although appropriate treatment for these conditions has been applied, and improvement and even arrest may result, it is no rare thing to see no amelioration, and in some cases such treatment seems actually to hasten the progress of the disease. Recently, dramatic claims have been made in respect of vitamin E (tocopherol acetate), given in doses of 3 mgs. thrice daily. This is said to arrest wasting and weakness and in early cases to effect rapid improvement. In a brief experience since this claim was made the present writer has been wholly unable to confirm it, and it must be accepted with the greatest reserve. It remains, therefore, to secure favourable conditions of life for the patient, and to maintain the general health in as perfect a state as possible. Massage and passive movements are useful as giving bodily comfort to the patient, and satisfying him that something is being done for him. In bulbar cases, the dysphagia must be aided by avoiding liquids and solids, and by serving all the articles of diet in pulvaceous form. Salivation, which is so troublesome in this condition, may be greatly helped by the administration of hyoscine by the mouth.

PERONEAL MUSCULAR ATROPHY

Synonym.—Charcot-Marie-Tooth Type of Muscular Atrophy, Neuritic Type of Muscular Atrophy.

This is an absolutely distinct and peculiar form of muscular atrophy, with a frequent tendency to occur in several members of the same family. It usually commences in mid-childhood, and after progressing for some twenty years or less, comes to a final arrest. The atrophy always commences

in the intrinsic muscles of the feet, and is throughout strictly distal in distribution. The muscles of the face and trunk and the proximal muscles of the limbs are never affected. The atrophy leaves a peculiar elastic fibrosis in the affected muscles, so that the incapacity caused by this disease is much less than in any other form of muscular atrophy of like degree. Sensibility is often slightly affected, and there may be deep sensory loss. The essential morbid anatomy is a primary neurone atrophy of the anterior horn cells and of some of the afferent neurones in certain regions of the spinal cord.

Ætiology.—The disease usually commences between the fifth and tenth years of childhood, but it may appear as late as the fourth decade of life. Males and females are both affected. Heredity plays an important part in the incidence, although isolated sporadic cases are not uncommon. It may exhibit every type of inheritance. The malady often occurs in families, and has been traced through five generations; it may skip a generation and then reappear.

Pathology.—The anterior horn cells of the affected regions show a slowly progressive atrophy and disappearance, with corresponding atrophy of fibres in the peripheral nerves. The cells of Clarke's column show signs of degeneration, as do also some of the fibres of the posterior columns of the spinal cord, and especially those of the postero-lateral column. Slight degeneration in some of the fibres of the pyramidal tracts is usually found. The affected muscles show a simple atrophy of the muscle fibres, indistinguishable from that seen when a motor nerve is divided. There is a simple shrinking of the fibres, which stain progressively and more and more deeply with hæmatoxylin, lose their striation, and finally disappear. Secondary fibrotic changes accompany the atrophy, together with sclerosis of the arteries of the muscle.

Symptoms.—Muscular atrophy dominates the clinical picture of this malady. It is strictly distal in distribution, and this feature will serve to distinguish peroneal atrophy from any other form of muscular atrophy. This is to say it does not affect one particular muscle, but the distal ends of all the muscles below a certain level on the limb, leaving the proximal ends of the muscles normal, and it advances up the limb inch by inch, the separation of the wasted portion of the muscle from the normal portion being always transverse to its length. In other words, the muscle fibres seem to waste in terms of the length of the spinal axons which supply them. The wasting commences always in the intrinsic muscles of the feet, and hollowness of the instep and thinness of the feet, together with retraction of the toes and the difficulty which the pes cavus so produced entails in fitting boots, first draws attention to the disease. As the process advances, the lower segments of the anterior tibial, peroneal and calf muscles become affected, and the limb is subsequently involved until the lower third of the thigh is reached, at which stage the disease is invariably arrested. This slow spread of the atrophy from the distal towards the proximal portion of the limb, gives rise to a most unique and characteristic feature in the appearance of the legs at the several stages of the disease. As an example, the complete atrophy of all the muscles below the middle and a well-developed musculature in the upper half of the leg, give rise to the inverted "fat bottle" calf. When the atrophy has involved the lower third of the thigh, the lower end of the femur, bare of muscle and covered only by skin and tendons,

contrasts strongly with the well-developed muscles of the upper thigh, and causes the thigh to resemble an inverted champagne bottle.

Some years after the atrophy has become marked in the lower extremities, and in the usual run of cases just before the age of puberty, the intrinsic muscles of the hands and first those of the thenar and hypothenar group begin to waste, and this wasting may extend as high as the middle of the forearm. It must be borne in mind that the disease may become arrested at any period of its spread, and especially that the upper extremities often escape altogether. With the exception of the lower part of the thighs, the proximal segments of the limbs do not become involved, and the muscles of the head, neck and trunk remain unaffected.

The affected regions of the muscles waste absolutely, and leave a very elastic fibrous tissue. The electrical excitability in the wasted regions becomes first lowered and then lost, and, in the earlier stages, may show a mixed reaction, in which there is lowering of excitability to faradism, with a tendency to an inverted polar reaction. Fibrillation of the muscles is an important sign. It is seen only when the disease is progressing, and in the muscles which are obviously wasting. It is never general, as in some cases of progressive muscular atrophy. And since peroneal atrophy is at times advancing and at other times stationary, fibrillation may be in one case conspicuous and in another never seen. It disappears entirely when the progress of the malady becomes finally arrested, and is, therefore, useful as a clinical indication of active advance of the disease. Contractures always occur, and from the nature of the distribution of the atrophy are necessarily confined to the feet and the hands. In the feet, pes cavus with retracted toes is the rule; but sometimes, and in some stages of the disease, the feet and toes may be dropped and the feet inverted. The sphincters are unaffected. The ankle-jerks are diminished or lost in proportion to the wasting of the calf muscles. In the final arrested stage they are usually lost. The knee-jerk is always retained and is usually brisk. The plantar reflexes are usually lost early so far as any response in the foot is concerned, but some response in the upper thigh muscles, upon stimulating the plantar region, often remains. Pain, tenderness and cramp are entirely absent. Conspicuous loss of sensibility is uncommon, but slight loss of deep sensibility, loss of the vibration sense and relative tactile loss, may often be detected upon careful examination: but in rarer cases all forms of sensibility may be severely affected, or even entirely lost. Perforating ulcers may be met with upon the soles of the feet, and are explained by the thinness of the feet and their deformity, which, coupled with the clumsiness of the use of the feet, lead to the formation of severe corns which break down into perforating ulcers. Loss of sensibility also is a factor in their production.

The most striking of all the clinical features of peroneal atrophy is the comparatively slight disability which the wasting of the muscles and consequent paralysis, and even the sensory loss, when present, cause.

Course.—The course is irregularly progressive for a number of years only, and the advance of the disease ceases usually in the third decade of life. Exacerbations of the weakness are likely to be followed in every case by considerable improvement, owing to the secondary fibrosis in the muscles.

Diagnosis.—Peroneal atrophy in the early stages is easily confused with progressive muscular atrophy, in that wasting of muscles and fibrillation

are the conspicuous features. The onset usually in childhood and the fact that the feet are affected first, the peculiar distal distribution and the presence of any familial incidence, are important. But the only distinction which is absolute is the distribution, for progressive muscular atrophy may begin in childhood and peroneal atrophy may not appear till after middle life, and often familial relations are absent in the latter malady. In the course of time the diagnosis always becomes clear, for progressive muscular atrophy never keeps to the classic distribution, nor is it followed by the peculiar fibrosis which characterises peroneal atrophy.

Dystrophia myotonica when commencing in the peroneal muscles may for a time closely simulate peroneal atrophy. The presence of the least sign of myotonia, the involvement of the face and the atrophy of the sterno-mastoids, will establish the diagnosis.

The usual forms of myopathy are at once separated from peroneal atrophy by the distribution of the muscular weakness and wasting, which in the former group of maladies is conspicuously upon the face, trunk and proximal muscles of the limbs, and in the latter upon the distal muscles. Peripheral neuritis is more rapid in its onset, and is apt to be associated with marked sensory disturbances, both objective and subjective, and the paralysis is in terms of individual muscles, which is not the case in peroneal atrophy.

Treatment.—The general health should be carefully maintained, and the nutrition of the affected muscles aided by the application of massage. Care must be taken, on the one hand, to avoid over-fatigue of the affected muscles, and, on the other, to ensure such regular exercise as is compatible with their capacity. Bicycling, for example, since it employs chiefly the thigh muscles, is a better form of exercise for these patients than is walking. In no circumstances should tenotomies be performed for the deformity of the feet, for such measures tend to destroy the effect of the conservative fibrosis, so essential to the production of a useful limb. The use of heavy mechanical supports is to be avoided above all things. Light, well-fitting boots, so as to interfere as little as possible with the exercise of the damaged muscles, are essential.

PROGRESSIVE SPINAL MUSCULAR ATROPHY OF CHILDREN

Synonym.—The Werdnig-Hoffmann Disease.

This is a malady of the first year of infancy, often incident upon several children of the same parents, and characterised by the gradual development of progressive muscular weakness and atrophy, which affects the proximal muscles first and most, increases to a complete paralysis of trunk and limbs, and finally affects the bulbar muscles. The disease is invariably fatal in from a few weeks to several months. The most striking pathological changes are a progressive degeneration and disappearance of the ventral horn cells of the spinal cord, and of their analogues in the brain stem.

Ætiology.—In some of the cases the paralysis is noticeable at the time of birth, and the disease is obviously of pre-natal development. In others the children are quite healthy at birth, and the disease develops some time during the first year of life, and most frequently within 8 weeks of birth. Though sporadic cases may be met with, yet in the majority of instances

several children of the same mother are affected. Both the pre-natal cases and the post-natal cases may be met with among the children of the same mother. The sexes seem to be equally affected. No maternal ill-health during pregnancy has been noticed, and nothing is known about any other aetiological factor.

Pathology.—The most extensive changes are found in the ventral horn cells throughout the spinal cord and brain stem, and at many levels no normal cells whatever are to be seen. Tigrolysis, swelling and glassiness of the cells, extrusion of the nuclei, disappearance of the dendrites, shrinking of the cells and final disappearance is the sequence of the changes. Degeneration of the anterior roots and of the peripheral motor nerve fibres consequently occurs. These changes are not confined to the lower motor neurones, for in our cases examination by the Marchi method showed extensive degeneration throughout the posterior columns of the cord, indicating that lower sensory neurones were also considerably affected.

The muscles show intense degeneration with hypertrophy of some fibres and atrophy of most of the fibres, waving, moniliform shape, hypernucleation of the spindles, general nuclear increase and fibrosis.

Symptoms.—In the cases which are pre-natal, the malady is noticed at the time of birth on account of the tonelessness, flaccidity and the pooriness of movement in the trunk and proximal muscles of the limbs. In the post-natal cases there is a gradual onset of similar weakness and flaccidity in the trunk first, and in the limbs afterwards, which usually commences within six weeks of birth, but which may not appear until towards the end of the first year of life. The weakness seems always to be least marked in the periphery of the limbs, where curious, slow, involuntary movements of the fingers and toes have been noted in a good many of the cases. The paralysis is followed by a rapid and extensive wasting of the muscles, accompanied by occasional fibrillary twitchings. Since these children are not only well nourished, but often put on much fat during the illness, wasting of the muscles may not be apparent on inspection or palpation. It can, however, immediately be detected by radiography, which distinguishes sharply between fat and muscle.

As the malady progresses the trunk muscles become completely paralysed, the intercostal muscles being always paralysed before the diaphragm. The limbs become progressively weaker, and, lastly, bulbar paralysis supervenes in those cases where death has not already occurred from respiratory paralysis. The reaction of degeneration is present in the affected muscles. Sensibility may be unimpaired; but in several of my cases there has been conspicuous loss of pain sensibility over the limbs and trunk. The sphincters are unimpaired until the very last stages of the disease. The superficial and deep reflexes are lost. The ocular muscles have not been affected, and intelligence is preserved throughout.

Diagnosis.—The peculiar and striking features of the disease make the diagnosis easy, if the symptomatology be known. Amyotonia congenita presents the same helplessness and flaccidity of trunk and limbs as does the Werdnig-Hoffmann disease, and further resembles it in being sometimes congenital, and sometimes having an onset very early in life. In amyotonia congenita, however, the paralysis is not complete, and it tends to improvement and not to progressive increase. Contractures also occur, which are

not found in the Werdnig-Hoffmann disease, and, lastly, the definite spinal cord changes of the latter malady are not found in the former.

Course and Prognosis—The course is invariably progressive, and is more rapid the earlier in life the disease commences, and it is most rapid of all in the pre-natal cases, which are usually fatal within a few weeks. With an onset some weeks after birth, life is usually continued for several months, and a few cases have been reported with an onset towards the end of the first year, in which death has been delayed until the third or fourth year.

Treatment.—No treatment is known to influence the course of the malady.

LESIONS OF THE PERIPHERAL NERVES

LOCAL LESIONS OF NERVE ROOTS AND NERVE TRUNKS

PHRENIC NERVE.—This nerve supplies the diaphragm. Paralysis results most often from disease of the spinal cord, but the roots may be implicated in disease of the spine, and the trunk may be injured, in its course through the neck and thorax, by wounds or tumours. Bilateral paralysis occurs in lesions of the cord and spine, and in alcoholic, diphtheritic, saturnine and other forms of peripheral neuritis. Other causes usually affect one side only. When the diaphragm is completely paralysed, the normal inspiratory protrusion of the upper part of the abdomen disappears, or is replaced by retraction of this part with each inspiration. During rest, so long as the lungs are healthy, the respiratory rate does not increase, but if bronchitis or pneumonia arises as a complication, or if the patient exerts himself, the diminished reserve of respiratory power is seriously felt. When one nerve only is affected the diaphragm does not descend on that side. This is rarely detected by observation of the abdominal movements, but is easily seen on the X-ray screen. It produces no discomfort.

THE LONG THORACIC NERVE.—This nerve supplies the serratus magnus muscle. When all the fibres of this muscle contract, the scapula moves upwards, forwards and outwards. It contracts with the pectoralis major in the action of pushing forward the point of the shoulder and in the rapid-thrust movement. It also assists the deltoid in raising the arm. When it is paralysed alone, the position of the scapula at rest is unaltered, but if the trapezius and the rhomboids are paralysed as well the scapula drops, and its lower angle is displaced inwards. Paralysis of the serratus magnus is best demonstrated by causing the patient to hold the arms outstretched before him. The arm is not raised so high on the affected as on the normal side, because the scapula is not fixed and the deltoid works at a disadvantage. Viewed from behind the deformity is characteristic. The vertebral border of the scapula stands out prominently and the hand can be pushed between this bone and the thorax—"winged scapula." On raising the arm from the side, there is difficulty in attaining the horizontal position, but the winging of the scapula is less apparent.

The nerve may be damaged by carrying heavy weights on the shoulder, by falls or blows on the shoulder, and by continued muscular effort with the

raised arm. The nerve may be injured alone in gunshot wounds, but as a rule it is associated with lesion of the brachial plexus. In addition, a serratus magnus palsy may develop suddenly in an otherwise healthy person after exposure to cold, or as part of a rare reaction to the administration of serum or antitoxin. In the cases caused by compression, severe neuralgic pains in the neck precede the onset of paralysis. Recovery is always very slow and the defect may be permanent.

BRACHIAL PLEXUS.—The brachial plexus may be injured by stabs in the neck, by penetrating missiles, by dislocation of the shoulder or fracture of the clavicle, or by pressure of a tumour, aneurysm or cervical rib. Further, the nerves may be torn by forcible dragging on the arm in accidents or during delivery. In most cases the lesion is partial and the symptoms conform in the main to one of the following types.

Upper plexus paralysis (Erb's palsy).—This results from an injury to the fifth and sixth cervical roots. The muscles paralysed are: biceps, deltoid, brachialis anticus, supinator longus, supraspinatus, infraspinatus, rhomboideus, subscapularis, clavicular portion of pectoralis major, serratus magnus, latissimus dorsi, teres major. The arm cannot be flexed at the elbow (flexors of forearm), nor raised and abducted (deltoid). The movements of the wrist and fingers are not impaired. Adduction of the arm is weak (pectoralis major), and rotation is feeble or absent (spinati). On attempting to oppose the shoulders, the scapula on the affected side passes farther from the middle line (rhomboideus). The hand of the affected side cannot be placed on the buttock of the sound side (latissimus dorsi).

The reaction of degeneration is often complete in the deltoid and flexors of the forearm and nearly so in the spinati. It is usually incomplete in the other muscles. Sensation is diminished or lost along the outer border of the whole limb immediately after the injury, but improvement sets in rapidly. For some time the patient experiences pins and needles and burning sensations in the affected area, which last longest in the thumb and index finger. The biceps reflex is lost. In this form the tendency to complete recovery is great. As a rule all the symptoms disappear completely in from 6 months to 2 or 3 years. Weakness persists longest in the deltoid and supinator longus.

Lower plexus paralysis (Klumpke's palsy).—This results from a lesion of the eighth cervical and first dorsal roots, or of the common trunk of the median and ulnar nerves. The intrinsic muscles of the hand and the flexors of the wrist and fingers are paralysed, and the inner border of the forearm and hand is anæsthetic. When the roots are damaged, sympathetic fibres may be implicated with the production of myosis, narrowing of the palpebral aperture, enophthalmos and alterations in sweating on the face, neck, arm and upper part of the chest, on the affected side.

Middle plexus paralysis.—This form of paralysis is a common result of gunshot injuries of the plexus. It affects the muscles supplied by the musculospiral and circumflex nerves—posterior cord. As the nerve to the latissimus dorsi arises from the same trunk, this muscle is often paralysed as well. In addition to these simple types, more complicated paralyses occur, in which various parts of the plexus are injured together.

In *paralysis of the inner cord of the plexus*, atrophy is confined to the intrinsic hand muscles, and the sensory loss is confined to the hand.

Lesions of the brachial plexus show a remarkable tendency to spon-

aneous recovery. In many cases recovery is complete in 6 months to 2 years, in others it is partial, and some muscles remain paralysed.

THE MUSCULO-SPIRAL NERVE.—Owing to its long course, its position in relation to the humerus, and its peculiar vulnerability to compression, paralysis of the musculo-spiral nerve is one of the commonest peripheral palsies; although it is a mixed nerve, containing sensory, motor and vaso-motor fibres, the symptoms of an injury are almost entirely motor. In the upper arm the nerve supplies the triceps and the anconeus, in the forearm the supinators, the extensors of the wrist and fingers, and the extensors and long abductor of the thumb.

Symptoms.—Injury to the nerve is followed by dropping of the wrist and fingers. The wrist and the first phalanges are flexed. The flexion is limp and easily reducible.

When the lesion is in the axilla the whole of the *triceps* is paralysed, and extension at the elbow is lost. Occasionally in wounds of the posterior aspect of the arm the nerves to the triceps are injured, whilst the main trunk escapes. The patient is then able to extend the arm powerfully by means of the anconeus, but if he is made to raise the elbow as high as possible with his fingers on the point of the shoulder, extension of the bent forearm is impossible.

In most cases the nerve is injured in the middle third of the arm and the triceps escapes, but the supinator longus and all the extensor muscles in the forearm are paralysed. Partial paralyses, such as are seen in lesions of the median and ulnar nerves, are very rare. The *supinator longus*, so-called, is not a supinator. Its action is to flex the forearm, whilst the hand is in a position intermediate between pronation and supination. Paralysis of this muscle is detected by the absence of contraction when the pronated forearm is flexed against resistance. Owing to paralysis of the *supinator brevis* supination is abolished. During the movement of flexion of the forearm the biceps acts as a supinator, during extension the external rotators of the shoulder, but feebly.

Paralysis of the *extensors of the carpus* abolishes both extension and lateral movement at the wrist. The flexors of the carpus play no part in lateral movements. The *extensors of the fingers* extend the first phalanges only. Extension at the distal joints is carried out by the lumbricals and interossei. Paralysis of the *extensors and long abductor of the thumb* renders abduction of the thumb and extension of the phalanges impossible. On attempting to abduct the thumb, it passes no farther than the radial border of the hand. In some cases, the second phalanx of the thumb can be feebly extended by the muscles of the thenar eminence.

Many muscles not supplied by the musculo-spiral work at a disadvantage when the extensors are paralysed. These defects must not be mistaken for signs of injury to other nerves. Owing to the flexed position of the hand the grasp is feeble, but if the wrist is extended passively the grasp is improved. The patient cannot make a fist properly, as the thumb does not oppose the index finger and the fingers cannot be flexed into the palm, until the thumb has been moved aside by the sound hand. The movements of the interossei in abducting and adducting the fingers are also feeble while the wrist is flexed, but are much stronger when the hand is resting flat on a table with the wrist and fingers extended. The complete reaction of degeneration is

often found in all the paralysed muscles from the onset. Atrophy becomes obvious in a month or two. Its extent and severity give important evidence for prognosis.

Sensory disturbances.—Subjective symptoms are rare. In a few cases, paræsthesiæ are felt on the posterior aspect of the forearm and on the dorsal aspect of the thumb. They are of brief duration, and are commoner with partial than with complete lesions. Severe causalgias are almost never seen in lesions of this nerve. Sensibility to light touch, superficial pain and temperature is impaired over a small area on the radial border of the hand, including the proximal joints of the thumb and first two fingers. The defect is often very slight, and is only discovered on very careful examination. Deep sensibility is rarely affected. Considering the extensive distribution of the external cutaneous branch of the musculo-spiral nerve, it is rather surprising that the sensory disturbances are so slight, when the nerve is injured above the origin of this branch.

Recovery.—It might be thought that recovery would take place in the order of the length of the branches to the various muscles. This, however, is not the case. As a rule the extensors of the wrist recover first, then the extensors of the middle, ring, little and index fingers in this order, next the supinator longus, and the extensors and abductors of the thumb last of all. On palpation of the muscles during attempted extension, contractions can be felt before any movement is produced. Other signs of impending recovery are the disappearance of automatic pronation and of the flail-like drop of the hand, also diminution of automatic flexion of the fingers after passive extension. Recovery of movement is complete when the patient is able to extend the wrist and all the fingers simultaneously or separately. After this becomes possible, restoration of power is rapid.

THE MEDIAN NERVE.—Whilst the clinical individuality of the musculo-spiral nerve is shown in the preponderance of motor symptoms and in the uniform completeness of the paralysis that follows an injury, that of the median is seen in the frequency of partial and especially of painful lesions. Isolated palsy of this nerve is infrequent except as a result of gunshot wounds and other injuries. It may be damaged by repeated violent contractions of the pronator radii teres, as in one of the forms of "tennis elbow."

Total paralysis.—The muscles paralysed are the pronators, the radial flexor of the wrist, the flexors of the fingers except the ulnar half of the deep flexor, most of the muscles of the thenar eminence (opponens, abductor brevis and outer head of the flexor brevis pollicis) and the two radial lumbricals. Stated briefly the symptoms are : inability to flex the phalanges of the index finger and the second phalanx of the thumb ; difficulty in flexing the phalanges of the middle finger ; defective opposition of the thumb. The appearance of the hand in total lesions is fairly constant. The hand inclines to the ulnar side, the index and middle fingers are more extended than is normal, and the thumb lies on a level with the fingers—the ape-hand.

Pronation is incomplete and defective. The patient tries to overcome the defect by rotating the whole limb at the shoulder. Paralysis of the *flexors of the wrist* is seen when an attempt is made to flex against resistance. The tendon of the ulnar flexor alone stands out, and the hand is drawn towards the ulnar side. Even at rest, the flexor tendons are more prominent on the sound than on the affected side.

Flexion of the fingers is good in the two ulnar fingers, though weaker than normal. The index cannot be flexed at all, and the third finger only incompletely. Flexion at the proximal joint is usually good in all the fingers including the index, and flexion at this joint with extension at the last two joints is usually well done by the interossei and lumbricals. If the proximal phalanx of the thumb is immobilised, it will be seen that flexion of the terminal phalanx is abolished, owing to paralysis of the *flexor longus pollicis*.

Paralysis of the *thenar muscles* renders opposition and abduction of the thumb defective. By means of the adductor the thumb can be drawn into the palm, but as the radial fingers cannot be flexed nor the thumb opposed, it is impossible to place the tip of the thumb on the tips of the fingers. Atrophy of the muscles becomes obvious in a few weeks. The outer part of the thenar eminence is flattened, and the bulk of the muscles arising from the internal condyle is greatly diminished.

Sensory disturbances.—In almost every case there is complete anæsthesia to all forms of sensation in the two terminal phalanges of the index and middle fingers. The skin outside this area may be unaffected even in complete lesions, but in most cases sensibility is diminished in the terminal phalanx of the thumb, and to a less extent over the remainder of the radial half of the palm, including the radial side of the ring finger. The stereognostic sense is lost in the outer fingers. This defect, together with the loss of power, renders the thumb and index finger useless, and makes paralysis of the median the most serious single nerve lesion of the upper limb.

Vasomotor and trophic changes.—In many cases the skin in the distribution of the median nerve is red, dry and chapped, and the nails white or purple. It is possible that these changes are due to an associated vascular lesion.

Recovery is extremely slow and is rarely complete. Sensation begins to return before power, but the stereognostic sense is often defective, long after movement in the fingers has returned. The pronator and the flexors of the wrist recover first, then the flexors of the thumb and middle finger. Flexion of the index finger and opposition of the thumb, if it is regained at all, remains defective for several years. In searching for signs of recovery, care must be taken lest some "trick-movement," due to contractions of healthy muscles, is misconstrued. For example, when told to flex the terminal phalanx of the thumb, the patient first over-extends and abducts, and then relaxes suddenly. The terminal phalanx then makes a slight passive movement of flexion, which may be mistaken for true active flexion. Recovery is complete when the patient is able to make a good fist with the fingers flexed well into the palm, and the thumb pressed firmly upon the dorsal aspect of the second phalanx of the middle finger.

PARTIAL LESIONS.—Partial paralysis of the median nerve is much commoner than the complete form.

Motor symptoms.—Flexion of the index finger and opposition of the thumb are most impaired. The flexors of the middle finger and of the terminal phalanx of the thumb may suffer also, but to a less degree, whilst the pronators and the flexors of the wrist often escape entirely.

Sensory symptoms.—Apart from the painful lesions to be mentioned later, sensory troubles are usually slight in partial lesions. Anæsthesia is rare, but sensibility to all forms may be diminished in the areas mentioned under complete lesions.

Vasomotor symptoms.—The skin is often cyanosed in the distribution of the injured nerve, and it may perspire more freely than in healthy parts. These changes are more distinct when the paralysis is complicated by a vascular lesion.

Recovery is naturally more rapid than in complete lesions. The order in which the muscles recover and the tests for complete return of function have been mentioned above.

PAINFUL LESIONS OF THE MEDIAN NERVE.—*Causalgia.*—In many cases the most prominent symptom of injury to the median nerve is *pain*.

Motor disturbances are always present, but are usually slight, the weakness affecting mainly the flexors of the index finger and the thenar muscles.

Vasomotor changes are a feature of this type. In many cases perspiration is diminished over the radial half of the palm, and the skin becomes dry and scaly. In others, perspiration is increased over the median area.

Sensory disturbances.—Pain comes on about a month after the injury, at first as tingling or pricking in the finger-tips and palm, later as a constant severe smarting, dragging or *burning* pain—hence the name *causalgia*. Added to the constant pain, which never ceases day or night, paroxysms occur, in which the pain increases suddenly in intensity. The application of cold water gives temporary relief, and patients often wear bandages or gloves which they keep constantly moistened. Many develop a phobia of dryness. They will not touch dry objects, even with the healthy hand, the sight of another person handling a dry object increases the pain, and any rustling or crackling sound, suggestive of dryness, may bring on a paroxysm.

In severe cases the limb is held flexed at the elbow and wrist, with the hand constantly raised and the fingers extended or hyper-extended. The whole hand atrophies, and irreducible ankylosis occurs with the limb in this position. The skin of the hand is thin and dry. The fingers taper, and the nails are long, brittle, blackened and striated longitudinally. The pain reaches its acme 4 or 5 months after the injury, and then slowly declines, but the limb remains useless. Even in slighter cases, without much deformity, recovery of function is extremely slow, and is rarely complete.

THE ULNAR NERVE.—The ulnar nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the hypothenar eminence, the interossei, the two inner lumbricals, and the adductor and inner head of the short flexor of the thumb. Its sensory area is the ulnar border of the hand, the little finger and the inner half of the ring finger.

Total paralysis.—Paralysis of the *flexor carpi ulnaris* may be detected by palpating the tendons when the wrists are flexed against resistance. The limpness on the affected side contrasts strongly with the firmness on the sound side. Lateral movements of the hand are unaffected, as these are carried out by the extensors.

Paralysis of the ulnar portion of the *flexor profundus digitorum*. In making a fist, flexion of the index finger is perfect and that of the middle finger good, whilst in the ring and little finger it is absent or very feeble. This weakness is best seen when flexion is attempted with the index and middle fingers extended. Even when the fingers can be flexed by the action of the flexor sublimis, the power of resisting passive extension is completely lost in the terminal phalanx of the two ulnar fingers. Paralysis of the *hypothenar* muscles abolishes lateral movements of the little finger, and

diminishes the power of flexion at the proximal joint. Paralysis of the interossei and of the inner two lumbricals leads to the production of the "claw-hand."

The action of these muscles is to flex the fingers at the proximal joints with the distal joints extended. In the "claw-hand" the posture of the fingers is just the opposite of this, namely, extension at the proximal joint with flexion of the distal joints. Although all the interossei are paralysed, the defect is only seen in the ulnar fingers, as the radial lumbricals supplied by the median are still healthy. It is produced by the action of the long extensors, which being now unopposed, over-extend the proximal joints, and by the flexor sublimis which flexes the second joint and draws the distal joint down with it. The clawing of the fingers is greatly accentuated when the nerve is paralysed below the point of origin of the fibres to the long flexors of the fingers. Other features of the "ulnar hand" are atrophy of the interossei and of the hypothenar eminence and persistent abduction of the little and ring fingers. The movements of abduction and adduction are lost in the inner two fingers, and often in the middle finger. Further, these fingers cannot be flexed at the distal joint, whilst the proximal joints are extended.

Paralysis of the *adductor pollicis* and of the inner head of the *flexor brevis pollicis* produces peculiar disturbances in prehensile movements. If the patient is asked to grasp a folded paper between his thumb and index finger, and to resist efforts to remove it by pulling, it will be found that this movement, which is normally very powerful, is grossly defective. He cannot grasp the object beneath the thumb with the second phalanx extended; but presses the tip of the flexed thumb against the outer margin of the index finger.

Sensory disturbances.—In complete lesions, all forms of sensation are abolished in the little finger, and along the ulnar border of the hand. Beyond this there is usually diminished sensibility on the ulnar side of the ring finger, and over a narrow area towards the centre of the hand on both aspects. Spontaneous pains are rare, and vasomotor changes are usually slight.

Partial paralysis.—In partial lesions the same symptoms are found in a less degree. The small muscles of the hand suffer most. Clawing may be slight or absent. Neuralgic pains may be felt in the distribution of the ulnar nerve; but causalgia is never seen in lesions of this nerve alone.

Recovery of sensation is usually complete before movement is regained. The flexor carpi ulnaris recovers first, then the long flexors of the fingers, and last the small muscles of the hand. In these recovery is extremely slow. When recovery of movement is complete the patient can abduct and adduct the middle finger with the palm flat on a table, and he can also scratch the table with the nail of the little finger without moving his wrist.

THE MUSCULO-CUTANEOUS NERVE is rarely affected alone, but is often implicated with the brachial plexus. It supplies the biceps, coraco-brachialis and brachialis anticus. Flexion of the forearm can still be carried out by the supinator longus; but the power of flexion is greatly diminished. Sensation may be diminished or lost along the radial border of the forearm.

THE CIRCUMFLEX NERVE supplies the deltoid and teres minor, and the skin over the deltoid. It may be injured alone in injuries to the shoulder and by pressure of a crutch. The chief symptom is paralysis of the deltoid with almost complete inability to raise the arm.

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INTERNAL CUTANEOUS NERVE—Division of this nerve produces a narrow area of anæsthesia on the inner side of the forearm.

THE LESSER INTERNAL CUTANEOUS supplies the skin on the inner aspect of the upper arm.

In war injuries lesions of the nerves of the lower limb are very frequent; but in civil practice, apart from sciatica, local lesions of these nerves are rare.

THE LUMBO-SACRAL PLEXUS.—The *lumbar plexus* may be damaged by abdominal tumours, and its roots by new-growth or other disease of the vertebræ. In a certain number of cases signs of inflammation of the lumbar plexus are found in association with sciatica or neuritis of the *sacral plexus*.

ANTERIOR CRURAL NERVE (L_2, L_3, L_4).—This is the largest branch of the lumbar plexus. It supplies the psoas, iliacus, pectineus, sartorius, adductor longus and quadriceps femoris. It may be injured alone by fractures of the pelvis or of the femur, by dislocations of the hip, or by implication in wounds, psoas abscesses or new growths.

The most prominent symptoms are loss of power to extend the knee, loss of the knee-jerk, wasting of the quadriceps, and sensory disturbances over the anterior surface of the thigh and inner surface of the leg. The psoas always escapes, unless the plexus itself is also damaged; but flexion at the hip may be imperfect through paralysis of the iliacus. Owing to the rapid dispersion of the branches in the thigh, wounds in this part often cause partial lesions. In these the *nerve to the quadriceps* is most often injured. The resulting paralysis causes serious disability in walking as the knee gives way at every step, especially in going down stairs, and lameness lasts for a long time after complete return of voluntary movement.

OBTURATOR NERVE (L_2, L_3, L_4).—This nerve is rarely damaged alone. It supplies the three adductor muscles, the obturator externus and the gracilis. The symptoms are weakness of adduction and internal rotation at the hip.

EXTERNAL CUTANEOUS NERVE (L_2, L_3).—This nerve supplies an area of skin on the buttock, and through its femoral branch the skin on the antero-lateral aspect of the thigh. As a result of injury, but more often without obvious cause, the skin in the territory of this nerve may show peculiar sensory disturbances, which have been described under the name of *meralgia paræsthetica*. Most cases occur in men. In women it is usually associated with pregnancy. The nerve is tender on pressure at the point where it passes from under Poupart's ligament, and neuralgic pain or numbness and tingling is felt in the skin, which may be slightly insensitive on objective examination or extremely hyperæsthetic, so that the slightest touch causes pain. The symptoms, which are always unilateral, are made worse by walking, and may cause serious incapacity by their persistence and severity. In severe cases the nerve should be excised.

The *sacral plexus* may be damaged by growths or inflammation in the pelvis, by compression during parturition, and by penetrating missiles. It is also often the seat of spontaneous neuritis.

THE GREAT SCIATIC NERVE (L^4, L^5, S^1, S^2).—This nerve supplies the flexors of the leg and all the muscles below the knee. It may be involved in pelvic new growths, or injured by fractures of the pelvis or femur. Next

to the musculo-spiral it suffers in gunshot wounds more often than any other nerve.

Total paralysis.—The foot drops, and the toes point downwards. Walking is possible, but the patient cannot stand on the heel or toes of the paralysed foot. The knee is raised high, but the steppage is not so marked in total lesions as when the external popliteal alone is paralysed. All movement below the knee is abolished. When the wound is in the buttocks flexion of the knee is very weak. The foot becomes œdematous if allowed to hang down. Sweating is often absent on the sole and dorsum of the foot, but is normal on the inner side of the foot, which is supplied by the anterior crural. The skin is dry and thin, and may be scaly. Hyperkeratosis of the sole is common. Subjective sensibility is rarely affected. The skin is completely anæsthetic over the entire foot, except the inner border of the sole and around the internal malleolus. The anæsthesia extends upwards on the postero-external aspect of the calf in its lower two-thirds, embracing the tendo Achillis and external malleolus. Beyond this area of complete anæsthesia there is a wide zone in which sensibility is diminished. The sense of position and passive movement is abolished in the foot and toes. The knee-jerk is present. The ankle-jerk is always lost. Stimulation of the sole may produce a contraction in the tensor of the fascia lata; but there is no response in the foot.

Partial paralysis.—In wounds of the sciatic nerve it often happens that the fibres of the external popliteal alone are wounded, since the sciatic trunk often divides into the internal and external popliteal branches as high as the great sacro-sciatic notch. The symptoms are described below under paralysis of this nerve. In other cases, the fibres of the internal popliteal are damaged either alone, or with some of the fibres of the external popliteal. In this case the outstanding clinical feature is pain of the same nature as that already described in lesions of the median nerve.

EXTERNAL SCIATIC NERVE.—This nerve may be injured as it winds round the fibula by wounds or fractures or by compression of a tight bandage. The paralysis is usually severe, all the muscles being equally affected. The foot is dropped and inverted, and the toes are slightly flexed. Dorsal flexion of the foot, extension of the proximal phalanges of the toes, and abduction of the foot are impossible. The patient can walk, and he can stand on tip-toe, but he cannot run, and walking is made difficult by the foot-drop. Subjective sensory disturbances are usually absent. The skin is anæsthetic over a narrow band which extends from the outer surface of the leg in its middle third, downwards beside the outer border of the tibia, and along the middle of the dorsal aspect of the foot as far as the base of the toes. For an inch or so, on both sides of this band, the sensibility of the skin is diminished. The knee-jerk and ankle-jerk are present. The plantar response is always flexor. Vasomotor changes are slight, and trophic changes are absent.

INTERNAL SCIATIC NERVE.—This nerve is rarely injured alone. It supplies the popliteus, the calf muscles, the flexors of the toes and the intrinsic muscles of the foot. When it is paralysed, the patient is unable to stand on tip-toe, or to extend or invert the ankle, or to flex his toes. Paralysis of the interossei leads to a claw-like deformity of the foot, associated with lowering of the heel and raising of the metatarsus—talipes calcaneo-valgus.

The calf muscles are flabby and the ankle-jerk is abolished. Sensation is lost on the sole, except along its inner border, on the outer border of the foot, and on the plantar surface of the toes. Causalgia, similar to that in paralysis of the median, is very often present.

POSTERIOR TIBIAL NERVE.—This nerve may be injured by a penetrating missile or a deep wound in the calf. Movements of the ankle are unaffected, and anæsthesia is confined to the sole of the foot and heel, or merely to its inner half. The paralysis of the intrinsic muscles of the foot may escape detection, and the lesion may easily be overlooked, especially when the nerve is injured below the origin of branches supplying the flexor longus hallucis and the flexor longus digitorum. The symptoms then are pain in the sole of the foot, anæsthesia on the sole, and paralysis of the plantar muscles.

Treatment of Local Nerve Lesions.—Treatment must depend on the nature and degree of the lesion. During the long period which elapses between the onset of paralysis and the first signs of recovery, even in cases of simple physiological interruption of the nerves, every effort must be made to prevent degeneration of the muscles, to keep the circulation of the limb active, and to prevent the occurrence of contractures and deformities. Massage, movements, electrotherapy and suitable appliances all have their uses. With regard to operative treatment, it must be remembered that more than half the cases of nerve injuries undergo spontaneous cure. It is advisable, therefore, to wait three or four months before an operation is undertaken. If, at the end of this time, the wound is soundly healed and all signs of sepsis have disappeared, and if, as a result of repeated examinations, no sign of recovery has been detected, no harm can be done by exposing the nerve. If it is found to be divided completely, the ends should be "freshened" and sutured end to end. If the nerve is notched laterally, the edges of the notch should be pared and sutured, care being taken to preserve the bridge of uninjured tissue. Sometimes the nerve at the site of the lesion appears as a fibrous, flattened band between two swellings on the nerve. In most of such cases the nerve is completely divided, and the condition calls for resection of this fibrous tissue and end-to-end suture. Another common finding, when the nerve is exposed, is a nodule or cicatricial swelling in the course of a nerve which has maintained its continuity. In these cases the continuity of the nerve should not be interrupted. It should be freed from adhesions, and incised in the long axis of the swelling. All operations which involve grafting of nerves are futile. For an account of the great advances in the technique of the surgical treatment of nerve injuries which have been made as a result of experience gained in the Great War, special treatises must be consulted.

The treatment of painful forms of nerve lesions is extremely difficult. In severe cases external applications and internal medication entirely fail. Simple freeing of the nerve sometimes gives relief. Where this fails, it may be advisable to practise complete division followed by immediate suture. Alcoholisation of the nerve trunk often gives immediate and lasting relief. Under general anæsthesia the nerve is freed, and then injected with 1 c.c. of weak alcohol at a point two or more centimetres above the lesion. This, of course, is followed by motor paralysis; but recovery occurs in about six months.

INTERSTITIAL NEURITIS

Synonym.—Neuro-fibrositis.

Definition.—A malady which commonly attacks the large nerve plexuses or nerve trunks, but which may affect any peripheral nerve trunk, and which is characterised anatomically by an inflammation of the connective tissues which surround and bind together the nerve fibres into the nerve trunks. This fibrositis, which may be local or diffuse in the affected nerves, is the result of the causes of fibrositis in general, and is frequently associated with fibrositis elsewhere, as, for example, when sciatica is associated with lumbago. The symptoms are those of irritation of the nerve fibres, namely, pain in the distribution of the nerve trunk, tenderness of the nerve trunk, muscular fibrillation and cramp. Loss of function of the nerve fibres in the way of loss of sensibility or muscular paralysis is the rarest of events in interstitial neuritis, and is seen only as the result of terminal cicatrisation in severe cases. Muscular wasting may occur in severe or in longstanding cases, but it is a general wasting of muscles of the painful region, not confined to the distribution of the nerve involved, and therefore resembling the muscular wasting which is seen in joint disease.

Pathology.—The malady is met with soon after puberty, and is incident chiefly upon the first half of adult life, being unknown in childhood and rare in old age. It is often associated with other forms of fibrositis such as lumbago. Often it arises spontaneously, without external cause; but exposure to cold may directly cause it, as also may injury such as stretching, bruising or wounding of the nerve trunk. Gout and diabetes are well-known clinical associations.

The morbid anatomy is well seen when the nerve is exposed during operative procedures for the relief of the condition. The affected nerve trunk is swollen and pink in colour; the sheath is distended, and droplets of fluid exude when it is incised, and sometimes the nerve is adherent to the surrounding tissues. This inflammatory condition may be local and appear as a pink bulbous enlargement of the nerve trunk, or it may spread widely over a long stretch of the nerve trunk and its branches. When the inflammatory process subsides there may be cicatrisation of the peri- and endo-neurium. Only in the rarest cases does the morbid process become so severe as to interfere with the more important functions of the nerve trunk with the production of motor and sensory paralysis, and even in these cases complete ultimate recovery is the rule. The local inflammatory condition causes a slight shortening of the nerve trunk, and this causes the affected limb to be held in that position which will keep the nerve trunk most relaxed. It is also the cause of the severe pain which occurs on any movement which stretches the nerve trunk.

Interstitial neuritis is sometimes an associate of arthritis. For example, in arthritis of the shoulder-joint it is not uncommon to meet with definite involvement of the brachial nerves, and again in chronic arthritis of the hip-joint the inflammatory process may extend from the capsule of the hip-joint directly into the contiguous sciatic nerve.

The nerves may affect any of the nerve roots or nerve trunks, and sometimes several of these may be co-involved. When the nerve roots

are affected "radicular neuritis" results. The sciatic nerve is by far the most common seat of the disease, producing the condition known as "sciatica." Next in order of frequency comes the brachial plexus, causing "brachial neuritis," the anterior crural nerve causing "anterior crural neuritis," the upper part of the cervical plexus producing "cervico-occipital neuritis," and the intercostal nerves producing the so-called "intercostal neuralgia."

Symptoms.—These are the same whatever nerve is affected, and consist in—(1) Pain radiating in the area of distribution of the affected nerve, of a dull, aching character with acute exacerbations and often very long-lasting. (2) Tenderness of the affected nerve to pressure and stretching. (3) Subjective peripheral sensations such as tingling, burning or numbness. (4) General wasting of the muscles of the surrounding region with marked hypotonus, not confined to the muscles supplied by the affected nerve and akin to arthritic muscular atrophy. This wasting may reach a very remarkable degree. (5) Increase of the deep reflexes of the limb. (6) Diminution or loss of the deep reflex in the supply of the affected nerve. This is a valuable indication of the severity of the lesion. In a case of sciatica, for example, all the muscles of thigh and leg are wasted, the knee-jerk and the adductor-jerks are markedly brisk, whereas the ankle-jerk, which is in the sciatic supply, is diminished in slight cases and lost in severe cases. (7) The affected limb is held in a characteristic position to avoid stretching of the nerve, and the gait is similarly modified. (8) Trophic and vasomotor changes are not uncommon. (9) Fibrillation is often present.

Diagnosis.—There is sometimes considerable difficulty in the diagnosis of cases of interstitial neuritis on account of the almost identical clinical picture which may occur in the early stages of pressure upon nerve roots or nerves by tumours. The following points are of value in distinguishing the two conditions: The pain of pressure lesions is rarely so severe as that of interstitial neuritis. Tenderness on pressure or stretching of the nerve trunks is absent in pressure lesions. Signs of loss of function—paralysis and sensory loss—come on early in pressure lesions. The most careful search should be made in every case for any possible cause for local pressure, such as primary and secondary neoplasms, spinal tumours, spinal caries and diabetes. To make a diagnosis of interstitial neuritis in the presence of a mammary or testicular carcinoma, removed or not, is to advocate the highly improbable, whatever the symptoms may be.

BRACHIAL NEURITIS.—This form of interstitial neuritis is somewhat rare, and is met with chiefly in women over the age of 35 years. Sometimes it follows injury to the brachial plexus from any violence causing undue separation of head and shoulder. More often it arises spontaneously. The pain, which is often of sudden onset, may be of great severity, and may be at first referred to the region of the plexus itself, the back of the scapula, the axilla, the forearm or the hand, is at first intermittent, but it soon becomes continuous and spreads over the whole upper limb. Tingling and numbness in the hand and trophic changes in the skin and nails of the fingers are the rule.

One of the great difficulties in this malady is that in the upright position the weight of the arm and shoulder carry the shoulder downwards and stretch the inflamed plexus, adding greatly to the pain. Therefore it should be treated with the recumbent position upon the back in bed. Further,

every movement of the hand or arm tends to increase the pain. Splints which keep the arm in the abducted position and the shoulder raised so as to prevent tension upon the plexus are invaluable.

There is little difficulty in diagnosis, the only confusable conditions being arthritis of the shoulder and cervical rib, in neither of which conditions is there any tenderness of the nerve trunks of the plexus.

CERVICO-OCCIPITAL NEURITIS.—This condition, which is by no means rare, is characterised by pain in the upper part of one side of the neck, radiating over the branches of the upper cervical plexus, the great occipital being the most common, and the supra-sternal, supra-clavicular and supra-acromial branches less common seats for the pain. The fibrositis not infrequently co-involves the fibrous structures in the region of the articular and transverse processes, giving rise to pain and stiffness of the neck on movement. When the pain is confined to the great occipital distribution alcohol injection is sometimes most efficacious.

SCIATICA.—In few common and familiar maladies is as much difference of opinion as to ætiology, pathology and treatment as in the case of sciatica. The classification suggested by Barnes Burt is probably the most useful. He recognises three types: (i) root sciatica, sometimes called radiculitis; (ii) trunk sciatica; and (iii) referred sciatica, and believes that the ætiology of the three is different in each case. Root sciatica is commonly due to spondylitis, or strain of the lumbar vertebra; trunk sciatica depends upon interstitial neuritis of the nerve sheath; and referred sciatica results from inflammation of some structure (muscle, bursa, joint) supplied by the sciatic plexus. He finds in his own series of cases that root sciatica accounts for 36 per cent. of the total, trunk sciatica for 18 per cent., and referred sciatica for 46 per cent. In previous editions of this book, the description was confined to root trunk sciatica. This account now follows, questions of differential diagnosis being discussed subsequently. It has been said that true sciatica as here defined is never bilateral, and that bilateral sciatic pain is always the result of gross lesions involving the nerve. It is important that this error, which has crept into so many textbooks, should be contradicted, for sciatica is occasionally bilateral, and the sciatica which occurs in glycosurics is usually bilateral.

The malady is not met with in childhood, but it begins to be common soon after puberty, and its incidence is greatest upon early middle life. In the majority of the cases it arises without assignable cause, sometimes injury to the nerve of any nature, as from a twist of the leg, a bruise or a fall is responsible. Only in rare cases does exposure to cold and wet seem to have excited the onset. It is important that the urine should be tested in every case of sciatic pain, for glycosuria is more often found in cases of sciatica than is usually believed. In the case of an apparent sciatica, the possibility of rupture of an intervertebral disk should be borne in mind (cf. page 1773).

Symptoms.—The chief symptom is pain along the course of the nerve or of its branches, and since the sciatic nerve often divides within the pelvis into the great internal and external sciatic branches, the pain may be confined to the distribution of one of these alone. One feature of the pain valuable for diagnostic purposes is that it never reaches above the crest of the ilium, but in this connection it must be borne in mind that fibrositis of the back (lumbago) not infrequently precedes or accompanies the onset of sciatica.

The pain may be partly intra-pelvic, for the sciatic nerve is formed within the pelvis. Where the interstitial neuritis is entirely intra-pelvic, tenderness of the nerve trunk to digital pressure in the thigh and buttock fails as a physical sign. The onset is occasionally sudden, and associated with slight pyrexia and constitutional disturbances as in other forms of fibrositis; but, as a rule, the malady sets in gradually with pain in the buttock, back of the thigh or leg, in movements and in postures which make the nerve tense, or cause pressure upon it. The pain gradually increases in severity. It may be both gnawing and burning and sharp and darting in character. It is usually continuous, with occasional severe exacerbations which occur spontaneously or are excited by movement. Its intensity generally increases at night. The seat of the pain often varies from day to day.

Extreme tenderness of the nerve on pressure is rarely absent, except in those cases where the neuritis is intra-pelvic, and the tender region usually indicates the situation of the lesion of the nerve trunk. Stretching the nerve by extending the knee with the thigh flexed is productive of great pain which may be lasting. The best method of testing the sensitivity of the nerve to stretching is to put gentle pressure with the thumb on to the popliteal space as the patient sits in a chair with the knee bent at a right angle. Sometimes there is considerable tenderness of the muscles. The muscles waste not only in the sciatic supply, but throughout the whole lower limb and buttock, surely from reflex irritation as in arthritis atrophy. Cramp in the leg and reflex spasm are common, and muscular fibrillation is often seen. Paræsthesia in the form of tingling, burning and numbness is the rule; but loss of sensibility only occurs in the rarest and most severe cases, and its presence should always suggest the presence of a pressure lesion. Barnes Burt states that in root sciatica (radiculitis) there is also some rigidity of the lumbar spine, tenderness along the lateral aspects of the spine in this region, and also pain along the distribution of the lateral cutaneous nerve of the leg.

Peculiarities of stance, gait and position arise from the tenderness of the nerve to stretching, that position being assumed by the patient which keeps the nerve slackest. In standing, the weight of the body is placed upon the sound limb, and the other limb is flexed at hip and knee and extended at the ankle, with the toes only resting on the ground. In walking, the patient limps in this same position without straightening his knee or extending his ankle, and in bed he lies with knee flexed and ankle extended.

The knee-jerk is sometimes markedly increased notwithstanding the wasting of the quadriceps, the only exception being when sciatica is complicated with anterior crural neuritis. The ankle-jerk being in the sciatic supply tends to be diminished in proportion to the severity of the neuritis, and in severe cases it is always lost. It may not return after recovery from the original attack.

Slight trophic and vasomotor changes in the periphery of the limb are commonly seen.

In very rare cases the cicatrization which follows the inflammatory process may cause motor and sensory paralysis of all the region below the knee. Such a case came under our care 2 years after the onset, and was explored by Sargent, who found the nerve in the gluteal region densely cicatrised and widely adherent to the muscles. The nerve was freed, and

incised longitudinally in many places, and this patient made a complete recovery. Every degree of severity may be met with from the mildest to the most acute, and from the most rapid lasting but a few weeks to the most chronic lasting 2 or more years.

It is a most remarkable fact that severe sciatica never occurs twice in the same limb. One severe attack seems to free the affected nerve from subsequent liability to the affection, and it is comforting to be able to assure the patient that he will never have the trouble again in the same limb. In a very large experience we have never met with an exception to this rule.

Diagnosis.—Root and trunk sciatica must be distinguished from “referred sciatica,” and from pains of sciatic distribution due to involvement of the nerve or its roots by growth.

In sacro-iliac disease pain may be referred along the sciatic nerve, but the nerve trunk is not tender to pressure or to stretching, while the joint is tender, and radiography may reveal a lesion in it. In osteo-arthritis of the hip the signs of sciatica are absent, while there is limitation of active and passive movement of the joint, and pain on movement. In this condition it is more common for pain to be referred down the front of the thigh than along the sciatic distribution. Fibrositis of gluteus medius and minimus may also produce pain of sciatic distribution of some severity.

In new growths involving the nerve, the clinical picture is not really that of a true sciatica. The pain differs in character, is frequently accompanied by muscular weakness and wasting and by sensory loss, and the patient's general condition is cachectic and suggestive of malignant disease. There may also be a history of primary carcinoma. The presence of bilateral sciatic pain is, as has been said, not pathognomonic of involvement of the nerve roots by growth. Indeed this involvement is as commonly unilateral as not.

The pains of tabes and other nervous diseases when confined to the sciatic distribution are distinguished by the presence of other physical signs of those diseases.

Course and Prognosis.—Sciatica commencing acutely tends in the course of time to lessen in severity and become chronic, but some acute cases cure rapidly. When the malady has a slow commencement it usually lasts longer than does an acute case, and it is much more liable to exacerbations than when commencing acutely. The traumatic cases show no essential difference from the spontaneous, except that in the former adhesions of the nerve to the muscles from bruising is likely to prolong the duration of symptoms. The prognosis is always absolutely good as regards recovery, and in severe cases there is no likelihood of relapse. In slight cases, however, subsequent attacks are not uncommon.

Treatment.—The first essential in the treatment of all recent cases is to secure rest and to avoid all those things which excite or increase the pain. The duration of the attack is often considerably prolonged by neglect of this most essential element in treatment. Sometimes the fixation of the limb in a semiflexed position by means of Liston's or Macintyre's splint gives great relief. The use of the bed-pan is advisable to avoid flexion of the hip and stretching or pressure upon the nerve in the act of defecation. On the other hand, towards the end of a chronic case, active exercise with massage and passive movements are necessary to restore the shortening of

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the nerve. The application of heat in the form of hot-water bottles, poultices and radiant heat is invaluable for the relief of pain, and for curative purposes. Counter-irritation is very useful, and is best used in the form of the strong tincture of iodine which should be painted in a broad strip over the course of the sciatic nerve, from sciatic notch to heel, daily until the skin becomes inflamed, just short of blistering. Massive injections of from 4 to 8 ounces of sterilised normal saline solution, made slowly into the region of the affected nerve at a temperature of 104° F., are said to be valuable both in acute and chronic cases. Acupuncture of the nerve with a series of specially designed needles is a useful and ancient remedy which acts by puncturing the sheath of the nerve, and allowing the escape of inflammatory exudation. Attempts to stretch the nerve by flexion of the thigh and extension of the knee may do great harm, and rarely do good. In chronic cases of sciatica or brachial neuritis and in fibrositis of the glutei, iodine given in the following manner is sometimes useful:

℞ Iodi . . .	gr. 1
Pot. iodidi . . .	gr. $\frac{3}{4}$
Sp. rect. . . .	mun. 12

Sig. min. 12 to be dropped into half a teacupful of water, and to be taken after being allowed to stand for 15 minutes. This may be administered thrice daily, and continued for 3 weeks. Some patients tolerate the doubling of this dose after the first week. The salicylates are of great service, especially in the form of aspirin, which may be given liberally. Urodonal in doses of 60 grains thrice daily is also often valuable. In the more chronic cases sulphur and guaiacum are serviceable. For the relief of pain heat is generally beneficial, but in some of the most acute cases the application of heat increases the pain, and in these an ice-bag will sometimes give great relief. All the analgesics of the coal-tar series, acetanilide, phenacetin, etc., are valuable adjuvants to relieve pain, and these may be conveniently prescribed with aspirin, or if sleep be difficult with barbitone or carbromal. When pain is very severe and rebellious to the above-mentioned remedies, opium or morphine is indicated. It is essential from the exhausting quality of the pain that the patient should be well fed, and alcohol is often of service.

RUPTURE OF THE INTERVERTEBRAL DISK

Synonym.—Herniation of the nucleus pulposus.

Definition.—A condition of pressure upon the spinal cord or on the roots of the cauda equina resulting from the extrusion into the vertebral canal of the nucleus pulposus of the intervertebral disk. It is traumatic in origin.

Ætiology.—The majority of cases give a history of injury at, or shortly before, the onset of symptoms. The injury is commonly of the variety known as a strain of the back, due to sudden bending, the lifting of heavy weights, or sudden movements of the back, as when striving to avoid a fall. Males are commonly affected.

Pathology.—Formerly the condition under discussion was one recognised on laminectomy, and spoken of as endochondroma of the disk. Actually in the circumstances enumerated above, the disk ruptures and its nucleus

(nucleus pulposus) herniates into the canal. The common seat of such rupture is the lumbar spine, commonly between the fourth and fifth lumbar, or between the fifth lumbar and the sacrum. Multiple ruptures have been recorded.

Symptoms.—The outstanding feature is pain, starting in the small of the back and radiating down one thigh, the outer side of the leg and the ankle. The pain is severe and lancinating, aggravated by stooping and relieved by lying down. There is some flattening and limitation of movement of the lumbar spine. Flexion of the extended leg at the hip may be painful. In some 60 per cent. of cases the ankle jerk is lost, other abnormal signs being absent. Occasionally more severe signs of root compression are present, and in rupture of dorsal disks some signs of compression paraplegia, mild in degree, may be found. The most typical picture is one of pain in the small of the back radiating down one leg. In other words, the symptoms usually mimic those of sciatica.

Diagnosis.—In addition to the history and signs mentioned, radiography of the spine may reveal narrowing of the intervertebral space at the level of the damaged disk, but the intrathecal injection of lipiodol may be necessary to confirm diagnosis.

Treatment.—Attention having been drawn to the condition, there is a not unnatural tendency to diagnose it somewhat too frequently, and to submit patients unnecessarily to lipiodol injection. Careful consideration of all the facts, and conservative modes of treatment, *e.g.* rest, massage, etc., should first be tried. The operative procedure for the removal of the extruded portion of the disk involves laminectomy and is a severe ordeal. For this reason it should not be undertaken unless the diagnostic indications are clear and unequivocal.

CERVICAL RIBS

Ætiology.—The development of the ribs at the thoracic inlet depends on the mode of formation of the brachial plexus, for the nerves are large structure in the embryo at a time when the ribs are soft and pliable. When the plexus is "normal," a well-formed first rib springs from the first dorsal vertebra. If, however, the plexus is "post-fixed," that is, when the contribution to the plexus from the fourth and fifth cervical segments is small and the fibres from the first and second dorsal segments form a powerful cord, this cord in rising over the first dorsal rib may compress and deform it to such an extent that it presents the characters of a rudimentary rib. On the other hand, and this is more frequent, when the plexus is pre-fixed, that is, when the contribution from the upper cervical segments is relatively large and that from the dorsal segments is small, a supernumerary rib is allowed to develop from the seventh cervical vertebra. When this pre-fixation is pronounced, the seventh cervical rib is often very large and is easily felt in the neck. In these cases symptoms are usually absent. In a certain number of cases in which the abnormality is intermediate in degree, symptoms are caused by compression of the lower cord of the plexus as it passes over the supernumerary rib, or over the deformed first rib. This compression may be exercised by the bony portion of the extra rib, but more often the nerves are damaged by a fibrous prolongation of the rudimentary rib which connects it with the first rib.

But these abnormalities in the ribs only cause symptoms in some 10 per cent. of the cases in which they are present. Further, the symptoms are often unilateral with bilateral supernumerary ribs, and the symptoms are often most prominent on the side of the smaller extra rib. Again, the onset of symptoms is usually delayed until adult life is reached. It is clear, therefore, that some contributory cause must come into play. This is found in the dropping of the shoulder girdle, which is normal in adolescents, and is often excessive in persons whose muscular tone is low. In a child the clavicle rises boldly as it passes outwards. In a normal adult male the clavicle is almost horizontal, in women it droops slightly, and in those who develop symptoms of pressure on the nerves, the outer is usually distinctly lower than the inner end. In the latter, the lowest cord of the plexus is submitted to constant rubbing against the extra rib which rises and falls during respiration, and it is compressed by any movement of the arm which depresses the shoulder girdle. Relief is obtained by raising the shoulders, and patients soon learn to support the limb and to assume attitudes in which pressure on the nerves is relieved.

Women suffer most often, the right arm being affected more often than the left. The onset is usually gradual, but occasionally it comes on suddenly after childbirth, or on lifting a heavy weight.

Symptoms.—These may be sensory, motor, or vasomotor, either singly or in combination. Subjective sensory disturbances are most frequent. They take the form of numbness and tingling or neuralgic pains. Paræsthesiæ are most often unilateral, and are frequently confined to the ulnar or to the radial side of the hand and fingers. It is rare for all the fingers to be affected. Pain, when present, is usually felt below the elbow. It is often neuralgic, darting down the arm, and again confining itself to one border of the limb. It hardly ever radiates from the neck.

Objective sensory disturbances are usually slight or absent. They may be found over the ulnar or radial border of the distal portion of the limb in an indefinite area, which does not conform to the distribution either of the ulnar or radial nerve.

Muscular atrophy is not so frequent as subjective sensory disturbance. In the "median type," wasting is confined at first to the abductor and opponens pollicis muscles, and the outer part of the thenar eminence shows a remarkable reduction in size, which contrasts strongly with the inner part, which retains its normal bulk. In the "ulnar type," wasting appears first in the small muscles of the hand supplied by the ulnar nerve. In some cases all the muscles of the hand and, to a less degree, the flexors in the forearm show considerable wasting. The atrophy is frequently bilateral and symmetrical.

Vasomotor disturbances are very common. The hands feel hot or cold, they may be œdematous or discoloured, and the changes may suggest Raynaud's disease. Pressure on the subclavian artery sometimes causes inequality of the pulse. This disappears when the arm is raised.

Diagnosis.—The presence of pain, paræsthesiæ or vasomotor disturbances in the upper limbs, or wasting in the muscles of the hands, should always arouse the suspicion of supernumerary or rudimentary ribs. When pain is the only symptom, its distribution along one border of the arm or hand, and the patient's account of the manner in which it may be increased or diminished by raising the shoulder girdle or performing movements which

depress it, usually direct attention to the cause. Symmetrical atrophy in the hands may suggest progressive muscular atrophy of spinal origin, but this diagnosis is usually rendered untenable by the association of sensory troubles or vasomotor phenomena, or by the findings on X-ray examination of the neck. For the differential diagnosis from syringomyelia, see page 1732.

Treatment.—Pain may be relieved by rest with the arms suitably supported. Atrophy calls for immediate operation to remove the offending rib. Pain is always relieved by operation, either immediately or after an interval of some months. The progress of atrophy is always retarded, and complete recovery may occur if an operation is undertaken early.

OBSTETRICAL PARALYSIS

It is important and useful to group together under this heading all those conditions of paralysis occurring, either in mother or child, which are the result of the processes of labour in the passage of the foetal head through the pelvis. Autopsies upon the still-born, and upon children who have survived birth for a few days only, have shown that hæmorrhage into the meninges is of common occurrence, and it has been argued that such meningeal hæmorrhages are the cause of many of the conditions of cerebral paralysis which are present immediately after birth, or which appear during the first year of life, and especially the cause of cerebral diplegia. The pathological conditions found in the brain in cases of cerebral diplegia, however, are such as make it absolutely impossible that they could be caused by meningeal hæmorrhage, for no sign of old hæmorrhage is ever found, nor could hæmorrhage cause a general cell atrophy of the brain without signs of any local lesion. It seems clear, then, that though meningeal hæmorrhage may be of common occurrence during birth, and may be the cause of still-birth, yet there is no clinical or pathological evidence to show that it gives rise to any lasting cerebral defect.

The following condition may occur : (1) In the child : facial paralysis ; hemiplegia from laceration of the brain substance ; fracture-dislocation of the spine with transverse lesion of the spinal cord ; injury to the brachial plexus from the separation of head and shoulder in traction ; and injury to peripheral nerve trunks at the elbow, axilla or groin, in using traction with the finger.

(2) In the mother : paralysis of the supply of the lumbo-sacral cord and obturator nerve from prolonged pressure of the head against the sacrum and pelvis.

Facial paralysis.—This is usually caused by the pressure of the forceps upon the facial nerve as it crosses the ramus of the jaw, but it has been known to occur where instruments have not been used. When unilateral, as is the common event, it gives rise to little or no difficulty with sucking, and is evidenced by the unsightly deformity of the face, which is drawn over to the sound side. When bilateral, it is one of the causes of complete inability to suck, and on account of the flaccid symmetry of the face may easily be overlooked. It necessitates spoon feeding for a considerable time. Obstetrical facial paralysis invariably recovers within a few weeks and does not give

rise to after-contraction. Gentle stretching and massage of the face with the finger is the only treatment required.

Hemiplegia from laceration of the brain may occur during delivery in contracted pelvis from the pressure upon the sacral promontory, and has been caused by the use of forceps. It is exceedingly rare, and is generally rapidly fatal from the associated hæmorrhage. It may occasionally be survived, with an irreparable hemiplegic condition.

Fracture-dislocation of the spine is produced by traction upon the after-coming head by pulling upon the trunk. We have seen it associated with injury to the brachial plexus. It occurs most often in the lower cervical region, and the transverse lesion of the spinal cord is usually complete.

Injury to the brachial plexus may occur in traction either upon the head, or upon the trunk, if the head is aftercoming, and is caused by an undue separation of head and shoulder on one side rupturing or straining the brachial plexus. The paralysis is usually of the upper arm or Erb type, the fifth and sixth roots being most affected, and the deltoid, biceps and supinator longus muscles being paralysed, but the whole plexus may be involved and even torn completely across. Traction upon a prolapsed arm has caused lower arm or Klumpke type of paralysis, in which the first dorsal and eighth cervical roots are most affected, and the intrinsic hand muscles and the flexors of the forearm are paralysed. The obstetrical lesions of the brachial plexus are for the most part serious lesions, many of the cases making no motor recovery at all, though sensibility is usually regained. The prognosis depends upon the severity of the damage to the plexus, as to whether the roots are actually torn or only bruised. The slight cases recover well enough.

Injury to the peripheral nerves from pressure or traction upon the flexures is seldom severe enough to prevent a rapid and complete recovery.

Paralysis of the lumbo-sacral cord and of the obturator nerves in the mother, immediately after parturition, is an exceedingly interesting clinical condition. In the first place, the lumbo-sacral cord is in a much more exposed position as regards the foetal head engaging the pelvis than are the other nerves of the sacral plexus, and may be subjected to such severe pressure as causes paralysis, and in the second place, the obturator nerve actually crosses the brim of the pelvis and must of necessity be pressed upon by any large foetal head which passes the pelvic brim. The lumbo-sacral cord paralysis is evidenced by dropped foot and paralysis of the anterior tibial and peroneal muscles and if it is severe, by loss of sensibility over the distribution of the fourth and fifth lumbar roots. Sometimes the third lumbar root area is affected. The obturator nerve involvement is shown by weakness or paralysis of the muscles supplied by the obturator nerve, namely, all the adductor muscles of the thigh. The paralysis may be noticed directly after parturition, or when the patient begins to get about upon her legs. The lumbo-sacral paralysis is usually unilateral, and is nearly always upon the right side. The obturator paralysis is not uncommonly bilateral, and both forms of the paralysis may coexist. There may be numbness, but no pain. This condition nearly always occurs with a first delivery, and often the child's head has been unduly large. It may recur with subsequent deliveries, but this is not a common event.

The prognosis is absolutely favourable, every case making a complete

recovery in from a few weeks to a few months. The treatment is rest in the first place, with gentle massage and passive movements, and when power begins to return the patient may commence to get about.

POLYNEURITIS

Synonym.—Multiple Peripheral Neuritis.

The clinico-pathological condition we know as polyneuritis, and seen in its most typical forms in diphtheritic paralysis or in alcoholic neuritis, represents a very striking and uniform reaction of the nervous system. Invariably associated with it is a reaction of the myocardium, so that there is in cases of polyneuritis a recognised liability to sudden fatal heart failure. It is in the case of diphtheritic paralysis and of beri-beri, another form of polyneuritis, that this mode of fatal termination is most often seen. Indeed, beri-beri may appear as a rapidly fatal cardiac illness before any signs of involvement of the nervous system have had time to develop.

Ætiology.—At first sight the factors that give rise to polyneuritis fall into three groups: (i) certain chemical poisons, (ii) the toxins of certain bacteria, and (iii) certain disorders of metabolism. Widely differing as these three causative factors may seem to be, there is reason to believe that a common underlying factor which is immediately responsible for polyneuritis may underlie them all. It is probable that in the case of groups (i) and (ii) the pathogenic substance gives rise to a disorder of metabolism in the course of which a toxic metabolite is produced in the body, this acting as the direct poison for nervous system and heart muscle. In the metabolic group (iii) the same process is in action.

Thus, in beri-beri, for example, the illness ensues upon the ingestion of a diet deficient in vitamin B₁. In the absence of this substance carbohydrate metabolism is disordered and a toxic metabolite is produced. Thus, beri-beri is not, as the biochemists formerly insisted, a starvation-degeneration of the nervous system, but an intoxication strictly comparable with that obtaining in other varieties, ætiologically considered, of polyneuritis.

The final and complete proof of this unity of causation of polyneuritis, in whatever circumstances it is seen, is not yet available, but there is an increasing body of evidence in favour of it.

Returning for the moment to the ordinary ætiological classifications of polyneuritis, we see that in the case of alcoholic or arsenical polyneuritis the poison is taken by the mouth, and presumably the final common toxic substance reaches the nervous system by the blood stream. In the case of diphtheritic paralysis, on the other hand, the exotoxins are produced locally at the site of the diphtheritic ulceration, whether on the fauces or, as in extra-faucial diphtheria, at some other local site on the body surface. This unique channel of entry gives rise in diphtheritic paralysis to a group of symptoms not found in other ætiological varieties of polyneuritis. This group includes palatal and accommodation paralyses, which precede the appearance of polyneuritis.

It is noteworthy that in the case of extra-faucial diphtheria this initial paralysis is not palatal, but is anatomically related to the site of the diphtheritic lesion (skin ulceration or wound). Yet the paralysis of accommodation

may occur whatever be the site of the diphtheritic lesion. It is believed, therefore, that the exotoxins gain access to the nervous system by conduction from the seat of the lesion via the axones of the nerves which innervate this region. They pass upwards in the axis cylinders to the central nervous system and produce their toxic action directly there, this action being reflected peripherally again as a motor and sensory paralysis of the muscles and skin (or mucosa) in the region of the lesion. Thus, a diphtheritic ulcer on a finger may be followed by a local paralysis of that part before polyneuritis develops. The subsequently developing polyneuritis is then probably produced in the manner described above, while the accommodation paralysis may indicate a specific action of the toxin upon the nervous mechanism concerned. We thus have a local, a specific and a general group of symptoms. The analogy of the local, specific and general phases of tetanus will occur to the reader.

Many of the intoxications of the nervous system commonly included under the heading of polyneuritis are associated with lesions and clinical manifestations which are not those of polyneuritis. Such substances, to name but a few, are lead, mercury, copper, carbon disulphide and carbon monoxide, and it would be erroneous to regard these as causes of polyneuritis. Many infectious fevers are stated to be not rarely followed by polyneuritis, namely, enteric, malaria and dysentery, but the present writer who spent four years with the Egyptian Expeditionary Force in 1915-19 where the two last named of these infections were common saw no case of true polyneuritis associated with them.

Finally, the categories of toxic polyneuritis, acute febrile polyneuritis and Landry's paralysis have no known causal factors. They make their appearance in apparently healthy persons, adequately nourished and free from all discoverable signs of infection, and it is extremely difficult in the present state of knowledge to account for them on any hypothesis of avitaminosis, or to suggest any possible mode of intoxication. In short, the pathogenesis of polyneuritis presents many unsolved problems.

Clinically, polyneuritis is a lower motor neurone type of paralysis accompanied by a varying intensity of sensory loss of much the same type of distribution as the paralysis, and, as already mentioned, by signs of myocardial poisoning.

1. ARSENICAL NEURITIS

Peripheral neuritis may be caused by a single large dose of arsenic, or it may result from prolonged use of the drug in the treatment of such diseases as Hodgkin's disease, chorea and severe anæmia. It is a rare malady, and the likelihood of its appearing under the last-named conditions is negligible. The toxic action of arsenic with alcohol seemed to be greater than that of either alone.

The description given below of alcoholic neuritis applies to this form as well. Hyperæsthesia of the skin and tenderness of the muscles are more constant and more severe in the arsenical form, and paralysis and atrophy of the muscles are often more widespread and more rapid in their progress. Hyperkeratosis of the soles and pigmentation of the skin are characteristic of arsenical poisoning. In a suspected case, the diagnosis can be confirmed

by the discovery of abnormal quantities of arsenic in the urine or in the hair and skin.

The mental changes described in connection with alcoholic neuritis under the heading of Korsakow's psychosis may be present, especially when repeated poisonous doses of arsenic have been taken.

2. ALCOHOLIC NEURITIS

In former years alcoholism was perhaps the commonest cause of severe peripheral neuritis. At present it is a rare disease. It occurs most often in women, especially in those who take small amounts of alcohol frequently. It has often been the first indication of secret drinking.

Pathology.—The changes in the nerves are those of parenchymatous neuritis. They are most intense in the small branches supplying the skin and muscles, and they diminish in severity as the larger branches are approached. They are best seen in the terminal branches of the musculo-spiral and anterior tibial nerves. The wasted muscles often show a reduction in the size of their fibres, and an increase of connective tissue—fibrous myositis. The spinal cord may be healthy, but in almost all cases examination by modern methods shows changes in the nerve cells and degeneration in the tract fibres derived from the posterior roots.

Symptoms.—The onset is insidious, and in most cases premonitory symptoms, such as numbness and tingling in the extremities or cramps in the muscles of the lower limbs, are present for several months before actual weakness occurs. Subjective sensory troubles are a marked feature, even in the early stages. Besides numbness and tingling, the patients complain of feelings of excessive heat or of coldness in the limbs, or of severe aching or cutting pains in the legs. Painful cramp in the calf muscles is a common symptom. It is often worst at night, and may interfere seriously with sleep. Objective examination usually reveals sensory loss, in which the various elements of sensation are affected in a manner which is almost pathognomonic.

Stated briefly, there is anæsthesia of the skin with hyperæsthesia of the deeper structures. Light touches are not appreciated at all or many are missed, the temperature sense is defective, and the prick of a pin causes no pain, whereas even moderate compression of the muscles may cause the patient to cry out. The sensory loss is greatest in the feet and hands and diminishes towards the knees and elbows. Muscular tenderness is usually greatest in the calves. The soles of the feet are also unduly tender. Hyperalgesia is often well marked before anæsthesia of the skin appears. To the disability caused by pains and spasms, weakness of the muscles is added in all but the slightest cases. The arms may suffer first, but in most cases the extensors of the toes, the dorsiflexors of the ankle, and the extensors of the fingers and wrists are attacked in progression, and double foot-drop and wrist-drop result. To overcome the foot-drop, the knees are raised high in walking. This gives to the gait the "steppage" character which is common to all forms of peripheral neuritis. In most cases the distal flexor muscles are also affected, but to a slighter degree. In severe cases, weakness extends to the proximal muscles and even to the muscles of the trunk. The affected muscles become soft and diminish rapidly in bulk. Unless precautions are taken, contrac-

tures occur in the flexor muscles and produce deformities of the limbs, which add greatly to the difficulties of treatment.

At the onset the knee-jerks are exaggerated, but in most cases by the time the patient comes under observation all the tendon reflexes are absent. The cutaneous reflexes may be unaltered, diminished or absent. Sphincter control is retained. Slight bilateral weakness of the face is often present but severe paralysis is rare. Ptosis, nystagmus, and weakness of the external ocular muscles have been observed.

Trophic and vasomotor disturbances in the extremities are common. The hands and feet often perspire freely, and they may be white and cold or red and hot. In some cases oedema of the hands or lower extremities is present. In chronic cases the skin of the hands and fingers is thin, smooth and shiny, and the nails are ridged and brittle.

In almost every case of alcoholic neuritis there is some *psychical defect*. One form—Korsakoff's psychosis—is characteristic of and almost peculiar to this disease. The most prominent feature is failure of memory and loss of appreciation of time and place. A patient who has been bedridden in a hospital for nervous diseases for several weeks, when visited by the resident physician who has attended her daily, will "recognise" him at once as Dr. X, whom she has not seen since he brought her first child into the world some years ago. She is now, she says, in a lying-in hospital which she entered yesterday, and has just been confined with her second baby, who is in bed beside her. She also "recognises" strangers at her bedside, and connects them with events of long ago. Everything is related in the most circumstantial manner, and if the facts were not known her tale might well be accepted as truth. In most cases the mental defects are not so gross. There is merely a failure of memory, to which is added moroseness and irritability, caused by withdrawal of alcohol.

3. DIPHThERITIC PARALYSIS

The exotoxin of diphtheria is highly selective for nervous tissues, and some form of paralysis occurs in a very high proportion of the cases. The intensity of the paralysis bears no constant relation to the severity of the local infection, for cases, in which the original disease has passed unnoticed, may be followed by serious damage to the nervous system. Walshe has classified the nervous manifestations of diphtheria into three distinct groups, namely, the local, the specific and the generalised paralyses.

Local paralysis occurs in parts related anatomically by nervous connections to the site of the diphtheritic lesion. In faucial diphtheria, the local palsy appears in the palate. In extra-faucial diphtheria, *e.g.* infected sores on the limbs, the local palsy appears in the muscles supplied by the segments of the cord to which afferent nerves from the infected focus pass. The reason for this is, that toxins elaborated by the diphtheria bacillus ascend from the primary focus to the cord or the medulla. Having reached the central structures, they diffuse to neighbouring motor cells and, by injuring them, cause paralysis of the muscles they supply. Paralysis of the palate therefore does not occur except in faucial diphtheria.

The *specific* manifestation of diphtheria is paralysis of accommodation. Like trismus in tetanus, it is not due to a local lesion, but occurs in many

cases, whatever the site of origin of the toxins. It is present in cases of both faucial and extra-faucial diphtheria, and is the local effect of exotoxin accepted from the ingeneral blood stream.

The third or *generalised* form of diphtheritic paralysis is multiple neuritis. It follows extra-faucial as well as faucial diphtheria, and is also a result of the action of exotoxin circulating in the blood.

Symptoms.—As faucial diphtheria is the commonest form, the most frequent nervous symptom is *paralysis of the soft palate*. It is shown by the nasal quality of the voice and by the regurgitation of fluids through the nose. As a rule, the weakness is bilateral and equal, but in some cases it is greater on the side on which the local lesion is more severe. It makes its appearance in most instances about the end of the second week, but may come on as early as the fourth day, and as late as the sixth week. The soft palate is relaxed, and its movement on phonation is diminished. The palate may be insensitive, and its reflex is often diminished or lost. Recovery usually occurs in a few weeks. In rare instances the muscles of the pharynx and the vocal cords are paralysed. Together with palatal palsy, it is common to find marked weakness and tenderness of the sternomastoid muscles and masseters. These are also local effects.

Paralysis of accommodation appears about the same time as the palatal palsy, perhaps a few days sooner. The reaction of the pupils to accommodation as well as to light, can almost always be obtained. The trouble is subjective, and is shown by defects of near vision—for example, by inability to read small print. Hypermetropes suffer great inconvenience. In myopes it may pass unnoticed. Paralysis of any of the extrinsic ocular muscles with strabismus and diplopia may occur, and this may be either nuclear or peripheral in type.

Multiple neuritis usually comes on three to six weeks after recovery from the throat infection. Its presence is often detected when patients begin to exert themselves during convalescence. Weakness and aching pains in the legs, unsteadiness in walking, clumsiness in performing fine movements with the hands, feelings of pins and needles in the extremities—all these are common early symptoms. Weakness affects in varying degree the muscles of the neck, trunk and limbs. It is generally slight in degree, greater in the lower than in the upper extremities, and greater in the extensor muscles than in the flexors. Marked local atrophy is uncommon. In severe cases, life may be endangered by paralysis of the intercostals and of the diaphragm, but fortunately one set of muscles has usually begun to recover before the other is seriously affected. The small muscles of the hands and feet and the muscles of the calves and forearms are almost always tender on pressure. They are soft and flabby, and often show a partial reaction of degeneration.

Sensory ataxy is almost always present, and is often severe when the paralysis is trivial. It causes the patient great inconvenience, as it interferes seriously with walking and with the finer movements of the hands. Objective examination reveals sensory impairment of the "glove-and-stock-ing" type. On the hands and feet, the loss to light tactile stimuli is often complete, pain and temperature being less affected. As the limb is accented, sensation gradually becomes normal. Even when the sensibility of the skin is but little diminished, the sensations of position and of passive movement

in the extremities are often seriously impaired, and the sense of vibration is often lost.

In the early stages and for a few days the tendon jerks are exaggerated, but are lost later in every case. Their return is often long delayed, and it is common to see patients months after recovery of normal power, in whom the knee-jerks are still absent. It is common also to find them absent many months after an attack of diphtheria in patients who give no history of nervous symptoms during the attack. The skin reflexes are usually retained, and stimulation of the sole gives a normal response.

Cardiac failure is a grave but uncommon complication. It is of myocardial origin. Vasomotor paralyzes and disturbances in the nutrition of the skin, which occur so often in other forms of peripheral neuritis, are never seen in diphtheria. In those that survive the attack, complete recovery from the nervous troubles always occurs.

4. DIABETIC NEURITIS

In many patients with glycosuria, symptoms are present which point to changes in the peripheral nerves, or in the fibres of the posterior roots. In many respects they resemble *tabes* rather than peripheral neuritis; but as the exact pathology is still unknown, it is convenient to describe them here.

Pathology.—Degenerative changes have been found in the peripheral nerves in some cases, in others these were healthy, whilst the intramedullary portion of the posterior roots showed degeneration, similar to that found in *tabes*.

Symptoms.—In some cases the only symptom is neuralgic pain in the distribution of one or more peripheral nerves. This is commonest in the lower limbs, where it simulates sciatica, and sugar is found in the urine in the absence of any other sign of diabetes.

In severe cases of diabetes the knee-jerks and ankle-jerks are diminished or lost in more than half the cases. This may accompany subjective sensory troubles in the lower limbs, or it may appear as an isolated symptom. The muscles are very often tender and the vibration sense of the feet is frequently absent. To objective examination, the sensibility of the skin is usually intact. Perforating ulcers of the feet have been observed.

Diagnosis.—The diagnosis of multiple neuritis from other diseases rarely presents any difficulty. It is made from the combination of symmetrical flaccid paralysis with sensory loss of the "glove-and-stocking" type, and tenderness of the muscles and nerves, confined to or most intense in the distal parts of the limbs. When sensory disturbances and diminished tendon reflexes are prominent symptoms and muscular weakness is slight, *tabes* may be suggested, and the resemblance is still greater when ataxia is present. Difficulty usually arises when the distinction has to be made between *tabes* and alcoholic neuritis, in a patient who has courted both diseases. In most instances the diagnosis can be made from the nature and distribution of the sensory changes. The lightning pains of *tabes* cannot be mistaken by any one who is familiar with their peculiar characters. Anæsthesia of the extremities is common to both diseases, but diminished sensibility around the

nose and across the chest is peculiar to tabes and is present in almost every case. In neuritis the calf muscles and nerve trunks are tender, whereas in tabes the sensibility of these structures is usually greatly diminished. Hyperæsthesia to touch and temperature, and great exaggeration of the abdominal reflexes, also suggest tabes.

5. ACUTE FEBRILE POLYNEURITIS

Synonym.—Acute Infective Polyneuritis.

At various times small epidemics of a form of polyneuritis characterised by a febrile onset and by the involvement of the facial nerves have been described (Osler, Gordon Holmes, Rose Bradford and others). Nothing is known of its ætiology and it is probably not essentially different from Landry's paralysis in nature, though less fulminating in onset and not so liable to grave involvement of the trunk muscles.

Symptoms.—The onset is with slight fever, headache and malaise, pains in back and limbs, and such general symptoms as a coryza or gastro-intestinal irritation. The fever persists for 2 or 3 days only. A few days then elapse before the signs and symptoms of polyneuritis develop. It is said that the proximal limb muscles are more severely involved than the distal muscles, a point of distinction from other forms of polyneuritis, but this relative incidence of weakness is not invariable and has probably been over-stressed. The trunk muscles do not escape, and the face is usually bilaterally paralysed. As in other forms the paralysis is of the lower motor neurone type, flaccid, atrophic and with loss of tendon jerks. As in Landry's paralysis sensory loss is very slight, and there is relatively slight muscular tenderness. The cerebro-spinal fluid may show a high rise in the protein content, but is otherwise normal.

The clinical course is variable, and sometimes fluctuating in the individual case. Death may ensue from paralysis of the respiratory muscles, and recovery in the majority of cases is fairly rapid. There is the usual tachycardia of polyneuritis.

Treatment.—The first essential in treatment is to remove the patient from the influence of the existing cause. In alcoholic cases, rigid precautions are necessary to prevent secret access to alcohol. To attain this, treatment in an institution is almost a necessity. In most instances when the cause, whatever it may be, is removed, gradual improvement sets in and complete recovery ensues, in a time that varies with the severity of the symptoms. The administration of vitamin B₁ preparations has now a great vogue in the treatment of both multiple (parenchymatous) and interstitial neuritis. In the latter its use has no theoretical justification or practical value, and even in the former, though its use is rational, it yet remains to be proved that the course of the malady is materially influenced. This may be due to inadequacy of dosage in the past, and the parenteral injection for from 2 to 4 weeks of 500 to 1000 units daily is the minimum dosage likely to be efficacious. During this time the physician's most important duty is to prevent the occurrence of deformities and contractures. From the beginning, each joint in the affected limbs should be moved passively to its full range several times each day, and care should be taken to ensure that the attitude of the limbs during rest is a suitable one, especially that the feet are main-

tained at right angles to the legs by the use of appropriate apparatus. Drawing up of the heel must be prevented at all costs.

Gentle massage is soothing in the acute stage. Later, more vigorous rubbing may be given, and the patient should be encouraged to move the limbs voluntarily. Electricity is of no value. Analgesic drugs and soothing applications may be needed at the onset. Thereafter, local treatment to the limbs is combined with measures to improve the patient's general condition. The possible rôle of B avitaminosis in the production of many, if not all, varieties of polyneuritis has naturally led to the administration of the B₁ complex as an important element in treatment. Not less than 1000 to 2000 units daily and given over a period of two or three weeks is adequate, but even with this dosage it is not yet clear that the course of any ætiological variety of polyneuritis is materially influenced, and extravagant claims should be treated with great reserve.

6. LANDRY'S PARALYSIS

In the year 1859 Landry applied the name "acute ascending paralysis" to a case in which acute flaccid paralysis with loss of reflexes and without sensory disturbances commenced in the periphery of the lower limbs, and rapidly spread upwards. The arms were next involved, first in the periphery, and later the trunk, respiratory muscles, neck, and lastly the cranial muscles were involved, and death occurred from respiratory failure. He made a careful microscopic examination of the spinal cord with the methods then at his disposal, and failed to detect in it any morbid changes. He subsequently described this symptom complex, which has since borne his name, from an analysis of 10 cases.

Since this time a large number of cases have been recorded which, from the acute nature of the onset, and from the spreading nature of the paralysis, have been described as cases of Landry's paralysis. This name should be restricted to those cases of acute spreading paralysis, in which disorders of sensibility and sphincter trouble are absent or little marked, and in which recovery is complete if the patient survives, and in which no gross lesion is found within the nervous system after death.

Acute poliomyelitis may also, in rare cases, give rise to a spreading paralysis, and cause much difficulty in diagnosis; but it is invariable that some permanent paralysis remains upon recovery, and, further, the lesions of poliomyelitis are both gross and characteristic.

The majority of authors who have written upon this subject have made the attempt to separate Landry's paralysis from the group of "acute toxic polyneuritis," both on pathological and on clinical grounds. Such a separation would appear to be entirely unsupported by the evidence. In both these conditions all the nervous pathological changes which have been discovered are confined to these lower neurones, motor and sensory, and are often in polyneuritis confined to the lower motor neurones. The clinical separation of Landry's paralysis and polyneuritis is equally artificial and impossible, though much stress has been laid upon the presence of disturbances of sensibility, and the strictly peripheral distribution of the paralysis in polyneuritis. As regards sensory disturbance, this clinical feature is dependent upon the peculiar selective capacity of the poison. The following

description of this malady is based upon the personal observation of 10 cases with 4 autopsies which have come under our observation at the National Hospital and at St. George's Hospital.

Ætiology.—What is known of the causation of the disease in general resembles very closely that of acute polyneuritis. It affects males much more frequently than females, and occurs chiefly in adult life between the ages of 16 and 54 years. The cases which have been reported in children were probably examples of the spreading type of poliomyelitis.

Pathology.—Slight hyperæmia of the spinal cord, and especially of the grey matter, with a few punctiform hæmorrhages, is the only change noticeable upon naked-eye examination. Very definite histological changes are found upon microscopic examination in the anterior horn cells and in the cells of Clarke's column, where any degree of change may be found, from an early pericentral chromatolysis to a complete loss of the chromatin granules and concentration of nuclei.

The cerebro-spinal fluid is in excess, and clear. In two of the cases under our care, it presented no abnormality either as regards cell or albumin content. In other cases there is an excess of albumin, and in this respect it resembles the cerebro-spinal fluid of polyneuritis, which is usually albuminous, and sometimes so highly so as to clot spontaneously.

Symptoms.—The onset is in some cases abrupt, with the appearance of the characteristic spreading paralysis. Much more frequently the paralysis is preceded by certain premonitory symptoms, which may last from a few hours to days or weeks. These symptoms may consist in malaise, headache, lassitude, insomnia, anorexia, constipation, gastralgia, vomiting and diarrhœa, and there is not infrequently slight elevation of temperature. More characteristic still among the prodromal signs are subjective disturbances of sensibility. Pains in the back and limbs are common, and may be of a dull aching nature, or they may be sharp and shooting in character. Numbness, tingling, "pins and needles" and other paræsthesias may occur over any part of the body, and are most commonly complained of in the periphery of the limbs. The muscles may be locally tender during this prodromal stage.

It is not uncommon for the paralysis to commence in the periphery of the lower extremities, to ascend rapidly, and to involve the muscles in the order of their innervation from the spinal cord, the trunk becoming affected before the upper extremities, and the intercostal muscles before the diaphragm. The muscular weakness may commence in any group of muscles, as, for example, in the face, neck, upper extremities or trunk, and when so commencing the spread of the paralysis is downwards, constituting a descending type of paralysis.

In Landry's paralysis, as in acute polyneuritis, the innervation of the respiratory muscles seems to be peculiarly resistant to the toxin.

In those cases which recover the advance of the paralysis ceases, and those muscles which have been most recently affected begin to show some recovery quickly.

When the disease does not prove fatal either from respiratory failure, pulmonary complications or sudden syncope, the paralysis ceases to spread, and the patient enters upon the stage of recovery, which presents many features of interest. The flaccid muscles show a moderate degree of wasting within 2 or 3 weeks on the onset, this wasting being much less in those cases

which recover rapidly. It is a general atrophy, and is not limited to particular groups of muscles. In rare cases, though fair power is regained, yet the muscles remain conspicuously small for life; but generally the muscles recover their bulk and tone completely. The paralysed muscles retain their excitability to faradism throughout, though there may be some slight diminution of faradic excitability in proportion to the general wasting of the muscles. Contractures and deformities do not occur.

The paræsthesias, which have been described with the onset, often persist, and there may be cramp-like pains. Not uncommonly the muscles are tender upon deep pressure; but there is never that severe degree of tenderness met with in some forms of peripheral neuritis as, for example, in alcoholic neuritis. There is exceptionally blunting of sensibility, most marked in the periphery; but this is never deep, and is rapidly transient.

Though from the general weakness of the trunk muscles there may be some difficulty in emptying the bladder and rectum during the first few days, and even retention with overflow incontinence that may require catheterisation from the same cause, yet these last but a few days. The deep and superficial reflexes disappear early with the onset of the first signs of the paralysis in the affected regions. The psychic functions remain unimpaired throughout.

Diagnosis.—The rapidly spreading character of the paralysis in Landry's disease is so striking as to necessitate distinction only from those few maladies in which a similar rapidly spreading paralysis may occur, and these are acute spreading myelitis, intrathecal hæmorrhage, acute poliomyelitis (spreading type) and acute polyneuritis. Acute spreading myelitis is at once distinguished from Landry's paralysis by the severe sensory loss and sphincter paralysis, which in the former condition develop *pari-passu* with the motor paralysis and, further, if the myelitis does not involve the lumbo-sacral enlargement of the spinal cord, an extensor plantar reflex will be observed.

The rare, spreading form of poliomyelitis presents difficulty in diagnosis, especially in the acute stage. The general symptoms and the pyrexia are apt to be more severe in poliomyelitis. An onset in childhood is more suggestive of poliomyelitis than of Landry's paralysis. A fairly high polymorpho-nuclear leucocytosis in the blood, and a lymphocytosis in the cerebro-spinal fluid, are in favour of poliomyelitis. The persistence of local atrophic palsy on convalescence is absolute evidence of poliomyelitis. The distinction of Landry's paralysis from acute polyneuritis is held by the writers of this article to be entirely artificial, since they argue that *Landry's disease is merely a striking type of acute polyneuritis*.

Prognosis.—In about one-half of the cases the paralysis advances until the respiratory and bulbar muscles are involved, and death occurs from respiratory failure, usually on the third or fourth day, but sometimes not until ten days or more have elapsed. So long as the paralysis is extending, and especially when the respiratory and bulbar muscles are failing, the prognosis is very grave. The extension of the paralysis may, however, cease at any stage, and when this occurs the prognosis at once becomes favourable, even though there be considerable involvement of the respiratory and bulbar muscles.

Treatment.—The patient must be placed at complete rest, and the discomfort and panic which are likely to arise from the utter inability to move must be assiduously relieved by frequent changes of posture. The greatest

care must be taken that the patient is adequately fed with nutritious and light food. Stimulants are usually indicated. A mercurial aperient should be administered early and the bowels regularly relieved, for in some cases obstinate constipation occurs. The bladder should be catheterised, if there is a difficulty in micturition. Both pain and pyrexia may be relieved by the administration of salicylates or aspirin.

Atropine tends to check accumulation of secretion within the bronchi. Oxygen may be administered where cyanosis occurs. When once the patient has shown signs that the malady has passed its height, and that recovery is commencing, little treatment is required except careful nursing and feeding. Gentle massage may then be employed.

7. PROGRESSIVE HYPERTROPHIC POLYNEURITIS OF DEJERINE AND SOTTA

Definition.—An extremely rare progressive form of polyneuritis, sometimes developing in infancy, showing an heredo-familial incidence, and characterised by thickening of the nerve trunks due to hypertrophy of the sheaths of Schwann.

Pathology.—The thickening of the nerves may be palpable during life, but is not invariably so. Microscopically this thickening is found to be due to masses of non-nucleated tissue arising from the sheath of Schwann.

Symptoms.—The malady develops and progresses very slowly with weakness, muscular wasting, sensory loss, loss of tendon jerks. There may be noted, in addition, kyphoscoliosis, nystagmus and ataxy of movement. It was formerly thought that the Argyll Robertson pupil was an integral part of the symptom-complex, but this is not the case.

Prognosis.—Death ultimately ensues from intercurrent disease.

Treatment.—There is no known treatment.

NEUROFIBROMATOSIS

Synonym.—Recklinghausen's Disease.

Definition.—A rare and complex disease characterised by multiple benign tumours on the skin and on the nerves and the ganglia of the somatic and autonomic nervous systems. Cutaneous pigmentation, and multiple tumours on brain and spinal cord may also be present.

Ætiology.—The malady is probably developmental in origin, and heredo-familial incidence is not uncommon.

Pathology.—The skin presents spots of varying size and number that have the colour of freckles. When these are extensive they are known as café-au-lait patches. They tend—like the cutaneous tumours—to increase in size and number with age. The skin is adorned with numerous soft fibromatous tumours, some sessile and some pedunculated. In some cases the trunk may be thickly sewn with these. Large plexiform neuromas on cutaneous nerves may form large masses on the skin, flattened and of irregular outline, and when this aspect of the disease is prominent it is given the name of *molluscum fibrosum*.

On the nerve trunks are more or less numerous, fairly firm nodular tumours of varying size. The cranial nerves may also bear similar tumours, especially the fifth and eighth nerves. Marked kyphoscoliosis is common.

Symptoms.—The cutaneous pigmentation and tumours are readily visible and tend to increase throughout life. Sometimes the neurofibromata on the nerves give rise to symptoms of irritation (pain in the distribution of the nerve) or of paralysis. Intrathecal tumours may give rise to spinal cord compression, while the symptoms of an auditory nerve tumour (unilateral or bilateral) may first bring the patient to notice.

Treatment consists in the removal of fibromata on the nerves when these give rise to irritative or paralytic symptoms.

LEAD PALSY

The effects of lead are confined almost entirely to motor neurones. Subjective sensory disturbances are often slight or absent, and in most instances there is no objective sensory loss.

Pathology.—Aub in 1923 showed that the first event was the local concentration of lead in those muscles which were about to be paralysed and that the paralysis was a muscular event primarily, and that, secondarily, the lead ascends along the motor axons and may finally cause the death of the ventral horn cell. The degenerative changes in the nerves are confined almost entirely to the motor fibres, and are most intense in the intramuscular twigs supplying muscles of the extensor groups. Normal and degenerated fibres are found side by side, the former becoming more numerous as the nerve is traced upwards. Degenerative changes due to the action of lead are also found in the affected muscles.

Symptoms.—In most cases of the common *antebrachial* or *wrist-drop type*, paralysis is limited to the extensor muscles of the fingers and wrists—that is, to the muscles supplied by the musculo-spiral nerve. But the supinator longus and the extensor ossis metacarpi pollicis, also supplied by this nerve, usually escape. Inability to extend the first phalanges of the two middle fingers, owing to weakness of the common extensor, is usually the first difficulty. The special extensors of the index and little fingers, the long extensors of the thumb and the extensors of the wrist are next attacked, and the characteristic wrist-drop appears. As a rule the paralysis becomes severe about a week after it is first noticed. By this time it is usually bilateral and symmetrical, but for several days, or even for several weeks, it may be confined to one side. The affected muscles waste rapidly and the back of the forearm becomes flattened, thus rendering the intact supinator longus more prominent. In this form, loss of power always precedes atrophy, and some muscles may show weakness without any wasting. Recovery is almost always complete. Simple weakness without atrophy usually passes off in a few weeks. If the wasting is moderate and the muscles still react to faradism, recovery may be expected in a few months. When the atrophy is severe, a year or more may elapse before recovery is complete.

Occasionally the deltoid, biceps, brachialis anticus and supinator longus muscles are affected, either alone or in company with the forearm muscles—*upper arm* or *brachial type*. Less often paralysis occurs in the legs, the muscles supplied by the peroneal nerve, namely, the long extensors of the toes and the peronei, being chiefly involved—*peroneal type*. Like the supinator longus in the arm, the tibialis anticus, although supplied by the peroneal

nerve, usually escapes. This type is usually associated with paralysis of the forearm muscles, and runs the same course.

In the form of paralysis described above the features are those of a traumatic lesion to a nerve. Loss of power precedes, and may be more extensive than wasting, faradic irritability of the muscles is lost or diminished while the reaction to galvanism is retained, and recovery is usually complete. It is therefore called the degenerative form. In the second form, the paralysis has the characters of progressive muscular atrophy. Weakness and wasting come on together, faradic and galvanic irritability of the muscles are both diminished in proportion to the wasting, and the paralysis is often permanent. This is known as the primary atrophic form. It occurs especially in the small muscles of the hand—*Aran-Duchenne type*—but is sometimes irregular in its distribution and affects many muscles in all four limbs. It is often associated with the first form, but may occur alone. Wasting comes on slowly, and accompanies the loss of power, instead of succeeding it. It is much more intractable than the degenerative form, and often persists after muscles showing the first form of paralysis have recovered. (See also Lead Encephalopathy, p. 366.)

MUSCULAR DISEASES

MYOTONIA CONGENITA

Synonym.—Thomsen's Disease.

Definition.—A very rare malady, commencing in early childhood, which is hereditary and familial, and characterised by a striking slowness in the relaxation of the muscle after voluntary effort. The muscles pass into a spasm on voluntary contraction, which relaxes very slowly, resembling the contraction of the veratrinised frog's muscle, and its subsequent slow relaxation. Peculiar changes in the electrical excitability of the muscle and hypertrophy of the muscle fibres are constant.

Ætiology.—Beyond the facts that the malady is usually hereditary and familial, only a few sporadic cases occurring, and its incidence in early childhood, nothing is known of the causes. Cold, heat, fatigue and hunger conspicuously increase the symptoms.

Pathology.—The affected muscles are actually hypertrophied, and are always firmer to the feel than normal muscles, while sometimes they show a board-like hardness. The individual fibres show considerable hypertrophy.

Symptoms.—The presence of the disease first becomes evident from slowness, clumsiness and awkwardness of movement, with a great tendency to fall if the balance is upset. This is often most noticeable after rest, when, on attempting to move, the limbs seem glued down and move very slowly. Often the patient is able with exercise to work the stiffness off, and the myotonia lessens in the muscles which are being used; but if he is suddenly called upon to put another set of muscles into action, as, for example, by losing his balance, he is at once caught up by the myotonia and so is apt to fall. In other

cases the myotonia increases or is uninfluenced by exertion. The muscles of the legs are as a rule most affected, but sometimes all the muscles of the body may be involved.

Passive movement does not reveal the presence of any rigidity except that following voluntary contraction. The abnormality affects the voluntary contractions and relaxations of the muscles only, and the peculiarities of these are—(1) their slowness, (2) their tonic character, and (3) the continuance of the contraction after voluntary impulses have ceased. The peculiarities of electrical excitability bear the name of the “myotonic reaction” of Erb. The contraction, either on faradic or galvanic stimulation, lasts much longer than the normal and relaxes very slowly, and this is more marked the stronger the current used; with the stable application of galvanism, slow wave-like contractions of the muscle are seen to proceed slowly from the cathode to the anode. There is no pain, no sensory disturbances or loss, and the sphincters and reflexes are unaffected.

Diagnosis.—The only malady which can be confused with Thomsen’s disease is myotonia atrophica, in which the myotonic symptoms and signs are identical. In the latter malady, the onset is at a much later age, the incidence of the spasm is upon local groups of muscles, and the characteristic weakness of the facial muscles and atrophy of the sternomastoids, etc., at once distinguish it.

Course and Prognosis.—Thomsen’s disease has no tendency to shorten and destroy life. It tends to become more marked from infancy to puberty, and then less marked again as age increases. It has never been cured.

Treatment.—This is entirely unavailing, except in the way of the avoidance of those conditions, such as fatigue, cold and hunger, which are known to increase the condition. Thomsen himself, who was afflicted with the disease, was always better with free exercise.

DYSTROPHIA MYOTONICA

Synonym.—Myotonia atrophica.

Definition.—A disease of familial incidence, which begins usually in the third and fourth decades of life, and which is characterised by muscular atrophy of peculiar distribution and unlike that of any other disease. This atrophy occurs first and most in the sterno-mastoids and facial muscles, next in the muscles of the forearms, and may also be found in the muscles of mastication, in the vasti, and in the dorsiflexors of the feet and peronei. Associated with this wasting, but not commensurate with it, nor necessarily occurring in the same muscles, is a peculiar difficulty in relaxing the muscles after effort, called “myotonia,” which gives to this malady an especial feature which at once separates it from all other forms of muscular atrophy. Signs of bodily dyscrasia are often present, the most important of which are cataract, premature baldness, atrophy of testicles, loss of sexual power and general bodily wasting. This disease was first placed upon a firm clinical basis by Batten and Gibb, and Steinert in 1909. Curschmann in 1912 adopted the term *Dystrophia Myotonica* as being more correctly descriptive.

Ætiology.—This condition is probably always familial, and the heredity is homologous—that is, it tends to appear in the same child-rank, in a number

of apparently unconnected families at a common distance from one and the same ancestor, and often it seems to be entirely confined to one child-rank. The descent of the latent tendency is equally through the males and females, but the males more frequently transmit. The presence of the heredo-familial disease in earlier generations is often betrayed by other signs, such as cataract, frequent celibacy, childless marriages, high infant mortality, and a dying out of certain branches of the family. The malady has been observed at the age of 10 years, but usually the onset occurs between the ages of 20 and 35 years. A large number of the patients have been unusually gifted and proficient in athletics prior to the onset. Both sexes may be affected. No exciting causal factors are known.

Pathology.—No definite changes have been found in the nervous system. The muscles presenting the myotonia have repeatedly been examined and found normal. In the atrophic muscles the morbid process singles out certain fibres especially, so that quick and thin fibres are found lying together. There is increase of the muscle nuclei round thick and thin fibres alike, though some atrophic fibres may be found with no increase of nuclei. Recent biochemical and electrographic studies by Brown and Harvey of a form of congenital myotonia in goats suggest that there is no functional disorder of neuromuscular transmission of the motor impulse, but that the disorder is in the muscles themselves.

Symptoms.—The onset is gradual and the course extremely slow. The first symptom to call attention may be, either the difficulty in relaxing after muscular effort—the clinging of the hand to the tool which has been grasped, the smile that is so slow to disappear—or the weakness and wasting of the muscles. The two chief signs of the disease—the myotonia and the wasting—seem to have no connection the one with the other, either as regards coincidence in time or place. The myotonia may appear years before there is any obvious wasting. Moreover, the muscles which show the most conspicuous myotonia are often those which are not wasted, and finally those muscles which waste greatly tend to lose any sign of myotonia which they may have had. The extent and the intensity of the muscular atrophy and of the myotonia show great variations. The atrophy may be widely spread, and many muscles may be myotonic, or the former may be severe and the myotonia slight, or both may be present in minor degree only. Lastly, there are cases in which only the atrophy or only the myotonia is present. The myotonia consists in an inability to relax a muscle immediately after it has been put into voluntary contraction, and the greater the effort used in contracting the muscle, the greater the difficulty with relaxation. The patient grasps one by the hand, and is unable to disengage the hand, but pulls it away still grasping, and it may take seconds to relax. He smiles quickly to a suitable stimulus, and the face remains fixed at the height of the smile for long after the emotion has vanished. In eating, his jaw becomes fixed, he is unable to perform any alternate movements in the muscles which are affected, but at a very slow rate. When the myotonia is severe and general, he is liable to fall like a log when walking, from inability to relax muscles which have been put into contraction. The myotonia is seen most often and to a greater extent in the flexor muscles of the forearm and in those of the face, but it may be quite general. In the same patient it may be very marked at one time and absent at another.

The muscular weakness and wasting usually have a most typical distribution, involving the sterno-mastoids and other muscles of the neck, the facial and masticatory muscles—giving rise to the sad “myopathic” face, the vasti of the thighs, and the dorsiflexors of the feet, and this is the usual order in which the muscles are affected. It is always in one or other of these groups that the wasting commences, but sometimes the sequence of muscles attacked is quite different. Fibrillation does not accompany the atrophy. The electrical reactions show a reduction both to faradic and to galvanic stimuli, with a tendency to a polar change. Some modification of the “myotonic reaction” is often superadded in those muscles which are wasted, and this usually is present in the muscles which are myotonic and are not wasted. This “myotonic reaction” consists in a very long, lasting contraction when the muscles are stimulated with every form of stimulus, and if the latter be strong it may last as long as 30 seconds.

The affection of the muscles of the face and jaw entails some alteration of articulation and phonation. The voice is low, it lacks intonation, and has a definite nasal quality. Sensibility is not affected.

The rule is for the muscle-jerks to be diminished or lost, and it is very rare for all the jerks to be present in any case.

Apart from symptoms and signs connected with the muscles, the most important sign of the dystrophy is cataract, which occurs in more than half of the cases. This cataract is often met with in otherwise healthy brothers and sisters of those who have the muscular changes, and in otherwise healthy members of earlier generations in the afflicted families. In succeeding generations after its first appearance, the age of occurrence of this cataract shows remarkable “anticipation”—that is, commencing at first as senile cataract, it appears at an earlier and earlier age with each successive generation, until with fully developed myotonia atrophica it appears in youth. The presenile cataract of the dystrophic generation begins as a star-shaped opacity, first in the posterior and later in the anterior cortical lamellæ, sometimes with fine point-like opacities scattered through the lens. It ripens quickly to a total soft cataract, with a small central nucleus.

The genital organs remain infantile in some cases; celibacy and childless marriages are common. More often sexuality is normal until the onset of definite symptoms, after which desire and power disappear. Early baldness is the rule. A general wasting of all the tissues of the body is seen in many cases. Ultimate atrophy of the testicles is usual.

Diagnosis.—There is no difficulty in the diagnosis when the distribution of the muscular atrophy is typical and when myotonia is obvious; it simply involves a recognition of the unique characteristics of the disease. When the myotonia precedes the wasting, the age of onset will distinguish this malady from Thomsen's disease, or myotonia congenita, and the oncoming of any sign of facial weakness or muscular wasting will make the diagnosis certain. When the myotonia does not appear until long after the wasting is apparent, the diagnosis is much more difficult.

Course and Prognosis.—This malady usually progresses very slowly, but occasionally very extensive and incapacitating wasting of muscles and weakness may develop within a year of the first symptom. Some cases seem to remain stationary for very long periods. The tenure of life is certainly short in all cases, and does not appear to be prolonged beyond the middle of

the fifth decade. The oldest patient reported in the records as still living was aged 50 years.

Treatment.—It has been found that the administration of quinine, grs. 10 to 15 daily, lessens the myotonia considerably. Neither electrical treatment nor massage has the slightest effect in altering the course of the disease.

JAMES COLLIER.

Revised by F. M. R. WALSHE.

MYASTHENIA GRAVIS

Definition.—A chronic malady of adult life characterised by—(1) a variable paralysis of muscles which is produced or rapidly increased by exertion, and which tends to disappear slowly during rest; (2) a permanent paralysis which shows no improvement with rest, and which succeeds the variable paralysis. This permanent paralysis may be very local in distribution, and may be associated with atrophy of the muscles; and (3) the affected muscles on strong faradisation soon cease to respond to faradism, but remain excitable by galvanism.

Ætiology.—The malady seems to be much more prevalent in England during the past 20 years than formerly. Rarely occurring before puberty, it commences most commonly in the third decade of life, and affects the sexes equally. Nothing is known of any causal factors either immediate or remote. The one clinical association which cannot be ignored is with exophthalmic goitre, for not only may myasthenia follow that malady, but the ophthalmoplegias which occur in Graves's disease bear no small resemblance to those of myasthenia.

Pathology.—The only changes found within the nervous system are slight atrophy of those nerve cells which supply long paralysed muscles, and these changes are certainly not primary. In a certain number of the cases a large persistent thymus gland, showing proliferative and degenerative changes, or thymic rests showing similar changes, have been found. The view expressed in earlier editions of this book that the seat of the disorder of function responsible for myasthenia gravis is at the myo-neural junction has recently received confirmation from observations made with physostigmine and the synthetic substance "prostigmine." It is believed that the normal transmission of impulses from nerve to muscle through the motor nerve endings depends upon the liberation in the endings of acetyl-choline. In myasthenia the failure of effective innervation may be due either to premature destruction of acetyl-choline, or to a failure in its liberation. The administration of physostigmine temporarily delays the destruction of the acetyl-choline by the choline esterase normally present in the blood, and during its period of activity renders muscular contraction normal.

Symptoms.—The first sign of myasthenia is the variable paralysis which may commence in any of the voluntary muscles. It may be unilateral at first, but soon becomes symmetrical. The paralysis appears first upon exertion and fatigue. The schoolmaster finds that towards the end of the day's work he cannot raise his arm readily to write upon the blackboard, or that his voice fails him in speaking. The housemaid finds that her broom

slips in her hands, because of an ever-weakening grasp. The theatre-goer, towards the end of the performance, finds himself tilting his head farther and farther back to escape from an oncoming ptosis, or he develops diplopia. Next morning these symptoms are gone with the night's rest, to reappear with fresh exertion perhaps earlier each day, until work becomes impossible. The affected region may be narrowly confined, the eyes alone, the face, the muscles supplied by the trigeminus, or the larynx alone may be affected, or it may be the muscles of the upper extremities, or of the lower extremities, or of the back which may be solely involved. Lastly, the myasthenia may be quite universal, though never in the same degree in all the affected regions.

The incidence is greatest upon the muscles innervated from the brain stem, next upon the upper extremities and trunk, and least upon the legs. When the initial incidence of the malady is upon the eye muscles, diplopia and ptosis are the first symptoms. The paralysis is usually of the nuclear type, involving both eyes in terms of the conjugate movements; but inequality in the paralysis upon the two sides usually gives rise to decided strabismus and diplopia, and we have observed one case in which the initial paralysis was confined to one external rectus. When the permanent paralysis sets in, the axes usually become parallel, and the diplopia ceases. Until this event occurs, the great feature of myasthenic ocular paralysis is its variability, and its increase with fatigue. An ocular paralysis which is well each morning on waking, and which develops in the course of each day, is always due to myasthenia. Short, very quick, jerky movements of the eyes on attempted voluntary movements are often characteristic, and are quite different from the movements of nystagmus. Though ptosis is the rule, sometimes there is retraction of the lids, and both von Graefe's and Stellwag's signs may be present. Permanent nuclear ocular paralysis follows the variable paralysis in nearly every case, though it varies in degree. When the face is affected, epiphora, dysarthria and lack of facial expression with a peculiar weak "nasal smile" are conspicuous. The facies of myasthenia, with inability to close or pucker the eyes and mouth, the motionless and slightly dysarthric speech and peculiar smile are unmistakable.

Involvement of the muscles supplied by the fifth nerve causes trouble with mastication, and when the palate is affected there may be nasal speech and regurgitation of liquids. We have seen one case in which total unilateral paralysis of the larynx preceded the onset of typical symptoms by twelve months. When the tongue is affected it usually shows some wasting, especially of the linguales, and dysarthria results. Sometimes a widely spread involvement of this region causes severe dysphagia and dysarthria, and the former may be so great as to necessitate nasal feeding. The permanent paralyses are rarely seen, except in the muscles supplied by the nuclei of the brain stem. The neck muscles are usually affected when the malady extends on to the trunk, and it is a common thing to see the patients sitting at rest, either with the head supported by the hands, or resting upon the table. In the extremities the variable paralysis appears more marked at the proximal joints in most cases, and the test for myasthenia in the upper extremity is to ask the patient to extend his upper limbs level with the shoulder, when they will be seen to fall slowly down from the increasing fatigue paralysis.

Involvement of the respiratory muscles is common, and constitutes the gravest danger in the disease, since any effort, and especially an emotional

outburst, may in a few seconds fatigue the respiratory muscles into a complete and fatal paralysis. Myasthenia often remains long confined to one region, and subsequently spreads rapidly. Wasting of the muscles occurs only when there is marked permanent paralysis, and may be seen in the muscles of the tongue, face and in the masticatory muscles. In one fatal case under our care there was marked wasting of the intrinsic hand muscles on both sides.

The myasthenic reaction is only seen in those muscles which are showing conspicuous fatigue paralysis. If a strong interrupted faradic current is applied to such muscles, there is at first a strong contraction, but this is not maintained as in normal muscles, and it rapidly decreases until there is no response. If the stimulation is discontinued for a few minutes, and again applied, there is a further response, which tires more rapidly than the first. After exhaustion of the muscles by faradism, some volitional contraction remains. Exhaustion of the muscles does not occur from galvanic stimulation. Sensibility and sphincter control are not affected. The reflexes are normal in all but the rarest cases. The knee-jerk is not abolished when the quadriceps is exhausted, either by voluntary exertion or by faradisation.

Diagnosis.—This is never a matter of any difficulty if the variable paralysis, increasing with fatigue and lessening with rest, is conspicuous, for this phenomenon occurs in no other disease. When permanent paralysis only is present, the diagnosis requires care. It must be remembered that any unilateral or bilateral palsy of muscles supplied by the brain stem may be myasthenic. Here the history of a slow onset with variable paralysis and fatigue phenomena can nearly always be obtained, and the absence of the usual signs of gross lesions of the brain-stem nuclei, or progressive diseases affecting the latter, should avoid confusion. When, as sometimes happens, myasthenia begins with a unilateral ophthalmoplegia or laryngoplegia, the diagnosis may really be difficult. The possibility of such a commencement should be borne in mind, and a careful watch kept for the appearance of conclusive evidence. Other forms of nuclear ophthalmoplegia do not show a long history of variability and fatigue phenomena.

Course and Prognosis.—Myasthenia is always a very dangerous disease, as the term "gravis" implies. Some of the cases, and especially those in which the brain-stem region escapes, recover completely; but no records are as yet available to prove in what proportion of the cases this event occurs. The disease has proved fatal within a fortnight of the onset of symptoms, and, on the other hand, in our two cases here recorded, strenuous work was followed for 24 years after the development of permanent ophthalmoplegia. Improvement and relapses are very common. Frequently a patient will get rapidly worse, and become bedridden, in spite of careful treatment; to recover completely for the time being, when treatment has been abandoned as useless. Sometimes a patient with severe myasthenia will live for many years, if life be carefully protected. We have had one patient under observation in this state for 20 years, and she is not materially worse.

Sudden death is very liable to occur in any of the cases, but especially in those with bulbar paralysis and implication of the muscles of respiration. Death has been attributed to respiratory failure and asphyxia; but some of the patients die much too quickly for any such explanation. Two of our patients, seated at a table with their heads supported by their hands, and

engaged in pleasant, quiet conversation, smiled, lowered their heads on to the table, and were dead without the slightest sign of distress or reaction, as if from syncope.

Treatment.—The life must be so ordered as to exclude all fatigue. Massage, electrotherapy, endocrine therapy and strychnine medication are ineffective when not harmful. Claims have been made for many different drugs and preparations, but they are based upon an ignorance of the fluctuating course of the malady and of its occasional spontaneous cure. Few things in clinical medicine can be more dramatic than the rapid and complete disappearance of all weakness and disability upon the hypodermic injection of prostigmin 2 to 4 c.c. (1 to 2 mgms.) combined with atrophine sulphate, gr. $\frac{1}{100}$. This abeyance of symptoms endures for some four or five hours only. When the effect passes off the patient lapses into his original state, or may even be weaker than before. In severe cases, this possibility of increased weakness after physostigmine or prostigmin administration has its dangers. It is not yet possible to use these drugs by repeated injection as a mode of treatment for myasthenia gravis, though their occasional administration may enable the subject to meet special calls from time to time. The drug may be given orally in tablets of 15 mgms., four to eight daily.

A few patients maintain an improved level of power in the affected muscles when given ephedrine sulphate or chloride (grs. $\frac{1}{2}$ to 1, t.d.s.).

FAMILIAL PERIODIC PARALYSIS

Definition.—A flaccid paralysis affecting the muscles of the trunk and of the extremities, associated with loss of the deep reflexes and diminution or loss of faradic excitability in the muscles. The paralysis is temporary in character, though it may be fatal during the attack, and it recurs at intervals. It is a rare malady, some 200 cases having been reported in the literature.

Ætiology.—It has been noted as early as the fifth year, and as late as the thirtieth year; but usually it appears about the age of puberty. Most of the cases occur in the male sex. Heredity is very marked, and the malady has been traced through five generations. Transmission may occur either through the male or through the female, and not infrequently a generation is skipped. Several members of the same family are usually affected.

Pathology.—Several cases have come to autopsy, but no lesion which could be associated with the symptoms was found. Biopsy of the muscles has given entirely negative results.

Symptoms.—The clinical picture is so striking as to be almost dramatic. The patient retires to bed feeling perfectly well, and awakens in the morning without pain or malaise, but with a flaccid motor paralysis, which always involves all four extremities, and which may reach all the muscles of the body, except those of the organs of speech, deglutition and respiration, and even these are often partially involved. Severe involvement of these vital muscles during an attack has caused death. The bladder and rectal functions are retained, and it is unusual for the patient either to void urine or feces during the attack. The paralysis is usually at its height on waking; but it may subsequently increase. After lasting for a variable time, from a few hours to

a few days, it passes off, sometimes gradually, sometimes rapidly. In the family under our care it was astonishing how the patients on waking in an attack could judge invariably how long the particular attack would last. They could judge with unfailing certainty when ability would return, and were in the habit of arranging their business accordingly. Most of the patients in addition to the severe attacks of paralysis suffer from what they call "morning weakness," temporary inability to grip with the hands, and slight disability with the feet on waking. It is curious that similar morning weakness, lasting a few minutes, is not very uncommon in normal children. The paralysis in periodic paralysis is flaccid, and there is loss or marked diminution of response to faradism during the paralysis. The deep and superficial reflexes are lost in the paralysed region. Objective sensation is not affected; but there may be subjective sensations of tingling and numbness, and the muscles may be a little sore and stiff after the attack. We have noticed flushing of the surface and sweating during the attack. There is an invariable tendency for the attacks to diminish in frequency and severity after middle life is reached.

Diagnosis.—This must be evident to any one acquainted with the symptoms of the disease.

Treatment.—Potassium chloride in large doses (up to 30 or 40 grs.) will avert or cut short an attack. No other remedial measure is known.

MUSCULAR DYSTROPHY; MYOPATHIC ATROPHY

Synonym.—The myopathies.

Under this heading, a disease is described in which the voluntary muscles undergo primary degeneration, independent of detectable disease in other parts. To facilitate description, a number of clinical types have been distinguished according to the age at which the disease appears, to the group of muscles first attacked, to the presence or absence of pseudo-hypertrophy, or to the prominence of the hereditary factor. The chief of these are—(1) the pseudo-hypertrophic type; (2) the juvenile type of Erb; (3) the facio-scapulo-humeral type of Landouzy and Dejerine; (4) the distal type.

The first type is fairly constant, but there is in reality no sharp division between the different forms. That the others do not represent separate diseases is proved by the appearance of more than one of them in members of the same family. The disease is familial, and it is also hereditary in the sense that it may appear in some or all the members of a family through several generations.

The changes in the muscles in the myopathies are the same as those which occur when muscles degenerate from any other cause, namely, a slow and progressive atrophy of the contractile elements, with a concurrent increase of fat and fibrous tissue. In the pseudo-hypertrophic form the connective-tissue hyperplasia is excessive in some of the affected muscles and their bulk is increased. In the other forms of the disease, and in those muscles in the pseudo-hypertrophic form which become weak without any increase in size, the overgrowth of connective tissue may balance the loss of bulk due to atrophy of the contractile tissues, and the diseased muscles retain their normal size; or atrophy may proceed faster than hyperplasia, and the muscles waste from the beginning.

1. PSEUDO-HYPERTROPHIC PARALYSIS

Ætiology.—The cause of the disease is unknown. In many instances no antecedent cases can be traced in the family. In others, a family history is obtained, always on the mother's side. Isolated cases occur, but more often several children are attacked in each generation. Boys suffer more frequently than girls in a proportion of about 5 to 1. Sometimes one sex alone suffers, sometimes both. It is rare for all the children to be attacked. The males who escape beget healthy children, whilst the females, who appear to have escaped, may transmit the disease to some of their offspring.

Symptoms.—The symptoms appear in early childhood. The onset is often delayed to the fourth or fifth year, rarely until towards puberty, and very rarely until as late as the twentieth year. In cases of late onset, enlargement of the calves has usually been present for many years. Weakness appears first in the muscles of the pelvic girdle. The child, who usually looks fat and strong, begins to walk late, he falls easily, and rises again with difficulty. He does not romp as other children do. He cannot skip or jump, and he has great difficulty in mounting stairs. At first the muscles may be normal in size, but, as a rule, some show obvious enlargement before the fifth year is reached. The enlargement is most conspicuous in the calves, the buttocks and the infraspinati. The erector spinæ, the quadriceps in whole or part, the deltoid, the supraspinatus and the triceps often show considerable hypertrophy. Occasionally the masseters are enlarged. At the same time other groups of muscles atrophy. This is most severe and most frequent in the latissimus dorsi and in the lower part of the pectoralis major. Later it extends to other muscles, and ultimately to those which were at first hypertrophied. The neck and face are spared. There is no exact correlation between the size of the diseased muscles and their power, but weakness is usually greatest in those which show most atrophy. The defects are greater in the proximal muscles, and diminish distally. The hands often retain good power to the end. This distribution of paralysis gives rise to certain characteristic defects of attitude and movement.

In standing the legs are placed far apart, and the upper part of the trunk is thrown back, so that a plumb-line from the most prominent vertebra falls behind the sacrum. This attitude compensates for the forward tilting of the pelvis, resulting from weakness of the glutei, which normally raise the anterior border of the pelvis by lowering its posterior border. In the sitting posture the lordosis disappears, for now the attachments of the flexors of the hip are approximated, and these muscles no longer lower the anterior border of the pelvis. On lying down the lordosis appears again, but can be abolished by relaxing the flexors of the hip-joint, that is, by flexing the hips passively. In walking, the feet are widely separated, and to clear the ground with the advancing foot the body is inclined first to one side and then to the other. This "waddling" produces a gait like that seen in congenital dislocation of the hip. The early preponderance of weakness in the extensors of the hip and knee is betrayed by the great difficulty experienced in mounting stairs.

The manner in which the child rises from the supine to the erect position is almost pathognomonic of the disease. He first tries to sit up, but fails. He then rolls over on his belly, and raises himself first on his knees and elbows, and then on his hands and feet. Next he places his hands on his

knees, and as it is impossible for him to raise the trunk actively owing to weakness of the extensors of the hip, he literally climbs up his thighs, pushing the trunk passively almost to the erect position. The remaining power in the extensors may be enough to enable him to complete the movement. If not, he jerks the shoulders back suddenly and gains the erect posture by a writhing movement, whose details are difficult to follow. To climb the thighs successfully a certain amount of power is necessary to hold the knees slightly flexed. When this power is lost he is no longer able to rise. The arms are also used to assist the weak legs in sitting down and in getting up from a chair.

As time goes on the weakness increases, and invades all the muscles of the trunk and limbs. Some of the muscles become shortened, and distortions are produced by permanent alterations in the position of the joints. The knees and elbows become flexed, the feet take up the attitude of talipes equinus, the spine becomes curved, and the whole body is grossly deformed.

The deep reflexes and the electrical excitability of the muscles diminish gradually as the wasting increases. Sensation is unaffected. The sphincters are not involved. The mental condition shows no abnormality.

Diagnosis.—The diagnosis is usually simple if a few of the outstanding features of the disease are known. The defects of attitude and movement, especially the mode of rising from the supine position, together with the characteristic association of enlargement of the infraspinati and calves with atrophy of the latissimus dorsi, form an unmistakable combination.

Prognosis.—This is most grave. Few patients reach adult life, and most die within 10 years of the onset of the disease.

Treatment.—Drugs have no beneficial influence. Massage and passive movement are useful in the prevention of contractures, and the efficiency of the muscles may be prolonged by suitable exercises. Walking should be practised daily, until it becomes impossible. Very often this is lost owing to contractions of the calf muscles, and is regained after tenotomy.

2. OTHER TYPES OF MUSCULAR DYSTROPHY

Ætiology.—The separation of the remaining types of myopathy from the pseudo-hypertrophic form is not an absolute one, as isolated cases are occasionally met with which seem to form a connecting link between the several varieties. The varieties, however, are habitually separate in occurrence, and in families in which numerous cases conforming to the types to be described hereunder have occurred throughout several generations, no cases presented the peculiar features of the pseudo-hypertrophic form. Moreover, the sex incidence as well as the period of onset is different in the two varieties, and it is possible that there is some essential pathological difference between them, and that they are separate diseases. With regard to the types of myopathy unassociated with pseudo-hypertrophy, no doubt exists as to their fundamental unity. They are merely varieties of one disease.

The influence of heredity is much more prominent than in the pseudo-hypertrophic form. Isolated cases occur, but they are rare. In most instances several members of a family are affected in the same and in succeeding generations.

The sexes suffer equally. The time of onset varies within wide limits—

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from infancy to old age. When the wasting begins in the face (*facio-scapulo-humeral type*) the disease frequently begins in childhood; but sometimes it begins there late in life. In the cases where it is first noticed in the muscles of the shoulder and pelvic girdle the onset is most frequent between the ages of 15 and 35 (*Erb's juvenile type*); but here, again, it may begin in childhood or early old age, and the term juvenile is hardly applicable to it. The same variations in the age of onset are noticeable in cases where the atrophy begins in the forearms and legs (*distal type*).

The various types may be exemplified in members of the same family, and in the same family the age of onset may show extreme variation.

The cause of the disease is quite unknown.

Symptoms.—In the so-called juvenile form weakness and wasting come on simultaneously. In most cases they are first noticed in the arms; but in some families the legs suffer first. Of the arm muscles the biceps, triceps, and supinator longus are most often first affected. The lower part of the pectoralis major, the latissimus dorsi, trapezius and rhomboids are attacked in most cases. Atrophy of the serratus magnus is common; but it may escape even in severe cases. The deltoid, supraspinatus, infraspinatus and subscapularis usually escape. Atrophy of the forearm and hand muscles is rare.

In the legs, the flexors of the hip, the extensors of the knee and the glutei are most frequently affected. The muscles below the knee often escape entirely.

In the face the zygomatic muscles and the orbicularis are attacked. The face is dull and expressionless, the naso-labial fold is obliterated, the lips are habitually separated and the lower lip projects—myopathic facies. The face does not light up in conversation, in blinking the eyes are incompletely closed, and the articulation of labial consonants is defective. In smiling the mouth forms a straight line, instead of its angles being drawn upwards and outwards by the zygomatici. The power of whistling is lost. When the patient closes his eyes, or compresses his lips as forcibly as he can, they can be forced open with great ease. The buccinators are often affected, the tongue and the masticatory muscles never. The spinal muscles often atrophy, and in a few cases the abdominal muscles have been involved. The excitability of the muscles to faradic and galvanic stimulation usually diminishes in proportion to the wasting. The muscles never show fibrillary tremors. Sensibility is unaffected, and all the other functions of the nervous system are normal. Deformities are neither so common nor so severe as in the pseudo-hypertrophic form.

Diagnosis.—When a family history of atrophy is obtained, myotonia atrophica and peroneal muscular atrophy must be excluded. Myotonia atrophica is distinguished by the peculiar prolonged response of some of the muscles to voluntary, electrical and mechanical stimulation, and by the distribution of the wasting. Atrophy of the sternomastoids, which is constant and severe in myotonia atrophica, is never seen in the forms of myopathy now under consideration. In peroneal muscular atrophy the combination of atrophy in the lower limbs and small muscles of the hands, together with sensory disturbances in the lower limbs, is distinctive. In an early case, when the hand muscles are still normal and sensory changes are absent, the differentiation from myopathy may be impossible for a time.

In isolated cases myopathy is suggested by the appearance of muscular atrophy in childhood or youth. The diagnosis of myopathy is based on the distribution of the wasting in the absence of any sign of disease of the nervous system.

Prognosis.—The disease shows wide variations in its course and duration. The atrophy may remain confined to the group of muscles in which it begins, or extension may take place after an interval of several years. It rarely extends beyond the muscles mentioned above. In most cases, even in those that begin in childhood, progress is extremely slow, and as no symptom of the disease is necessarily fatal, death usually results from other maladies unconnected with the disease.

Treatment.—Owing to the variable course of the disease, it is impossible to estimate the value of any treatment that may be employed. Massage, and especially voluntary exercises designed to bring the weakened muscles into play, seem sometimes to retard the progress of the disease.

AMYOTONIA CONGENITA

Synonyms. — Oppenheim's Disease ; Myatonia Congenita.

Definition.—A malady of early childhood, usually congenital and sometimes familial, characterised by extreme flaccidity, smallness and weakness of the muscles, which are not actually paralysed, by lowering of the faradic excitability of the muscles, by loss of the tendon jerks, and by contractures in the region affected.

Ætiology.—In most cases the disease is present at the time of birth ; in a few cases it has appeared during the first year of life in an apparently healthy child, and sometimes following an acute illness, such as bronchitis or diarrhoea. Usually sporadic, it has occurred in several children of the same parents.

Pathology.—The chief morbid changes are found in the muscles. In these very conspicuous pathological conditions are present, closely resembling those found in the myopathies. The three most striking conditions are—(1) the minute size of the majority of the muscle fibres, from 7μ to 12μ ; (2) the presence of a few very large or "giant" fibres reaching 140μ in diameter, and larger than any fibre occurring in normal muscle ; (3) marked regressive changes are seen in the giant fibres. There is increase of the connective tissue between the muscle bundles and a notable determination of fat. Reduction in numbers of the ventral horn cells of the spinal cord occurs, and the ventral roots are small and poorly myelinated.

Symptoms.—The extreme flaccidity of the affected muscles is noticed from the time of birth. They are small and weak, and though there is no muscular wasting and no absolute paralysis, yet in many cases the limbs cannot be raised against the action of gravity, nor can the head be held up. The great relaxation of the muscles and ligaments allows of the most fantastic attitudes being assumed without pain. When the child gets older, he is unable to sit up, but when placed in the sitting position the spine bunches up from absence of any muscular support, and he is unable to support his weight upon the weak legs. The amyotonia is symmetrical, and affects the legs always, the trunk often, the arms not infrequently, but never the face.

Notwithstanding the flaccidity, some degree of flexor contracture is usually present. The faradic excitability of the muscles is much lowered, but not lost. Sensibility and the sphincters are not affected. The superficial reflexes are normal, but the deep reflexes are invariably absent in the affected regions. The children are usually intelligent, with good bodily development, and growth proceeds normally.

Diagnosis.—This presents no difficulty on account of the presence of the flaccidity at birth, the absence of the deep reflexes, and the tendency slowly to improve. It has to be separated from those maladies to which it bears a superficial resemblance, namely, the myopathies, rickety weakness, obstetrical, infantile and diphtherial palsies.

Course and Prognosis.—Some of the children succumb during the early and severe stages of the disease, but the tendency of the disease is to improve slowly in the course of years, and in some cases almost complete recovery with return of the knee-jerks occurs.

Treatment.—This consists in aiding the natural tendency to improve with massage, passive movements and exercises, in treating contractures with tenotomy, and in attending to the general health and nutrition.

JAMES COLLIER.

W. J. ADIE.

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SECTION XXI

PSYCHOLOGICAL MEDICINE

INTRODUCTION

PSYCHIATRY is concerned with forms of illness as widespread and diverse as those of somatic medicine. There are almost as many beds in mental hospitals as in all other hospitals put together ; and there is an undoubtedly large, if unnumbered, part of the population who have mild mental disorder not needing mental hospital care : hysteria, obsessional neurosis, hypochondria, chronic depression, paranoid states, and so forth. The diversity of this widespread group of illnesses depends on their being disorders of mind—disorders, that is, of the human function which comprehends and sums up all other functions of the organism, serves to relate a human being to his complex environment, and is the chief token that he is an individual, and not a sample. Mental disorders are therefore varied, as are the people who suffer from them. It is only by ignoring most of what is individual in these illnesses that a few common types or categories can be recognised, comparable to the “diseases” of somatic medicine. Such a procedure is necessary for practical ends ; material must be classified. Moreover, a biological foundation may be assumed for the syndromes with which psychiatry works. They stand for the main ways in which a human being can become mentally unhealthy. There are only a few such ways, and they are determined by the structural and functional patterns inherent in the organism. Diversity arises through their becoming manifest under the influence of each individual's special environment and in combination with his other inherited tendencies. Diversity, therefore, can be due to a combination of single hereditary causes and to the effect of each individual's environment throughout his life upon his development and behaviour. There is always interplay between inheritance and environment. Part of the psychiatrist's business is to discover how this interplay has led to the present illness. The interplay, moreover, is sufficiently varied in the course of each patient's life to make prognosis and the effect of treatment a matter of individual study, rather than of summary inference from the diagnosis, once made.

Treatment is only another special instance of the environment acting on the patient ; its power and limitations for him must be studied by seeing what effects this or that experience has had on his previous life. Consequently the psychiatrist, even more than the general physician, must study illness in two ways : first, as showing some typical pattern of morbid behaviour, and tending to run along well-known lines ; and secondly, as a patch of personal biography, something to be understood, rather than classified in terms of psychology and biology. The two methods are complementary,

though, in a brief text-book presentation, the former must be the more prominent.

There is no dividing line between somatic medicine and psychiatry. Psychiatry, although it has to work in part with social and psychological conceptions of which general medicine has felt little need, suffers greatly when it limits itself to this way of regarding mental phenomena. It cannot safely ignore the relationship between bodily happenings and the patient's state of mind. Crude instances of this relationship are the delirium that accompanies an acute fever and the irritable fatigue (*neurasthenia*) that may follow it; the insanity that is due to cerebral tumour or general paralysis of the insane; the obsessional neurosis that follows *encephalitis lethargica*; and the hysterical symptoms of disseminated sclerosis. There is no mental disorder, mild or severe, in the causation of which bodily disease may not play an important part. Moreover, it is not only in crude instances of structural or chemical disease that the relationship between bodily and mental illness may be recognised. A human being does not exist as a rarefied mind united with a solid body; he is an organism all of whose subsidiary functions contribute to this highest function—his mind—which brings him not only consciousness, but also an integrated behaviour in relation to his surroundings. Disturbances, transient or permanent, of these part-functions (for example, in the sensory apparatus or the circulatory system) will have some effect on his state of mind. Changes in the central nervous system are the most obvious instance of this, but the endocrine glands, the autonomic nervous system, and the metabolic processes are often of notable significance in the various maladjustments summed up as mental disorder. A human being is constantly responding to, and influencing, his surroundings; but his doing so is conditioned by the various parts of his body and the way they are working.

Before the categories and clinical features of mental illness are described, the principles of psychopathology, prognosis, and treatment call for some very brief consideration, since without them psychological medicine written down is a repellent catalogue of details. Though the principles set forth may seem trite or too obvious to be worth stating, it is unfortunately the case that they are seldom applied as fully as they might be to the clinical study and treatment of mental disorders.

Psychopathology.—**INTRINSIC CAUSES.**—The intrinsic causes of mental disorder are those which depend on heredity and on normal phases of development, *e.g.* puberty or the climacteric. Extrinsic causes, which come from the environment, are either mental experiences or physical damage. The distinction between intrinsic and environmental, like that between physical and mental, is convenient but artificial; a long sequence of related happenings both within and without the patient's body goes to the causation of any mental illness. It is, of course, possible in many instances to discover some indispensable link in this chain of causes—an intoxication with alcohol, for example, a syphilitic infection of the brain, an inherited predisposition to periodic insanity, a bereavement—which may legitimately be singled out as the chief cause and classified as intrinsic or extrinsic, but this is more valuable for formal and didactic purposes than clinically. Actual cases usually show a complicated *ætiology*. Thus, a man whose parents had both been subject to melancholia became himself profoundly depressed after the death of

his wife, and attempted suicide by drowning. He survived, but during the resulting pneumonia he was delirious and threw himself from an upper window, crying out that he must go to his wife. The causes of the mental disturbance in this case were many and obvious; numerous they always are, but not always obvious. One cause may, of course, be prepotent.

The more detailed the analysis of a patient's endowment and experiences, the more entangled physical and psychological, internal and external factors seem to be.

Heredity and constitution.—The hereditary factor is not a general neuropathic taint; there are specific predispositions to one or other anomaly. These predispositions are transmitted in accordance with familiar genetic principles, summed up in the modern gene-theory of inheritance. Studies of families and of twins have proved the importance of the hereditary factor in the major non-organic psychoses, though they have not yet sufficed to reveal the number of genes concerned in the transmission of the hereditary types of morbid reaction.

Among the main reasons for this incompleteness in our knowledge is the impossibility of concluding that an inherited trait is not present, merely because it is not manifest in some recognisable form. Other inherited factors and, most of all, the environment, will in many cases determine whether an individual predisposition is to become evident or not. Thus a man may have an inherited tendency to melancholia which remains latent until a financial reverse or disease of the cerebral arteries provides the conditions necessary for its manifestation. It is true that some inherited predispositions, *e.g.* to Huntington's chorea, are almost independent of the environment in this respect, but such are exceptional.

Since the Mendelian and other conceptions of modern genetics are held to obtain for the heredity of mental disorders, it can be understood that more than one type of proneness to mental disorder can be inherited by the same person. He may, for example, be prone not only to periodic insanity, but also to schizophrenia. Mingled proclivities of this kind account for anomalous clinical pictures, frequently met with and difficult to classify as either one syndrome or another. The "either-or" kind of diagnosis is often out of place or misleading in psychiatry because of the commonness with which more than one constitutionally rooted type of illness may be found in the same patient. Syndromes are frequently combined; to grasp their clinical meaning it may be indispensable that one investigate the patient's family not only as to mental disorder, but as to normal characteristics too.

The signs of a transmissible tendency to some mental disorder may not be actual illness, but only a special kind of personality. There are certain varieties of personality which show some or all of the essential features that characterize certain types of mental illness; the differences between personality and illness seem then to be of degree rather than of kind. Moreover, those who manifest one or other type of illness are often found to have had the type of personality that is functionally similar to it. So close may the similarity be that it is difficult to decide when the illness has begun, because there was no sharp dividing line, in time or in form, between the more or less normal previous personality and the actual disorder. This frequency of association and similarity of form

between the normal state and the illness points to the constitutional background of mental illness, and shows how hereditary tendencies can express themselves in more or less normal ways in personality before the catastrophe of an obvious illness has directed attention to them. Nor is it only in the personality that inherent proclivities may be revealed; certain types of bodily structure, too, occur much more frequently in those with a particular mental constitution or mental illness than in the rest of the population. The most striking instance of this is the frequency with which a "pyknic" bodily habit and a "syntonic" personality are found among those who have periodic attacks of mania or melancholia (see p. 1855). Such constitutional features, whether mental or physical, indicate that inherited tendencies can body themselves forth in normal physical and psychological structure before morbid exaggerations of them make an appearance. The varieties are sometimes called by appropriate names, *e.g.* schizoid, cyclothymic, syntonic, obsessional, hysterical, paranoid. The relationship is not a simple one. There are very many people with these types of personality who never fall mentally ill.

A pronounced personality, belonging to one or other of these types, does not indicate that the person who exhibits it is likely to have a mental illness, but only that if he should have a mental illness, it will probably be of the corresponding type. As with all inherited anomalies of which the crude manifestation is delayed until adult life, there may be for many years none or only mitigated signs of the proclivity; these may be indistinguishable from what occurs in normal people. The more pronounced the anomaly of personality, the more likely that it portends a mental illness, or, at any rate, a proclivity to the mental illness in specially adverse circumstances.

In the foregoing, personality and constitution have been spoken of as though they were static, innate attributes of the human organism. Neither of these epithets, however, is appropriate, not even in respect of bodily constitution. Responsiveness and plasticity are essential to human development of every kind; there is a constant interplay of personality with the outer world, modification of it and by it. The main pattern of development is doubtless determined by innate, inherited factors—bodily structures grow, instincts come into play, and the general direction of functional activity is predetermined. But general directions and main patterns mean little unless they are given body and content by individual experience. Nutrition, for example, can deflect the body from its ordained pattern or enable its fulfilment; all sorts of physical interference can maim it or improve it: the same is profoundly, if obviously, true of the mental side of human growth and maturity. Consequently, each patient's personality is not only to be assessed as conforming to a frozen artificial type, but as a complex of dynamic functions, changing in outward form, sometimes in unstable equilibrium, and none the less powerful for being subterranean. Here, as was said earlier of psychiatry in general, there must be two ways of viewing the data: in classes, and as individual living biographies to be understood rather than schematised. Both methods are necessary to any complete psychopathology.

Phases of development.—A concrete instance of the foregoing is the change that occurs at certain turning-points, such as puberty, pregnancy, or the climacteric. Endocrine and other physical changes at these epochs are

accompanied by psychological disturbances, the severity and form of which may bring them under the notice of the psychiatrist. They are dramatic episodes in a lifelong and universal process of growth, maturity, and involution or decay, which is marked by plasticity and development of varied functions in the first stage, stability and differentiated adaptation in the second, emotional lability and suspicion, intellectual narrowing of interests, rigidity, failing grasp and memory, in the last. The mental disturbances which may occur at different ages are much influenced by these intrinsic factors and tendencies.

EXTRINSIC CAUSES.—The outer world impinges on human beings from the day of their birth, or even their conception, in more and more complicated ways, as they themselves become more complicated. In other words, the environment is, for the individual, as complicated as he can make it; and that will depend on how far he has himself developed hitherto. Human beings deal selectively, not merely passively, with experience. At each stage of their growth, previous experience helps to determine what they will select from their environment, and how they will use this and integrate it, to serve in its turn as the partial determinant of further growth and integration (the other determinants being innate ones). "Experience" is here being used in the widest sense to denote the response of a human being to the impact of the outer world upon him, whether it be consciously recognised as such or not at the time.

It is, therefore, impossible to give adequate consideration to any aspect, including the psychological, of a human being's way of dealing with the outer world unless one pays regard to his previous experience, mental and physical, and to the present state of his whole organism, mental and physical. The cultural milieu in which he has grown up must be taken into account. Too partial a regard for subsidiary functions, whether physiological or psychological, may lead one away from the living human being, who is an integrated organism, not a collection of disparate mental and physical systems; similarly, too concentrated a gaze on this latter aspect, *i.e.* on the socially organised person, to the neglect of part-functions, may make one see only a disembodied spirit, as remote from medicine as from daily life.

Physical experiences.—Some external happenings influence the mental state chiefly by way of the body: infection, physical trauma, intoxication, and metabolic and endocrine disturbances due, wholly or in part, to environmental influences may result in mental disorder. In many of these instances, the mental change is mediated by way of some cerebral damage, and the clinical picture is of the organic neurogenic kind, *e.g.* dementia. It would be wrong to attribute the whole of the mental disorder to the cerebral damage; but to it is referable the core of the psychosis. Some diseases have an incidence on special functions and parts of the central nervous system, which determines characteristic features in the mental picture, *e.g.* the anxious fidgetiness of the patient who has had chorea, the stiff mind and obsessional thoughts and movements of the post-encephalitic, the hysterical phenomena of the elderly arterio-sclerotic patient or the man poisoned with carbon monoxide, the aphasia and apraxia of the post-apoplectic, the silly "moria" of the cerebral tumour. In the main, however, it is not possible to correlate mental symptoms with special areas or kinds of cerebral damage—partly because the brain is not the only structure concerned, partly because it acts as a whole,

and also because the presumptive changes in it are too evanescent and delicate to be accessible to our crude methods of examination.

To limit oneself to the brain in studying the somatic correlates or basis of mental phenomena would be an error. In the physical accompaniments of emotion, the whole body participates through the mediation of the vegetative nervous system and the endocrine glands. This is significant, because emotional upset is one of the most important phenomena of mental disorder. The sequence of psycho-physical happenings of which an emotional upset is the climax and the outward sign, may be started not only by some mental happening, but also by physical experiences—intoxication with a drug, or a circulatory disturbance, or a metabolic upheaval such as acute hypoglycæmia. Whether, for example, this hypoglycæmia comes from outside, as an injection of insulin, or arises (as it rarely may) from within the body, as a “spontaneous” deficiency, is of little consequence in its bearing on the mental disturbance engendered. The chief emphasis lies on the physical apparatus through which so widespread an affection of the whole organism can be evoked, just as in other circumstances the emphasis would lie on the psychical apparatus which serves the same end. This applies more widely than to emotional disturbances alone. Where a symptom is, on the face of it, definitely physical or definitely mental, its causation may not be inferred to be exclusively of the same order; the chief cause of, say, an anorexia may be a series of mental experiences or an attack of migraine or a uræmia or a pituitary disease. Study of the anorexia alone cannot serve to discriminate them; not even study of the psychological state alone, or of the physical state alone may suffice. Very often the physical and psychological factors in causation are mingled almost inextricably—they represent, in fact, different facets of the same series of phenomena.

Mental experiences.—Mental growth is dependent on daily experience for its material. Experience can be subdivided into perceptual, emotional, and other kinds, but such a division is fictitious. The means by which daily experience is incorporated with our mental equipment and acquires an influence over our subsequent behaviour in all respects can only be understood if we avoid thinking of emotions, instincts, perceptions and other abstractions as real entities, as distinct and separately operative forces. Memory, for example, is not merely an intellectual function by which we recall a happening into consciousness in more or less verbal terms, but a device, or function, by which past happenings are able to influence subsequent behaviour; the ways in which they do so, and the form in which the earlier experience is reproduced into consciousness, will be greatly influenced by its original emotional, as well as more purely perceptual, aspects, and by other physical and mental experiences—a distressing repetition of the experience, for example, or a physical happening like concussion or cortical atrophy.

There are general tendencies in mental life—instinctual tendencies—which bring us into relation with our immediate surroundings, direct us to feed ourselves, maintain our lives, reproduce, and aim at other ends, which are variously formulated by philosophers, saints and psychologists. These biological forces, however denominated or classified, are not peculiar to human beings, but in respect of human beings are so much more accessible to minute inquiry along verbal lines, that an unduly complex conceptual system has been built up to describe them. Comparative, behaviouristic and experimental

psychology have partly corrected unreal refinements of a verbal kind, as also can physiology and a truly medical psychology. Metaphysical subtleties and speculations, *e.g.* psycho-analytical ones, cannot meet medical needs, nor enable one to understand the human being as a whole organism in the way that the doctor must. The influence of previous experience on all subsequent behaviour is as evident in physiological happenings as in the mental field; the special language and formulations and hypotheses of psychology are not to be taken as wholly and permanently separate from those suitable to less highly integrated functions, though something must be conceded to the special complexity of psychological phenomena. Such terms as projection, sublimation, conversion, symbolism, identification, repression, amnesia, perseveration, displacement of affect, cover special instances of the general relationship between inherent tendencies of the organism and their material substrate, and the influence of past on present experience and behaviour.

In dealing with the multifarious world about him, a human being is constantly obliged to select what he will perceive, and in what form he will perceive it; pure "objective" perception never occurs. To perceive things at all, he must give them meaning by relating them to himself and to his previous experience. Unless he can do this, not necessarily consciously, he is at the mercy of his environment, as a new-born baby is. Perception is therefore an active process; it has instinctual and emotional, as well as cognitive aspects. It depends partly upon memory for being able to give meaning to what it perceives; such memory need not be conscious. Consciousness, it is well to bear in mind, is only an attribute of psychological happenings, not their essence or their criterion; mental life goes on with varying degrees of consciousness attaching to it. There is no sharp division between conscious and unconscious mental life: no region called "the Unconscious" with its own rules and contents. Many of the psychological happenings most significant for psychiatry go on without clear consciousness of them, but in appropriate conditions they may be accompanied by much more, or by full, consciousness. Biologically and psychologically regarded, consciousness is an attribute, like memory or movement, immensely important for us human beings, but not a "present-or-absent" factor decisive for our mode of mental conduct.

Perception being thus an active process, which makes use of past experience, it not only selects its material and invests it with meaning, but in doing so may distort it, and give it a special "false" meaning. Unwelcome emotions may be thus projected on to external objects or happenings, which are then regarded as hostile or contemptuous, or in some other way significantly related to oneself. This is not remote from the process in visual perception, whereby one projects the image on one's retina into the external world, and is convinced of its reality there; the further process of clothing it with emotional significance depends on one's inherent tendencies and one's previous experiences. Paranoid symptoms, ideas of reference, grandiose and self-reproachful delusions exemplify this. Hallucinations and kindred phenomena are a special instance of the interplay between material substrate (*e.g.* in cocaine poisoning), inherent tendencies (*e.g.* visual fantasies of children), and past experience (*e.g.* hallucinations of homosexual abuse or divine commands). Similarly, by fantasy and imagination the outer world can be manipulated or denied according to the heart's desire, just as by body-images

of proposed movement the way is prepared for purposive muscular action. In giving meaning to present things, personal connections between them and earlier experiences are established; whether normal or morbid, this ascription of "symbolic" meaning to every-day objects is indispensable to thought, and is most striking in our use of spoken or written language, where sounds and shapes are conventional symbols for the most diverse experiences. Some of our words are personal to ourselves, and are used in an individual way; in morbidly heightened form, this process may issue in schizophrenic neologisms, or oddities of expression. Similarly an obsessional patient may feel towards some word or object a superficially incomprehensible mixture of attraction and repulsion, which is due to this word or object being the symbol of some earlier experiences that have been of great moment in his life. To see how it has come to be such a symbol calls for minute study of his earlier experiences. Physical happenings in one's own body may symbolise present emotions or earlier experience of a momentous and emotionally painful kind. A gesture of disgust may normally be evoked unconsciously by a banal happening, which has somehow become emotionally coloured by past experience. A headache may embody our dissatisfaction with a present situation. So hysterical "conversion" symptoms may reflect and symbolise an inner emotional struggle, as may also some obsessional movement, schizophrenic stereotypy or hypochondriacal fear. The body, with all its functions, is the background of psychic life, and resonates to it.

What experiences will be important in determining the form of mental symptoms, depends much on the emotional disturbance they originally provoke, and this, in turn, on the instinctual drives which they touch on and disturb. Instincts may conflict, and the emotion accompanying the conflict prove so disturbing that it cannot be borne in its naked form; "repression" serves the end of making this more or less tolerable, through disguising or distributing it. So emotion may be shifted from one object to another, and paradoxical or unexpected emotions be thus aroused by objects on to which the affect has been displaced. Or energy mainly directed to plain ends, *e.g.* sexual love, may be diverted into less obvious channels, and when thus "sublimated" and mingled with features derived from other instinctual sources, its origins may be hard to recognise. Sexual instincts so often conflict with others that many of the most powerful motives for the production of mental symptoms come from the struggle.

To describe the whole of instinctual life, however, in terms of sex and aggression, as is sometimes done, is only possible if one strains the meaning of these words out of all knowledge. It is as unwise to make the sexual paramount in explaining psychogenesis as to burke it.

The patient's present symptoms, it is clear, must be examined in the light of his earlier experience. Thus one elucidates in detail the content of his illness and some of the causes of its occurrence. In doing so it is not necessary to push back one's inquiries to a supposedly crucial stage of early childhood. The experiences of the first two or three years of life are, like all subsequent experience, contributory to mental development, and they show certain sequences of phenomena characteristic of such development. Moreover, their relative simplicity makes it possible to recognise in these early reactions the instinctual drives, or (more correctly) the "inherited functions" which become manifest when the environment supplies the

necessary material, though, of course, it cannot supply the necessary energy and direction ; these last must come from within. On the other hand, the functions recognisable in the relatively simple reactions of early childhood are not the same as those which may be seen in later years when the organism is more fully grown, any more than an infant's physical structure and functions are identical with those of the more differentiated adult. The obvious continuity of the actual happenings in a human being's lifetime does not justify one in trying to analyse and reduce all adult mental phenomena into terms of supposed child psychology, nor does clinical practice usually require it.

The effect of war upon the incidence of mental illness has obvious importance. The psychiatric disorders which occur in war do not differ in kind from those of more normal times, but certain forms of disorder, especially panic, psychogenic stupor and gross hysteria in men, become commoner and sometimes more severe. People are exposed to unaccustomed dangers ; their privations are both material and emotional ; they have to surrender some of their independence and individuality ; and they are thrown together in groups and therefore prone to share in group-feelings and group-behaviour. It is doubtful whether the losses, fears and psychological infections of war directly lead to an increase in certifiable mental illness, though such exogenous factors as syphilis, alcoholism and malnutrition may do so, under the conditions of thoroughgoing modern war. It is, however, neurotic disorders that are most prominent then.

Course and Prognosis.—The making of a correct diagnosis may in psychiatry indicate the general drift of an illness—towards recovery, chronicity, progression, or relapse—but is of even less use than in the rest of medicine for showing how far this will apply to a particular patient. For this, careful study of the individual history and illness are indispensable. The prognosis can be inferred from the ætiological factors, the mode of development, and the form of the disorder.

Where a known external cause has been at work, its point of attack, its severity and persistence will affect the issue. This applies equally to such "organic causes" as poisons and cerebral diseases and to "mental causes," like economic misery or frustrated love. The physician must consider how long the environmental cause has been acting, what changes it is known to produce—whether in the way of cell-degeneration or habitual gloom, fibrosis or fantasy—and whether it is likely to persist. He must also ask if the patient's previous history has shown that he is specially sensitive to such a trauma. This brings in the intrinsic ætiological factors. How has the patient previously reacted to this sort of interference or to any disturbing circumstances ? Has he fallen more and more into unsatisfactory habits in meeting his daily life and its difficulties ? How has his whole character developed ? Is there good evidence of his being able to cope with partial deviations from mental health ? Has he inherited tendencies to benign or to progressive illness ? Which seem to be the most useful reparative or stabilising features in his personality ? How far are his struggles with the world an outcome of his intrinsic endowment, evident in various guises since his childhood, how far have they been forced upon him by an adverse milieu ? How old is he ? There is more chance, if he is young, of his being adaptable, so that the removal of various stresses

may help him, and his instinctual energies be diverted into less morbid channels; as he grows older, he may gain in stability, but gradually become more disposed to fear and suspicion, bodily preoccupations, and fixed attitudes of mind.

An abrupt onset is favourable, other things being equal. A gradual, especially an insidious, onset may indicate a rooted abnormality that will be hard to shift. The longer an illness has gone on, the more will it have become autonomous, *i.e.* independent of its immediate causes of occurrence, and prone to become a gross or text-book example of some chronic anomaly. A study of the ups and downs in the course of an illness may show favourable influences that can with profit be deliberately brought to bear on it, as well as harmful ones that must be avoided. The more reconciled the patient has become to his illness the less satisfactory the outcome.

As to the form of the illness and its prognostic value, there is much empirical knowledge at our disposal. Thus, a predominantly affective attack will very likely clear up, but may recur; a schizophrenic syndrome is in the long run usually ominous; hypochondria and depersonalisation, especially in young people, tend to last a long time, even years; sexual perversities can seldom be got rid of; hysterical symptoms can easily be changed, but hysterical reactions are persistent; obsessional attacks are either periodic or very chronic; melancholia is often a fatal disease, through suicide or refusal to eat; delirium tremens commonly ends by crisis or lysis after about seven days; untreated general paralysis of the insane goes downhill towards dementia and death, with partial remissions on the way: and so forth. There is a wealth of such special prognostic knowledge, based on clinical observation and statistics.

Obviously prognosis must always take account of treatment. Will treatment be efficacious? Will it be practicable? It is absurd to forecast how general paralysis will turn out if one does not know whether one will be giving artificial fever and tryparsamide, or how hysteria will turn out before one has decided whether psychological and social treatment will be possible. In every mental illness this is one of the essential points to be weighed in prognosis: what will be the conditions, beneficial, neutral and adverse, under which the patient is going to live henceforth; and, in particular, what will be those specially devised conditions of every kind, social, psychological and physical, which can be regarded as likely to have therapeutic effect?

Treatment.—**PROPHYLACTIC.**—Much can be accomplished by social measures; also by individual care, though that is less certain. A striking instance of social influence in preventing mental disorder may be seen in alcoholic psychoses, which have been cut down in this country to a third of what they were before the War of 1914–1918. Morphine and cocaine addiction and lead encephalopathy are now rare, typhoid delirium is exceptional, and typhus unknown. It is the organic mental disorders that have been more accessible to these preventive methods so far, because they have one indispensable cause that can be controlled. The “functional” disorders are partly dependent, it is true, on social factors, such as economic security, lack of employment, imposed moral, cultural and educational standards, competition and ill-judged interference. But the remedy for these is to seek, and their total removal is utopian. There is, nevertheless, much

room for prevention here. A preventable social cause may be well seen in "compensation neurosis" where the administration of a humane statute involving lawyers, insurance companies, doctors, employers and employees often has the inhumane effect of evoking hysterical symptoms, anxiety and a depressive or paranoid invalidism in the injured man.

Individual preventive measures cover both the intrinsic and the extrinsic causes. Eugenic precautions, such as birth-control or voluntary sterilisation (if legalised), may under skilled guidance prevent some mentally unstable persons from being born to parents who, having had mental illness themselves, do not wish to propagate it. If physical factors, *e.g.* diabetes, be prominent in causation, it may be possible to prevent this mental illness, or at any rate to scotch it in its early beginnings, by dealing with the somatic disorder. Thus, there are far fewer cases of syphilitic psychoses now that syphilis is less often contracted and earlier treated. The psychological reactions to a physical disease or blemish may be favourably modified or averted, when foreseen. It is for obvious reasons impossible to counteract mental disorder by regularly protecting the patient from physical or psychic trauma: a life that is guarded against risks and painful experiences is almost certain to issue in mental ill-health, out of its very emptiness. By altering a patient's environment and way of living one may, however, be able to avert an impending illness: only study of the individual patient can show how this end may be achieved.

The difficulties in the way of making the patient's environment easier for him are immensely greater during war, and it may be inadvisable to attempt any such change. Social needs have to come before individual ones in so many instances during war that the measures which would appear most favourable to the patient's mental health are often quite impracticable. Much, however, can be done by careful selection to lessen maladaptation among soldiers and other large groups. The mental hygiene of war is a complicated problem which cannot be divorced from the political and economic issues, as well as the military ones.

How far the treatment of behaviour disorders and neurotic traits in childhood can be trusted to avert outbreaks of definite mental illness in later life is a disputable matter, but it is certain that by taking advantage of his plasticity and responsiveness, a bent can often be given to the energies of the mal-adjusted child, which will result in his being socially better adapted and better able to deal with his problems. The more persistent the beneficial influences one can bring to bear on development at this impressionable age, the more valuable the prophylactic effort.

Measures of mental hygiene that may be recommended to the community as a whole are still of a negative kind: what to avoid rather than what to do. This applies most obviously in the field of sexual practice and belief where needless fears and harmful education are rife, as with regard to the masturbation of adolescence—a normal and comparatively harmless phase of sexual development.

TREATMENT OF THE ACTUAL ILLNESS.—This is almost as varied as ætiology and symptomatology. To use only one method of treatment, however simple or complicated its theory, is to fight illness with one hand behind one's back. There is no valid distinction between palliative and curative therapy: the distinction should be between more efficacious and

less efficacious. The nearest approach to a successful causal therapy is attained with those mental disorders which are closely related in time and form of occurrence to some indispensable cause, *e.g.* a toxic delirium, a reactive depression or anxiety, an interstitial syphilis of the brain. But these are rare conditions if one considers the whole of mental illness. The treatment of general paralysis by fever is not causal, its theory is dubious, its basis quite empirical; yet its success is such that it is the most important therapeutic advance in psychiatry for a hundred years. One cannot despise any measure that promotes the recovery or well-being of the patient: the giving of drugs, the prevention of suicide, occupational therapy, analysis of motives, removal into favourable surroundings, hypnosis, re-education and other means of helping the patient are not to be graded in a hierarchy with an arbitrary scale of values, in which recovery is called spontaneous unless psychotherapy or a novel chemical treatment has been employed.

Sometimes a patient's condition demands energetic intervention; sometimes it demands restrained symptomatic treatment; sometimes social adjustment is called for; sometimes endocrine injections. Whether the accent in treatment shall fall on the physical or the psychological or the social side will often be less important than care that all the available resources are used. It should not be regarded as a matter of course that a diagnosis should connote a method of treatment: *e.g.* that psychoanalysis is the only thorough treatment for obsessions, while for mania continuous narcosis is the "proper" method. Nor, to mention another common error, should it be lightly assumed that a heavily tainted family history or other evidence of a strong constitutional factor indicates that treatment is out of the question, a superfluous struggle against fate.

Treatment may be considered as social, psychological and physical. For some types of illness obviously much more stress will fall on one than on another of these, *e.g.* in hysteria, general paralysis of the insane, epilepsy.

Social and occupational treatment.—The first task in social treatment is to decide where the patient is to be looked after. Is he fit to be at home, should he be in a mental hospital, or in some environment intermediate between these extremes? The decision as to the need of a mental hospital rests in the first instance on the danger the patient presents to others, or the chance of his committing suicide. These two problems of behaviour were at one time almost the only grounds of admission to a mental hospital, but such questions of "certifiability" need no longer preoccupy the psychiatrist, since voluntary treatment has broadened the scope of the mental hospital and modern conditions made it suitable for many patients who would ordinarily be regarded as "neurotic," rather than "mental" "psychotic" or "insane" (*e.g.* early cases of general paralysis masquerading as neurasthenia, or obsessionals who fear their own impulses and want to be protected against themselves). Psychiatric hospitals and clinics dealing only with voluntary cases also bridge the gap between out-patient care and certification.

The social decisions in treatment cover much more than merely the mental hospital issue. If the patient's immediate environment contains many disturbing influences, it will be desirable for him to be away from them temporarily at least, so long as this does not entail worse troubles; summary decisions are here impossible. It may be useless, for example, to get a woman who is

paranoid about her neighbours to move to another district to escape them, unless it is the actual conduct of the neighbours and not the patient's morbid attitude that is provoking her suspicion of them. It requires a close knowledge of the facts as well as wisdom and psychiatric experience to give advice on matters that may wholly alter the course of a patient's life—advice, say, about separating from his wife, giving up his job, or emigrating to the Dominions. Many instances of this might be offered. Neurotic patients are often advised to get married, especially if loneliness and sexual needs trouble them, as though marriage were a panacea; such advice by rule of thumb too often makes their condition worse, ruins the life of the person they marry, and results in offspring that have to be treated at a child guidance clinic. Weary, depressed patients are often harmfully urged to go to dances and lively seaside resorts where they must try to look happy. Hysterical patients do not benefit by being put among people who are hostile and contemptuous, any more than in an atmosphere of mawkish sympathy and compliance.

In the social treatment of patients indispensable help can be given by trained psychiatric social workers. Their assistance is not restricted to the patient's economic problems, though it is most obvious in that field.

Occupational treatment is important for all kinds of mental disorder. Where there is acute overt emotional disturbance, rest is at first desirable, as also for confusional and delirious states. In these conditions opportunity for occupation must be gradually offered to the patient as his disorder subsides; steady, simple work is preferable to the restless unsatisfying fickle activity in which he would often engage, if left to himself. The less acute any mental disturbance, the more necessary is it that occupation should be urged upon the patient, and that it should be disciplined and congenial. This applies equally to gross psychoses and minor affections of the neurotic sort. Allowance must be made for the patient's bent, his symptoms and personality, and especially his more or less conscious reasons for working and not working; hence there will be much diversity in the conditions of his occupation, whether it be therapeutically contrived in a hospital, offered at a Rehabilitation Centre, or sought out as remunerative work in the open market. Mental health cannot be permanently retained unless one does some satisfying work; often it cannot be recovered unless one does. Work is not satisfying, in the long run, if it is done mainly as a diversion, to fill in time.

Psychological treatment.—There is no form of treatment which has not a psychological aspect and result. The term psychological treatment or its synonym "psychotherapy" is, however, conventionally limited to those forms which depend upon direct and personal relationship between the patient and the physician. They have separate names, and are divided into schools and techniques. Stress may be laid upon the prestige of the physician (as in hypnosis), the patient's attachment to him, in all its complicated phases ("transference"), the trained understanding and thoroughness with which he clears up the patient's problems (persuasion, re-education, distributive analysis), or on his qualities of personality—enthusiasm, energy, warmth, candour, wisdom. In so far as psychological treatment is necessarily based on a personal relationship it cannot be made a routine except in its non-essentials: whatever rules the psychiatrist follow or whatever the training he has undergone, he himself will be more important than his method in

benefiting the patient. To that great extent psychotherapy is not a scientific procedure. That is not to say that method and training are of no consequence—far from it—but only that they are devices whereby the influence of one human being upon another's mind and conduct can be turned to the best medical ends, and the dangers inherent in such a relationship minimised.

The more specialised, intricate or esoteric the method, the less suitable is it to be used by any but the most expert. It is not proposed here to detail the many kinds of technique that have been employed. The general rules that must be followed in any psychotherapy are :

1. To regard the removal of symptoms as a good thing, but the maintenance of normal social adaptation as far better. It is bad to get rid of one symptom only to see it replaced by another, but much worse to get rid of all symptoms only to see the patient at the end of treatment a dependent and introspective hypochondriac of the mind, a social invalid.

2. To seek for the psychological cause of the patient's illness only to the extent that the patient's wellbeing demands, which is often far short of what one's own interest and psychological curiosity would demand.

3. To consider carefully whether any shock to the patient, any aggravation one produces in his illness even temporarily, may be a sign of bad treatment. It is on the whole unnecessary to make the patient worse in order to get him better, though many psychotherapists believe the contrary.

4. To be satisfied with the patient's recovery, and not to aim at his promotion to a state of ideal mental health and self-understanding. It is better that treatment should be quick and effective than drawn out to meet theoretical standards.

5. To understand the development of the patient's illness, and to interpret it both to him and to oneself, in terms of real experience and not of hypothetical forces.

6. To treat the patient without allowing one's own emotions to be more concerned in the course and outcome of the treatment than is usual in the treatment of a physical illness.

7. To aim at harmonising the patient's mental life by giving his ill-managed energies fitter material to work at, and release from the burdens laid on them by past experience.

It is impossible to describe in general terms what the psychotherapist does, otherwise than by metaphor or analogy : he promotes the ventilation and desensitisation of emotional disturbances ; he elucidates latent or obvious muddles, disentangles conflicting tendencies, giving them new incentives and a different direction ; and so guides the patient through the maze of his life's experience, as recalled in memory, that he is afterwards better fitted for dealing with current experience, knows himself better and has somewhat purged himself of past harms. All "analytic" methods review the patient's life as he recalls it under special conditions, *e.g.* of free association, hypnosis, biographical scheme, etc. They stop at different points, some aiming at emotional clearance by abreaction, some at a redirection and liberation of the instinctual bases of character, while others remain content with an educational achievement.

Whether psychotherapy, in the above sense, is to be applied to a case will depend on the following factors : the patient should be willing to co-operate in the treatment ; free from such hindering disabilities as, say,

deafness ; able to give the necessary time ; of at any rate average intelligence ; still capable of modification (as he would not be in old age, or with very long-standing and indurated habits of faulty reaction, or with organic cerebral disease) ; and, finally, endowed with a considerable residue of normal mental functions with which one may work. The more profound his aberrations, as in schizophrenia, or the more extreme his emotional disturbance, as in agitated melancholia, the less is he fit for psychological treatment of this individual and specialised kind. Psychological treatment, however, in the literal and larger sense of the words, is essential for every variety or stage of mental illness, and every degree of co-operativeness or intelligence. It is a wide notion, including all that may ease or reassure the patient, bring him to a better relationship to those around him and with himself, and protect him from being distressed by the ignorance, lack of tact, or thoughtlessness of others. It is as much negative as positive. One must avoid arguing with the patient, telling him lies "for his own good" or to avoid unpleasant scenes, cajoling him, making promises that will not be kept, threatening or punishing him, jesting at his expense, losing one's patience with him, assuming he is indifferent to what goes on because he looks indifferent, provoking him by petty supervision or frequent rebukes ; one should not assume that he is quite irresponsible or quite responsible, nor talk theory to him, nor get on a false footing through ready assent to his delusions and his point of view. The physician and the rest of those who are in contact with the patient must do certain positive things : make due allowance for his disorder influencing his conduct, use their understanding of the psychological happenings without saying so, take advantage of every opportunity created by other methods of treatment. When occupation, narcosis, hydrotherapy, massage, a physical illness or other happenings bring him more closely into contact with nurses and physicians there are chances of unobtrusive psychological treatment in the wide sense.

Physical treatment.—"Mechanical restraint" and violence are now foreign to the treatment of insanity ; the patient may be unrestrained and violent, but his treatment may not. It is still necessary, however, to restrain a patient who is bent on harming himself or others, and physical force may be the only way of doing so, or of giving a patient by tube enough food to keep him alive when he abjures the natural way of eating. But force must always be a last resort ; and chemical substitutes for it seem only a little less of an evil. Drugs have their place in the treatment of all kinds of mental disorder, but their use easily turns to abuse. Whether one is giving morphine and hyoscine in an emergency to an acutely excited catatonic, or prescribing aspirin for a mild hysteric, the chief danger must be borne in mind, which is not overdosage, habituation, or suicidal misuse, but the habit of stupefying or satisfying a patient with drugs when other means might be taken, better suited to his condition. Sedative drugs should not be a short cut ; neither should they be eschewed. They should be given when other measures will not serve, as for some obstinate form of insomnia, anxiety, agitation and restlessness, or when their use obviates greater troubles, *e.g.* the pulling of bandages from an operation wound. The symptoms of intoxication must be watched for with more than usual vigilance when bromide is being given, because if unrecognised as such they may lead to certification—for an avoidable drug-made psychosis. Continuous narcosis for nearly a fortnight,

with the patient sleeping through 18 or more of the 24 hours, is sometimes efficacious in abbreviating an acute attack of mental illness; it is a dangerous method except in skilled hands.

There are other drugs to which the above cautions scarcely apply, *e.g.* endocrine preparations, remedies specific and otherwise for the physical basis of "organic psychoses" (*e.g.* arsenical treatment for syphilis of the central nervous system), and aperients. Insulin for promoting hunger, calcium for those with hysterical hyperventilation fits, amphetamine (benzedrine) for anxiety, and a number of substances—from nitrous oxide to amytal—that relieve catatonic stupor, or facilitate psychological inquiry and treatment, have all been found useful on occasion, though their field of application is small.

Two methods of treatment have been widely employed of late years, especially in the treatment of schizophrenia. Insulin has been employed in large doses to produce hypoglycæmic coma repeatedly; and certain convulsant drugs (pentamethylenetetrazol and cyclohexylethyltriazol) given to produce fits. The benefits to be obtained by these methods are not yet clear. Undoubtedly in some cases the duration of a schizophrenic illness can be shortened by the insulin treatment, and recovery brought about in cases regarded as having a bad prognosis. It is, however, in those that would have a favourable outlook by other methods of treatment that the majority of insulin successes are obtained. The technique demands skill and experience, if considerable risks are to be avoided. It consists essentially in daily administration of the appropriate dose of insulin to produce coma. The duration of the treatment is usually not more than two months. Treatment by the artificial production of convulsions was first instituted for schizophrenia, but the results have not proved as satisfactory as was at first supposed, and it is of very limited application. The method has proved effective in terminating obstinate melancholia, chiefly involuntal, and in abbreviating attacks of depression which would otherwise have taken months to clear up. The chief objections to the method are the disagreeable experiences many patients have immediately after the injection, and the likelihood of cerebral impairment, and of fracture of the long bones or dorsal spine during the fit. The last complication is not infrequent, and may occur also when the convulsions are induced, not by a drug, but by electrical stimulation of the brain, a method employed within the last two years.

Exercise or massage and hydrotherapy are beneficial as much for their psychological as for their physiological results; the latter, however, are not negligible, as may be seen in the effect on an excited or an anxious patient of a continuous bath at body temperature. The chief importance of diet lies in the frequent refusal of food by patients depressive, hysterical, stuporose, paranoid, hypochondriacal, or over-active. Feeding by the nasal or oesophageal tube is a necessity in many such instances, after every other method has failed. Rarely, special diet is called for, as in epilepsy, the symptomatic psychoses of diabetes, pernicious anæmia or pellagra, and also for some temporary disabilities of the alimentary tract—*anorexia nervosa*, psychogenic hyperchlorhydria. As a rule, however, such dietetic regime, and indeed all physical treatment of localised psychogenic disturbances of function in a bodily system, is an expedient rather than a settled and adequate mode of treatment. Many patients with a visceral neurosis, a hypochondriacal

preoccupation, a hysterical anomaly, or a somatic delusion are greatly harmed by the prolonged physical investigation and treatment they receive : it confirms the symptom, localises it all the more, and brings fresh ones in its train. Sometimes one has no choice ; a progressive hysterical contracture, a dermatitis artefacta, a sore infected by constant picking, a tooth loosened by obssional knocking at it demand treatment.

The caveat against lightly resorting to physical treatment of psychogenic anomalies is especially applicable to operative surgery. In general there is no more reason for "cleaning up the septic foci" in a person with mental disorder than if he were a mentally healthy person—other things being equal. Removal of teeth, appendix or tonsils, scraping of sinuses, and searchings of the pelvis are operations that in psychological medicine seldom yield the results expected of them.

CLASSIFICATION

The ideal classification would be on a uniform basis, according to the nature of disordered physical and psychological function, or according to innate and external causes. Since we do not know enough to do this, a mixed ætiological, functional and clinical grouping is used, whereby the same illness can belong in several categories. It is obviously provisional. The chief division is between those mental changes accompanying distinctive somatic disorder and those for which no such physical relationship has been demonstrated. The former are called symptomatic, or organic ; the latter constitutional or functional. It is needless to illustrate the point that everything found in the latter may be seen also in the former. The reverse of this is not true, because there are some symptoms—due to the loss or damage of essential tissues, especially in the central nervous system—which can only occur when the material substrate is grossly damaged.

Although the "functional" group is made up of those conditions for which no distinctive somatic disorder can be found responsible, it by no means follows that their causes or basis are therefore purely psychological. Theoretically, such a belief is untenable ; and as a matter of observation certain physical disturbances so regularly accompany these disorders and a physical configuration may be so linked with them that there is small doubt that eventually the somatic disturbance of function in them will be well enough worked out for the terms "organic" and "functional" to lapse, and only the crudity of the physiological changes remain as a point of difference.

As mental disorder thus comes closer to general medicine so must the whole of general medicine reveal its psychiatric side, which is now as little illumined as the physiological side of the psychoses.

The first or toxic-organic group is large, the chief syndromes in it being neurasthenia, confusion, and delirium and dementia. Such phenomena as apraxia, aphasia, agnosia, amnesia and hallucinosis are fairly frequent in this group.

The second group, comprising three-fourths of the recognisable mental illnesses, includes the insanities or psychoses, and those anomalies, outwardly less alien to the normal mind, commonly called "neuroses." The distinction

between neuroses and psychoses is at times convenient, but without substance. To argue whether a dubious case is neurotic or psychotic is like arguing whether a man of medium size is thin or fat : he is both and neither. A genuine decision as to ætiology, prognosis or treatment turns not on whether a case is regarded as neurotic or psychotic, but on more solid findings. Since such words die hard, the best use of them is to term a patient with mental disorder "neurotic" if he has insight into his illness, is co-operative and unlikely to need care in an institution, and to term him "psychotic" if the contrary is the case.

The toxic-organic group is divided into diseases located in the nervous system and those affecting it indirectly, as uræmia or lead poisoning may. Some are toxic, *e.g.* delirium tremens ; some degenerative (senile psychoses) ; some inflammatory, *e.g.* encephalitis lethargica ; some plainly hereditary, *e.g.* Huntington's chorea or "primary" mental defect ; and some privative, *e.g.* pellagra or myxœdema.

The "functional" conditions are arranged according to whether emotional disturbance is evident and predominant (affective disorder), or whether there is profound derangement of thought, feeling and contact with the real world (schizophrenia), morbid false beliefs have become fixed without intellectual or emotional deterioration (paranoia), repetitive and seemingly irrelevant phenomena hamper mental activity (obsessional), signs of physical or mental ill-health, especially dissociation, readily appear when an unpleasant situation may thereby be escaped from (hysteria).

As will be seen in the special sections, the personality of the patient may also be a criterion of these groupings, with the proviso mentioned earlier that illness does not only occur in those with the appropriate psychopathic anomaly of personality, nor does the latter by any means regularly issue in definite symptoms. Unless, however, psychiatry takes account of the psychopathic personality, even when not accompanied by symptoms of illness, it cannot study delinquency, disorders of behaviour in children, sexual perversions and other not obviously medical anomalies which touch very closely on psychiatric problems in the stricter sense, but are omitted here for reasons of space.

The following is the classification used here :

1. Organic Disorders :

(a) Degenerative and Hereditary Brain Disease.

(Senile dementia, cerebral arterial disease and hypertension, Huntington's chorea.)

(b) Syphilis of Central Nervous System.

(c) Other Cerebral Diseases.

(Lethargic encephalitis, Sydenham's chorea, disseminated sclerosis, cerebral tumour, cerebral trauma, epilepsy, etc.)

(d) Intoxications.

(Alcohol, morphine, cocaine, bromide, etc.)

(e) Infections and Exhaustive Disorders.

(Infectious toxæmias, hæmorrhage, etc.)

- (f) Metabolic, Endocrine and Visceral Disorders.
(Diabetes, pernicious anæmia, pellagra, exophthalmic goitre, myxœdema, tetany, pituitary diseases, sexual epochs, cardiac disease, uræmia, etc.)
- (g) Mental Deficiency.
- 2. Affective Disorder.
 - (a) Excitement.
 - (b) Depression.
 - (c) Anxiety.
- 3. Schizophrenia.
- 4. Paranoia.
- 5. Hysteria.
- 6. Obsessional Disorder.

In the above classification mental deficiency is given a separate heading, though properly it should be systematically distributed among the preceding groups, since it differs from the rest of mental disorder only in the age at which the damage is done. Custom and convenience compel the old distinction to continue.

The above are great clinical groupings, types of morbid reaction, which are the nearest to a valid and useful classification we can get at present. There are subordinate symptom-complexes or syndromes, which are likewise innate and preformed, and likewise evoked by circumstances, but which are not limited to any one of the major groupings—they are the web that runs across the psychiatric pattern. The most important of these are depersonalisation, hypochondria, twilight states, stupor and other disorders of motility, and spasmodic attacks and seizures of different kinds. Between symptoms (classified on a psychopathological basis) and the main groupings which best serve clinical purposes, these symptom-complexes have an intermediate place, comparable say, to that of mononuclear leucocytosis or coma in general medicine.

All the categories of psychiatry stand for mixtures of symptoms due to disturbance of control, capacity or co-ordination and synthesis in mental life. Nor is this true only of mental life, since the same symptoms may arise through physical disturbances of function which may be classified in the same way; this may be plainly seen in such a disease as encephalitis lethargica.

ORGANIC DISORDERS

A. GENERAL DESCRIPTION OF TYPES

The varieties of form and course in organic psychosis are essentially few and simple, in contrast to the causes, which are numerous. In other words, there is no support for the hope that to each physical disease there corresponds a characteristic mental disorder. It is not possible in an organic psychosis by study of the mental picture alone to infer its physical cause; for that the methods of somatic medicine are needed. Many different poisons and lesions

may produce the same effect on the mental state. Differences depend on the degree and duration of the physical damage and its site, which may determine neurological and other symptoms of a typical kind; *e.g.* in G.P.I. or encephalitis lethargica.

They are the least constitutional of all mental affections, yet even in them constitutional factors are far from negligible. To such factors it is due that one man will show a psychosis with physical illness that in another would lead to no such mental upset, and that one patient responds with a manic extravagance to the cerebral disease that makes another patient depressed. Moreover, hereditary factors can be of great importance in these organic affections, as may be seen in amaurotic idiocy or Huntington's chorea.

The few syndromes commonly met with here, though they are not restricted to organic disease, must be described before seeing how particular diseases colour them and determine their course and treatment. In the organic syndromes, a diminution in mental capacity is the central finding. Nearly all of them may occur in cases and types of illness in which no structural damage can be found, as might be expected seeing that the available patterns of structure and function are in all cases much the same. Delirium or confusion may occur typically in non-organic syndromes, though much less frequently than in organic ones.

(1.) NEURASTHENIA.—This term has been over-used and ill-used, like most of the more palatable diagnoses (*cf.* anxiety neurosis), but it need not therefore be discarded now. It denotes a form of irritable, hypersensitive weakness and depression that is not uncommon after infections, exhausting experiences (*e.g.* hunger, lactation, insomnia, worry, hemorrhage), cranial injuries and chronic poisoning (*e.g.* with alcohol or coffee). It is true that a clinical picture indistinguishable from it frequently arises where physical causes are unlikely and emotional causes are obvious: this clinical finding has the same significance as the fact that the anxiety of exophthalmic goitre is like psychogenic anxiety. Just as the anxiety of exophthalmic goitre or constant fear can pass into delirium, so can physiogenic neurasthenia be aggravated until it becomes plain dementia.

The symptoms are partly somatic—active deep reflexes, increased sensory irritability, feelings of pressure on the head and pains in the muscles and elsewhere, giddiness, vasomotor lability, delayed peristalsis and feelings of fullness in the abdomen, diminished libido, slight clumsiness, and tremor of the muscles of the face, tongue and hands. On the more psychological side, there are feelings of languor, and incapacity to concentrate on any mental work, doubts as to the accuracy of memory, loss of interest, slight depersonalisation, irritability and tenseness, lessened control of emotion, and perhaps slight paranoid, obsessional or hypochondriac trends. This general condition is, when physiogenic, less influenced by a change in mood than would be the case with psychogenic neurasthenia, and the patient is better able to control his motor unrest than his features, which are expressive of his agitation. The chief reliance, however, must be put on the history and physical findings for telling whether the neurasthenia is physiogenic or not; psychological causes which seem adequate to explain the illness may be deceptive.

The course of neurasthenia is towards recovery unless the noxa continues to act; where the noxa persists, extreme chronicity can result. Sometimes

an original physical noxa ceases to act, but meanwhile other emotional ones have entered the field, *e.g.* unemployment, domestic fears and frustrations, and so the illness drags on. Treatment depends on assessment of the causes and the possibility of removing them.

(2) DELIRIUM.—Delirium, most familiar in fevers, can also be produced by drugs and other causes of acute cerebral disturbance: severe affective disturbance also may be accompanied by delirium. Its characteristics are general malaise, restlessness, irritability and sensitiveness to external stimuli, headache, anxiety and troubled sleep, or insomnia. Mild forms of this are met with in so transient an affection as cold in the head. Severe forms are marked by illusions and hallucinations of all the special senses, especially vision. Anxiety often becomes extreme, and the patient is terrified of his fantastic visions. Thought becomes as chaotic and fleeting as in dreams, activity is incessant and past experiences of daily life are revived, as in the occupational delirium of alcoholics. Attention is weakened, and orientation in time and space much impaired. There are striking variations in the severity of the condition in the same patient: it becomes worse in the evening or when the patient has hardly any external stimuli to keep him in touch (cf. delirium at night and after a cataract operation). The extent to which consciousness is clouded usually corresponds to the amount of perceptual and affective disturbance. Auditory hallucinations occur with clearer consciousness, visual ones very profusely with a clouded mind. The auditory hallucinations are commonly of an elementary, undifferentiated kind—not voices. Vestibular hallucinations may occur, *e.g.* of floating in the air. Distressing and incoherent ideas pursue each other—ideas of being torn to pieces, burnt, poisoned, buried alive, and so on; also ideas of grandeur.

Closely akin to delirium, and indeed shading into it, is the *confusional state*, in which thought is very incoherent, but the patient is more eager to get in touch with his environment than in typical delirium. If consciousness is not grossly clouded, the patient is perplexed and troubled by the disordered perceptions through which alone he can learn what is going on about him. The picture may be indistinguishable from that seen in some forms of manic excitement and in some catatonic states. Differentiation rests, not on the immediate psychiatric symptoms, but on the history and discoverable causes of the illness. The same is true of *acute hallucinosis* in which orientation and grasp are very little impaired, but auditory hallucinations—especially threatening sounds and voices—abound, and there is a tendency to the formation of delusions on the basis of these and other perceptual disturbances. The name "*twilight state*" is applied to another syndrome in which consciousness is changed chiefly because of some powerful affective influence; anger or fear may so overwhelm psychic life that the patient cannot grasp his surroundings, his thinking is interrupted and slow (except where it falls in tune with the affective disturbance), and his motor behaviour is in keeping with his mood. It is as often of psychogenic as of organic origin—one can hardly, for example, by direct observation tell an epileptic twilight state from a hysterical one. Like delirium and the other conditions just mentioned, it is prone to subside and to be followed by amnesia for what happened during it: where there is some recollection, it may be associated with a conviction that the hallucinations and other morbid phenomena were real external happenings.

(3) DEMENTIA.—Of all gross encephalopathic syndromes this is the gravest and most typical. It corresponds to a diffuse cerebral disease, and is made up of intellectual impairment and lessened control of emotion. Its form depends so much on the stage of the patient's development at which it occurs, that it is customary to consider as dementia only those cases in which the cerebral damage has occurred in later childhood, adolescence, or adult life, and to regard earlier cases, *e.g.* cretins as showing mental deficiency or arrest of development. The distinction is rather artificial, at whatever age it be made. For convenience, only the adult form will be described here. The order in which functions are impaired corresponds to Hughlings Jackson's principle of dissolution: thus, recently acquired memories are soonest lost. There is intellectual weakness—the patient cannot reason, grasp and remember as he could, his attention is less concentrated and sharp, his ideas are fewer, he cannot take in anything complicated or be sure about time and place, he loses himself. His emotions are likewise affected—he weeps over trifles in spite of efforts to control himself, his feelings are shallow and transient, he may be foolishly euphoric, or may burst into anger whenever he cannot get his own way. There are wide variations in the severity of the condition, and its symptoms may be much influenced by the local incidence of the pathological changes in the brain. The extent to which various cerebral functions are impaired may differ widely in the same patient: a man who seems hopelessly demented may be able to play a good game of chess, while another in whom it is hard to demonstrate any intellectual impairment may micturate into his shoes or do something equally stupid and inappropriate; unexpected sexual misdemeanours are not uncommon in demented persons who do not as yet show gross intellectual damage.

Closely connected with dementia are the *amnesic* syndromes, known by the name of Korsakoff. Here the memory disturbance is in the forefront. The incapacity to receive, store and reproduce experience is remedied, as it were, by lying, *i.e.* the patient confabulates to fill up the gaps in his memory. These patients are often ready to adopt suggestions, so that one can lead them to tell absurd tales about their recent movements, *e.g.* that they were yesterday at Vladivostok to see some polar bears. They do not show an intellectual damage or incapacity to deal with ideas that is at all comparable in degree to their memory disorder, but they are always out in their appreciation of time-relationships, especially where the present is concerned. At first blush they often seem to be behaving like mentally healthy people, but one presently discovers that their memory is much impaired, their orientation as to space, time and personal identity correspondingly poor, and their interest and general mood duller than is normal. The disorder of memory is never, as in dementia, a general weakness reaching back even to childhood.

The Korsakoff syndrome is most often seen in alcoholics, in whom it was first described associated with polyneuritis, but it also occurs in a great variety of organic disorders, *e.g.* intoxication with lead, carbon monoxide, and other poisons, uræmia, cranial trauma, cerebral syphilis, and arteriosclerosis—apoplexy may precede it and the amnesic syndrome be thus complicated by aphasia. That it should sometimes follow on delirium is not surprising, since in delirium the same memory disturbance is present,

but covered up by the concomitant excitement, disturbance of consciousness, and hallucinations. Whether a Korsakoff syndrome will clear up depends on the cerebral damage which produces it; the alcoholic form occasionally does so eventually in uncomplicated and treated cases.

Mental deficiency is a special instance of cerebral impairment, as is dementia. It is considered, for the sake of convenience and tradition, in a separate section. (See p. 1846.)

B. DEGENERATIVE AND HEREDITARY BRAIN DISEASE

There is a group of disorders occurring in late middle life and old age, which are clinically and even pathologically near to one another. At the one end of the scale is senile dementia, at the other climacteric anxiety and depression. It includes Pick's presenile dementia, Alzheimer's disease, cerebral arterial disease, and arterial hypertension.

1. SENILE AND PRESENILE DEMENTIA

Ætiology.—Constitutional factors are obviously the most important. A tendency to become dotards may be evident in successive generations of a family; heredity is held responsible for the wide differences in mental health among elderly people. The symptoms of senile psychosis may not be revealed until the patient is exposed to some sudden stress—the death of his wife, the need to move house, the loss of his occupation, some new set of circumstances. Senile psychoses are more common in people with lifelong nervous symptoms.

Pathology.—**PHYSICAL.**—The tissues show the general signs of age, *i.e.* a diffuse atrophy, which makes the convolutions narrower and the weight of the brain less. The nerve cells and fibres are fewer, while the mesodermal and neuroglial tissues ^{are} increased; fatty pigment accumulates. There are also, however, in ^{senile} dementia striking histological features in the grey matter, especially in the cortex, namely, thickening of the neurofibrils, which are characteristically twisted and aggregated, and there are remarkable plaques, seldom seen except in this condition. There is no close correspondence between the kind or extent of the tissue changes and the mental state. Plaques and neurofibrils are reported to occur also occasionally in the brains of mentally healthy old people.

PSYCHOLOGICAL.—The previous tendencies of the patients may greatly colour the symptoms. Obscure somatic preoccupations and disturbances in time appreciation lead often to fantastic delusions about eternity and what is happening in their body.

Symptoms.—Memory is poor for recent events; the extent of the damage may increase until only the recollections of childhood and early adult life remain. People and places are falsely identified with those once familiar, and transient pseudo-memories are invented. Events with a strong affective tone, especially if unpleasant, are remembered better. The memory of the remote past is not entirely spared; even matters of personal identity may at last be forgotten. Grasp and judgment, the capacity to follow a train of thought and to eliminate the irrelevant are faulty. Obstinacy and

perseveration go with a rigid adherence to old habits. Prolix and garrulous, the patient does not recognise how little interest there is for others in his repetitive and ill-arranged talk. He may partly cover its emptiness with long and sounding sentences; on the other hand, some patients become monosyllabic, because of their failure to find words to express themselves, and others again will use a word loosely associated with the one they are vainly seeking, or will quite seriously give a punning meaning to a word, and even act accordingly (*e.g.* whistling because "You said I could whistle for my money").

There is a narrow range of interests, in which food, possessions, and bodily well-being are prominent. Grotesque hypochondriacal delusions are common. Patients hoard rubbish and are angry if interfered with in this. On the whole, however, their affective responses are greatly reduced; they meet calamities with composure, partly due to their failure to grasp what has happened. Now and then they show depression and resentment at a slight, and may bear a grudge long after. Their activities are sometimes considerable, on the lines of determined rummaging and collecting; in others a dull inactivity is all. They become dirty and unable to look after themselves. This applies as much to those who are excited and active as to the inert. The former may fight against being fed and washed, and it is not possible to get them to understand what is being done. Delirium and confusional states are prone to occur at night, accompanied by fear and bewilderment. Sleep is bad, and often the patients busy themselves about the place all night long.

Legal difficulties arise through the heightened readiness to accept some suggestions (as in the matter of making a will, or giving away property), the poorer judgment and the lessened capacity to control sexual desire, which is sometimes seen in the early stages. Hoarding may lead to petty thieving. Occasionally the patient sets fire to the house during his nocturnal prowls.

The symptoms need not be obvious. Often the illness has so slowly developed that no one can say when it first passed beyond what is normal in old age. An apparent change of character—a kindly man becoming selfish, a respectable churchwarden assaulting little girls sexually—may usher it in; this is not so much a change in character as a release of primitive trends, hitherto controlled. The psychosis may take various forms—depressive, manic, and paranoid. In the *depressive* variety there is seldom retardation, the affect is rather empty, the patient is irritable, and hysterical symptoms may be commingled with hypochondriacal ones. Ideas of poverty, wickedness and disease are often grotesque in their exaggeration—the patient's urine drowns the whole world, his body is an undying shell of corruption, he is as tiny as a baby—and are monotonously reiterated. The *manic* variety is rarer: pointless activity and a diarrhoea of words, with silly boasting, may be accompanied by a disturbance of memory, giving a total picture of the Korsakoff type: it is sometimes called "presbyophrenia." Many of these patients have always been of hypomanic temperament; their illness may be only slightly progressive and not so severe as to call for hospital care. The *paranoid* variety is especially likely to occur in people who have always been of a suspicious turn of mind. They hide things because they feel surrounded by thieves, and then forget where they have hidden them;

their failing senses, especially of hearing, feed their distrust, and they project their awareness of sexual impotence or waning intellect. Hallucinations and delusions are mingled—gases are pumped into their room, their food is poisoned, people throw bombs at the house by night, greedy heirs are doing them out of their possessions. Some of these patients barricade themselves against their enemies or call in the police. Whereas the depressive and manic forms are commoner in people with corresponding heredity, this paranoid form is genetically often connected with schizophrenia, though the distinction between the three varieties is not a sharp or important one. The name “*involutional paranoia*” has been given to the chronic delusional condition of this type that may develop in single women between the ages of 40 and 52.

Bodily symptoms are those of old age, especially in the central nervous system, where it leads to a slow, careful gait, with short steps and legs wide apart, apraxia and poor co-ordination, tremulous rather whining utterance, small sluggish pupils, and occasionally epileptic seizures. The disorder of movement is conspicuous in the handwriting—pointed, small or erratic in size, and sometimes jerky and tremulous.

The conditions known by the names of Pick and Alzheimer are to be regarded as atypical senile or presenile psychoses.

Pick's dementia consists pathologically of a circumscribed cerebral atrophy, mostly in the frontal or the temporal lobe, or in both; the motor area, however, is seldom affected, nor are Wernicke's zone and the transverse temporal convolutions; other areas of the brain may be involved. Histologically, the ganglion cells are swollen and contain argentophil globules. There is a hereditary determinant. The onset, which is gradual, can be at any age from 40 onwards, but is usually between 50 and 60. Symptoms depend on the localisation of the atrophy. Memory and affect are not impaired till late; they are preserved at a stage in which the patient behaves stupidly—stealing, lying, or otherwise making a fool of himself. Spontaneous attention is poor; at first moody, the patient becomes dull and unresponsive; judgment deteriorates and initiative fails. Stereotypies, echolalia, and repetition of empty phrases, monotonous talking and laughing or singing, and outbursts of bellowing or whining appear in the later stages. There may be aphasia. Diagnosis is difficult during life; it may be assisted by an encephalogram showing the shrinkage of cerebral tissue from atrophy.

In *Alzheimer's disease* the senile plaques and neurofibril changes are very numerous. The onset may be between 40 and 60. Indefinite premonitory symptoms (headache, irritability, forgetfulness) are quickly followed by progressive dementia; aphasia and apraxia are prominent, though less coarse and sudden than in cerebral arterio-sclerosis. In the earlier stages the patients are in fair contact with their environment, and look as though they grasp much more than they actually can. Their deficiencies are shown up in writing and talking. As the disease advances they are less open to affective influences: they sink into themselves and say little. Stereotyped words or syllables and movements take the place of embarrassed remarks and gestures. In the aphasia there is a rather characteristic stringing together of syllables like each other in sound, but meaningless. Muscular rigidity may lead to contractures. The progress of this disease to severe

dementia is faster than in typical senile deterioration and the onset is rather earlier.

Prognosis.—This depends on the previous rate of development of the condition, the general physical health of the patient and any special pathological basis, *e.g.* Pick's atrophy, that may be recognised. Delirious and confusional phases may give a deceptively bad impression, for sometimes, after they clear up, the patient can resume his old routine tolerably well.

Treatment.—This consists in providing for the patient as easy and familiar an environment as possible. Whether institutional treatment is necessary depends not only on the mental impairment but also on the patient's social level and the willingness of his relatives to look after him well enough. Patients often fit surprisingly well into hospital life and routine. Drugs are best avoided, and caution is necessary in letting the patient have the aperients he demands to relieve his—mainly delusional—constipation.

2. CEREBRAL ARTERIAL DISEASE AND HYPERTENSION

The characteristic features here are the focal symptoms. All else is indistinguishable clinically from senile and other cerebral conditions; of course, pathologically many senile brains show arterial degeneration too. The early or mild symptoms of cerebral arteriosclerosis are the same as those of "essential" hypertension; and very like those of many benign melancholias of late middle age.

Pathology.—Atheroma of the cerebral arteries is accompanied by nutritional changes—softening—in the brain tissue, falling into three stages, *viz.* necrosis, degeneration (with masses of granular phagocytes, containing fats and hæmosiderin) and sclerosis (in which cavities and scars of glial—astrocyte—and mesodermal tissue take the place of the necrotic cells). (See also p. 1597.) The cortex on the convexity of the brain may show microscopic areas of perivascular gliosis, but no softening. It is not yet possible to correlate the mental and the cerebral changes in these psychoses, except for the focal lesions.

Symptoms.—Since "essential" hypertension often precedes definite vascular disease and itself produces mental symptoms, a description of these symptoms serves also to describe the earlier stage of cerebral arterial degeneration. Along with headache, giddiness, tinnitus, faintness and insomnia, there may be disturbance of speech and writing—the former becoming slow and at times indistinct—and transient pareses and apraxia. Certain traits of personality may be intensified: the patient becomes irritable, egotistic, moody and easily tired, his conversation lumbers along where once it moved easily: he is depressed or paranoid; but there may be wide variation in the intensity of these changes, which are by no means always found. Brief phases of disturbed consciousness, lasting up to three weeks, may suddenly occur either in a form very like the "absences" of the epileptic, or as twilight states with hallucinations, ecstasy, incoherence, disturbed motility and agitation.

After this stage of neurasthenia and episodic disturbances, the patient with cerebral vascular disease may begin to have trouble in finding words: he perseverates a little, and is at a loss when anything unusual is required of him. His depression and hypochondriacal worries increase, he is distressed

by his own slowness and failures, and may attempt to kill himself. Emotional control falls off so that he weeps and storms when he would rather be calm. Nihilistic ideas may abound—his bowels have not been opened for six months, his trunk is a hollow cavity. Nocturnal delirium is frequent. Aphasia and apraxia are commonest after a focal complication.

The most important feature is the way the patient continues to look normal and sensible when already mildly demented. Sometimes transfer to the strange surroundings of hospital is too much for the hitherto well-preserved outward normality, and the patient goes to pieces, as he also may if he has to give up his usual work or move house.

Diagnosis.—Because a patient has generalised arterial disease, it does not follow that any neurotic symptoms he may show are due to the cerebral vessels being thus affected. Unless there are definite focal symptoms, or evidence of dementia, it is unsafe to hold the cerebral arteries responsible and to give a prognosis based on this. There is no known means of distinguishing many benign "neurasthenic depressions" and involuntional hypochondrias from those due to disease of the cerebral vessels. If there has not been any history of such tendencies until an attack at the age of 60 odd, the probability that it is an organic vascular disease is much higher. The distinction is all the more difficult because so many unstable persons develop arterial disease in later life; especially those prone to anxiety and other affective disorders. Neurological findings (see p. 1599) may be decisive in a doubtful case. The condition of the retinal arteries is not a reliable guide.

Course and Prognosis.—In definite cases of cerebral arterial disease with mental disorder the prognosis is necessarily bad, though the mental symptoms may only progress slowly, and the patient live another ten or twenty years. Much will depend on such sudden accidents as thrombosis or hæmorrhage. An episodic confusional state, perhaps even one produced by drugs, may suggest a needlessly gloomy prognosis. In cases of "essential" hypertension, the course of the mental illness is dependent on the general disturbance, and is often quite favourable. Symptoms that are apparently hysterical, occurring for the first time in middle life, are of bad omen.

Treatment.—Besides the general medical care of such patients, not a little can be achieved by psychiatric methods. In the early stages, where there is much anxiety and depression, too energetic physical investigation and treatment may do harm: reassurance and sedation can do much good. The less said to the patients about their blood pressure and their arteries the better. They should keep at work and in their accustomed surroundings as long as they can, unless an acute phase of the illness or depression intervene. Emotional upsets often aggravate their condition than physical ones, so they should be cushioned against such jolts. Their depression may necessitate hospital care, especially because of the risk of suicide, or because they are too irritable and neglectful to be at home any longer. If there be dementia, even of mild degree, the patient will probably remain in a mental hospital once he has gone there. It is, however, not easy to be sure about mild dementia being present; it can be counterfeited by passing disturbances, e.g. emotional ones.

3. HUNTINGTON'S CHOREA

(see p. 1703)

C. SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

Only the mental symptoms will be described here. Hypochondriacal and depressive reactions sometimes follow infection, or the risk of infection: such psychogenic illnesses do not belong under this rubric; occasionally, however, a patient's anxiety lest he be developing neuro-syphilis turns out to be justified. A syphilitic neurasthenia can occur in the early stages of the disease, due to a mild meningitis. The more severe meningo-encephalitis—*cerebral lues*—may be accompanied by disturbance of consciousness, even to the point of delirium or mild dementia: loss of initiative, euphoria or moroseness, poor judgment and impaired memory may persist and the patient be aware of them in greater measure than he is in general paralysis. These conditions are often complicated by the signs of premature arterial degeneration in the brain. The psychoses that accompany tabes are due to syphilitic changes in the brain, often complicated by alcohol, trauma, heart and kidney disease, and other exogenous factors; there are also depressive hypochondriacal reactions to the pains and other disabilities which the patient suffers.

GENERAL PARALYSIS OF THE INSANE.—Dementia is the constant sign of this mental picture; the old descriptions of a "classical" course with an expansive onset are fallacious, but general dementia is almost certain to occur in every case that is not treated early. All the other symptoms are either neurological and focal, or due to the patient's constitutional predisposition and previous experiences.

The dementia may at first be quite undetectable as such, because it appears under the deceptive guise of a neurasthenia, melancholia or mania; only gradually does the intellectual impairment become manifest. In the beginning of general paralysis, which is seldom abrupt (though it may need a careful inquiry to verify the prodromal symptoms), "functional" syndromes can be so "typical" and organic changes so slight that the most expert psychiatrist is misled; only by physical and serological examinations can he avoid a blunder. A faint degradation of personality, a lapse in social refinements may be the first indication of what is wrong. Then memory for the events of yesterday and last week becomes less trustworthy, what seemed at first a trivial absence of mind becomes serious incapacity, and yet the patient remains serene and outwardly indifferent to his lapses. As in senile and arteriosclerotic dementia, he may be all right so long as he is in an accustomed rut, but a holiday or a change reveals his infirmity. His mood and interests as the illness goes on become dull or labile, his rages are fleeting, his activities fussy; if, however, he is in a manic excitement, with little dementia as yet, the affective changes can be violent, and indeed dangerous, just as in a depressive phase the patient may kill himself. Sleepy and slow, careless about social usages, inattentive and ignorant of what he once knew well, the more demented patient cannot escape recognition as having an organic cerebral affection. Elementary problems in arithmetic and questions of general information are more than he can cope with. He gives easy assurances that he can do them, or puts his questioner off with airy explanations (*e.g.* that he has not had his spectacles by him lately); when pressed, he makes bad mistakes or becomes angry. The extent of his failure will, of course, depend not only on his dementia, but on his previous

intelligence and habits (*e.g.* a bank manager retains the capacity to do mental arithmetic when much else has gone). Inability to receive new impressions and to relate them to earlier memories co-operates with impaired judgment to give a gross but patchy and fluctuating amnesia. Because of these disturbances, and especially the bad judgment, patients may commit offences, ruin themselves by grotesque extravagance and brush aside facts that stare them in the face. They will put up with restrictions on their freedom, forgetting their protests soon after making them; silly reasons are sufficient for their compliance, and a tactfully offered cigarette or joke may divert their thought and feeling from some serious matter that angers them. Their delusions are due to the same disorders of memory and judgment, coloured by their general personality; sometimes they are confabulations, rationalisations for their having forgotten or spoilt something. If the patient had in health tendencies to euphoria and expansive behaviour, grandiose delusions and boasting will be to the fore. It is, however, not uncommon to find a fatuous euphoria, though there had not previously been affective swings and hypomania; in such patients one finds abundant proof of gross impairment of judgment, especially shown as defective insight. The most advanced dementia appears as a helpless, vegetative, bedridden state, sometimes accompanied by gross focal symptoms, such as aphasia and agnosia. The physical symptoms (see p. 1638) are much intermingled with the mental ones, as in the patient's clumsy movements and disturbed speech and handwriting: thus, in his writing he leaves out letters, syllables and words, repeats and transposes them, messes the paper with blots and sputters, writes across the lines, puts in meaningless strokes and leaves his mistakes uncorrected; the tremulous script shows interruptions in the usual smooth alternation and tempo of movement, the letters are of very uneven size and ill spaced. Articulatory and aphasic disturbances may affect the sense, intonation, timbre, rhythm and precision of utterance; they must not be evaluated in diagnosis, any more than the writing disorder may, without regard to the patient's previous normal script and speech and the circumstances under which he was writing or talking, since people, habitually untidy in their enunciation or handwriting, can exhibit many of these symptoms when tired or in a hurry.

Besides the above, atypical mental pictures may be seen either ordinarily or as the outcome of treatment with artificial fever. Paranoid states, hallucinosis, a Korsakoff syndrome, epileptiform excitement, hysterical disorders and catatonic symptoms of every kind (except *flexibilitas cerea*) may occur. Hallucinations are uncommon, except during fever or after malarial treatment; in the latter case they are often of paranoid colouring. Not the expansive form, but a simple progressive dementia is by far the commonest clinical picture; depressive, confusional, and hyperkinetic states are almost as frequent as the expansive.

In the "Lissauer" form the slowness of the dementia is remarkable in comparison with the conspicuous focal symptoms, such as the seizures without convulsions or loss of consciousness.

The effects of treatment upon the mental state are of great social moment. In a majority of the cases who do well the personality has the edge taken off it, there may be less initiative and force in mental activity, and emotion may be less controlled, especially in the proneness to anger or to frivolous

levity, yet the patient is able to return to his former work, even though it is responsible and complex; he could scarcely, however, except in the most favourable cases, learn a new job or adapt to new and exacting situations.

In the "*juvenile*" form there may be premonitory symptoms of excitability, grizzling, timidity and backwardness at school. Gradually the symptoms of dementia become plain, and if the onset be early enough, symptoms usually found in severe mental deficiency naturally appear, such as rhythmic or iterative movements, grimaces, repetitive chewing and sucking of an automatic kind, great restlessness and screaming attacks. Simple dementia is the usual form; grandiose ideas are exceptional. If the illness begins before the age of 10 or 11 years speech and writing may be completely lost, or reduced to a senseless smattering.

For *prognosis* and *treatment*, see pp. 1640, 1641, and 1653.

D. OTHER CEREBRAL DISEASES

LETHARGIC ENCEPHALITIS.—The mental disturbance of the acute attack may merge into a hyperkinetic excitement, with choreiform and athetoid movements, insomnia, generalised pains, mild delirium and, occasionally, catatonic symptoms: this seldom lasts more than a few weeks. There may be subsequently a neurasthenic fatigue and irritability with headaches and poor sleep. The distinction between what is neurological and what is psychiatric in the symptoms could scarcely ever be more difficult than in this disease. The motor disturbances, such as oculogyric crises, are not merely responsive to emotional and other psychogenic influences, they are inseparable from concomitant mental happenings (*e.g.* the surging up of anxiety or obsessions), and whole patterns of complicated behaviour, *e.g.* breathing, may be involved. The motor rigidity of the patient's Parkinsonian state may be paralleled by a lack of the normal drive and fluidity of thought or behaviour. Memory, however, and grasp are unaffected. The obsessional symptoms sometimes occur quite apart from oculogyric crises, and may greatly distress the patient. Depressive phases may result in suicide, which is fostered, as it were, by the keen appreciation which many patients have of their ruined careers and their almost imbecile appearance, so different from what they were and, indeed, from what they still know themselves to be. Paranoid, and especially schizophrenic, symptoms may develop in the later stages.

The younger the patient the more likely is it that he will develop disagreeable anomalies of personality, and have attacks of restlessness or even be permanently restless. Many children and adolescents after their acute attack become social problems: they play stupid or cruel tricks, they set everyone they can by the ears, they may steal, behave sexually in an outrageous way or accuse others of sexual offences against them. Their activity is not always purposive, nor always antisocial; they make the same impression as a monkey might who is sometimes mischievous but always on the move. There may be no Parkinsonism in these cases. The prognosis is not good, and they almost always do better when subjected to the regime of an appropriate institution; they do badly at home or in places where

what may be termed normal delinquents and "social problems" are cared for.

SYDENHAM'S CHOREA.—The usual mental changes here are lability of affect and irritability. These are seen as naughtiness, outbursts of anger or crying, resentment at sudden noise or light; in others there is lessened spontaneity, often masked by the choreic movements. In more severe cases, especially in older children, these changes are accentuated; in the fleeting phases of anger or terror there may be slight delusional trends. Still more severe forms, with delirium, hallucinations, delusions of persecution and much excitement, are seen in adults, *e.g.* in chorea gravidarum.

The tics and compulsive utterances (Gilles de la Tourette's disease) which may follow chorea are evidence of the interplay between hereditary, psychic and structural factors. Chorea is more prone to occur in those whose families show nervous disorders, especially schizophrenia. The motor after-effects, especially tics, appear and disappear under emotional influences; they are also conditioned by the original choreic disturbance of neuromuscular function. The obscene ejaculations of la Tourette's disease are dependent on much the same articulatory and respiratory hyperkinesias as are the breathing spasms of encephalitis lethargica, though they are also dependent on psychological tendencies and experiences. They illustrate how psychological influences work through available bodily structures and functions, whether morbid or healthy. The obsessional element in this affection is comparable to that in encephalitis lethargica.

DISSEMINATED SCLEROSIS.—Slight deviations from mental health are frequent, but obvious ones rare in this disease. Affective lability may be conjoined with a slight disorder of judgment, so that a baseless euphoria develops, but this is not universal, and many of the patients are depressed. Acute outbursts of excitement, hallucinosis or delirium occur in a few cases, and dementia in the advanced stages. The most important mental disorder in them is that which appears as hysteria. A hysterical personality has not been present in these patients before the disease began, and the symptoms are in that respect only dubiously hysterical: they do, however, in other respects conform, in that they can be evoked psychologically and removed psychologically; they may centre on, and elaborate, actual anomalies, *e.g.* of movement or sensation, and may still yield to hypnosis or other psychological measures. They can greatly confuse the diagnosis.

SCHILDER'S DISEASE.—In this disease profound dementia gradually develops along with the blindness, deafness, aphasia and agnosia and other focal symptoms. In the juvenile cases there may be at first disturbances of behaviour like those of juvenile encephalitis lethargica.

PARALYSIS AGITANS.—This may be accompanied by hypochondriacal depression. Sometimes this is an expression of the cerebral disease which also causes the Parkinsonism, and in that case the prognosis is bad; sometimes it is a recurrence of depressive attacks which have occurred at times of stress earlier in the patient's life, and then the outlook is fairly favourable. Senile dementia is, of course, not infrequent in these elderly patients.

CEREBRAL TUMOUR.—Apart from any aphasia and apraxia, the mental state here is more closely related to general intracranial tension than to any local disturbance. The size and rate of growth of the tumour are therefore important in this regard. If rapidly growing, there is more disturbance of

consciousness, with impaired memory, disorientation, incoherence and, sometimes, hallucinations and confabulation; this clouding of the mind fluctuates a good deal. In more slowly growing tumours, lucidity is preserved and change of disposition is the prominent feature. The patient's earlier tendencies get freer play, unsuspected ones appear, and a series of foolish investments, for example, or homosexual escapades may for years divert attention from the organic disease. The moria, or fatuous wit and cheerfulness, often attributed to frontal tumours but also found in other cerebral diseases, may give the impression of being a hysterical pseudo-dementia; other apparently psychogenic symptoms may prove misleading. A straightforward depressive attack can occur, or indeed any "functional" syndrome.

Hallucinations may depend on a focal lesion, as in the cases in which they are limited to the hemianopic field, or are solely of taste and smell.

In CEREBRAL ABSCESS the mental symptoms are those of tumour with or without others due to meningitis. In ACUTE MENINGITIS there may be delirium, preceded during the prodromal stage by irritable apathy, and followed by months of moody neurasthenia.

CEREBRAL TRAUMA.—After concussion there is retrograde amnesia; the extent of this and the rapidity with which it diminishes depends on the amount of cerebral damage. In rare instances delirium ensues: it has little that is characteristic, and is more frequent in alcoholic and elderly people; a Korsakoff syndrome may develop. Twilight-states are rather more common; during them acts of violence may be committed, as in epilepsy, and afterwards quite forgotten. Traumatic epilepsy may follow. The later changes in personality are commonly those that may be found lingering after any toxic or other structural impairment of the brain. But sometimes the disturbance of consciousness is more persistent, the intellectual damage greater, the deterioration progressive; in such cases there is usually cerebral arterial disease, an unrecognised alcoholism, cerebral tumour, G.P.I., or some other complicating factor. In predisposed persons the cranial injury may be responsible for a melancholic attack, schizophrenia, or other "functional" syndrome; the prognosis is usually good even if the illness lasts many months.

Hysterical symptoms occur frequently after cerebral trauma. This is partly because of the site of the injury, which favours vague physiogenic symptoms that respond readily to emotional and other psychological influences. Many of these symptoms are, however, produced by psychical rather than physical mechanisms. Not injured cells, but mental attitudes are at the bottom of the tremblings, faintings, weakness, paræsthesiæ and other troubles so often the sequel of a trauma in itself little likely to have such effects. They are not responses to the actual injury, but to the situation created by the injury. Compensation may play a large part in this. It is as unwise to dub all such vague post-traumatic phenomena hysterical as to attribute them entirely to the direct injury. If there is slight amnesia of the typical kind, with difficulty in concentration and headache, it is fairly probable that these are physiogenic residues; if there has been an interval between the actual concussion and the appearance of the indeterminate symptoms, and an adequate psychogenesis (*e.g.* claims for compensation, with repeated medical examinations, and patent uncertainty among the experts) the condition is likely to be hysterical. Much will, of course, depend on the neuro-

logical and other findings, including the demonstration of localised lesions; thus, damage to the frontal lobes may much change the personality, and in other sites be responsible for an apraxia, say, or a visual defect.

EPILEPSY.—Although the motor seizure is the chief symptom of epilepsy and the only decisive one in diagnosis, there are minor or equivalent symptoms, as well as delirium, twilight-states and dementia, to be included among the mental disorders of this illness. The minor symptoms are much rarer in symptomatic epilepsies than in the "idiopathic" form.

Instead of a major fit the patient may become unconscious; or he may pass into a twilight-state in which for a few minutes or longer he wanders about in a dazed way and does inappropriate things, having afterwards complete amnesia for all this; or there may be a sudden interruption of action and speech, during which the patient remains immobile or makes some automatic or aimless movements. Epileptic furor is a delirious state in which acts of violence may be committed: it lasts often for several days, is accompanied by disorientation and hallucinosis, and is much rarer than is popularly or forensically supposed. All the states of disturbed consciousness mentioned above are most often seen as equivalents for a seizure; the twilight-states, however, may precede the motor attack, follow it, or be accompanied by a few violent clonic movements.

Apart from their seizures epileptics are prone to swings of mood—towards anger, shallow sentimentalism or depression—which may pass over into a fugue, during which the patient wanders a long way from home.

The likelihood of dementia later cannot be inferred from the symptoms of the epilepsy, except that it is greater if attacks occur very often. Apparent dementia may be the result of intoxication with bromide, or of the idleness and sterile life in an institution. When there is genuine dementia, it begins as a faint loss of interest and concentration, with increased sensitiveness to supposed slights, then memory falls off somewhat, the trivial and the important are muddled together, and the patient talks with much circumlocution; he is fond of needless system, assumes and parades virtues he has not, *e.g.* an intellectual bias or a devout spirit, and is childishly pleased when anyone praises him. Later, a profound dementia may supervene, but this is not common; it is open to question whether the changes of character just described are necessarily part of a dementing process. It is certain that many epileptics who exhibit some of the most disagreeable features of this sort never become plainly demented, and that many severe epileptics are free not only from dementia but also from these traits. There is ground for regarding this impulsive, pretentious, fawning and snarling way of some epileptics as partly a variable expression of their constitutional predisposition (to which the motor seizures are likewise due), and partly as a reaction to their situation. Consequently it is much less evident, or not evident at all, in those who in spite of their fits live comparatively normal lives.

E. INTOXICATIONS

1. ALCOHOLIC DISORDERS

Alcohol is so permissible and trusted a poison, so easy of access for those who wish to escape from their troubles, that it is resorted to in excess

by maladjusted persons of every type ; consequently its effects may complicate or be complicated by the psychopathic anomaly which favoured the taking of the drug, *e.g.* episodic excitement or depression, anxiety, cerebral arterial disease, syphilis, paranoid states, epilepsy, hysteria. The acute effects of a single dose of alcohol are either the well-known phenomena of intoxication, or an excitement (*mania a potu*) sometimes with clouding of consciousness. The excitement is commoner in people with cerebral trauma, arteriosclerosis, epilepsy, or unstable hysterical personality, and in them may lead to acts of violence ; rarely it may occur in normal persons who have taken alcohol when they were exhausted or upset.

In chronic drunkards, a dementing *demoralisation* can occur. Their narrowing of interest, superficiality of thought, weakness of memory and moral decrepitude are reminiscent of what happens in many epileptics and some early general paralytics. The crudeness and even brutality of their conduct is in ill accord with their maudlin prating about virtues and their pothouse jollity. The mood of these men can be as labile as their abandonment to it is constant : they pass from rage to weeping, and laugh soon after, with no shame for themselves and no thought for the miseries they put on their families. Such degradation is of course far from being the rule : some chronic alcoholics become only cheap editions of themselves, with their former qualities underlined or smudged rather than defaced ; they are perhaps weak and irritable, untrustworthy or lying, but not given to savage fury, nor grossly damaged in judgment and social feeling. Some of them develop delusions, especially of jealousy. They collect, as paranoid people of other kinds do, scraps of alleged evidence which they piece together to prove their suspicions right ; complicated delusions of persecution, however, they rarely develop. Sometimes the delusions of jealousy fade as the patient gets more and more facile, but more often they persist as a chronic insanity and are of the greatest danger to the suspected wife ; murder is not unknown in such cases. The nature of the delusions is to be attributed in part to the lessened sexual potency of chronic drunkards and to the domestic wretchedness and aversion they often create, as well as to the same causes as in "functional" paranoid states, where such delusions are also common, especially in middle life.

The symptoms of *delirium tremens* would appear to differ in nothing but severity from the essential symptoms of any delirium (see p. 1824). Some observers, however, deny this. The anxiety amounts to terror, mixed oddly enough with euphoria ; optic and cutaneous hallucinations are vivid and restlessness can be extreme. There is almost complete sleeplessness, and much disorientation as to time and place, but not as to personal identity. The patient's attention wavers between his hallucinated and his actual surroundings, but can usually be caught and held for a few moments. He is very suggestible, as most chronic drunkards are ; pressing on his eyeballs, for example, will very likely make him see whatever one tells him he sees, and he will read aloud from a blank sheet if one wants him to. Among the visual hallucinations may be miniature ones (*micropsia*), and many illusionary perceptions. The content of the hallucinations changes rapidly, and a false perception in one field (*e.g.* a vestibular one) tends to evoke others (*e.g.* of sight, touch, or hearing). Insight is commonly lacking ; afterwards there is patchy amnesia for what has happened in the

delirium. The death rate, with adequate treatment, has been about one in seven ; and of those who die most of the men are under 40, and most of the women under 45.

In *acute alcoholic hallucinosis* auditory hallucinations of a persecutory kind are prominent and consciousness is not notably clouded. It is rarer than delirium tremens, and is more prone to follow a bout or orgy of drunkenness. The patient is frightened, but not obviously out of his mind ; he is correctly orientated and may be able to go about his business for days. Auditory hallucinations are vivid and insistent, after a premonitory phase in which there are sensitiveness to sounds, and roaring, singing, hissing, etc., in the ears. Tormenting voices, sharply localised but seldom fastened upon bystanders, abuse, threaten or discuss the patient : they may say his wife plays him false, order him to kill himself, describe his every movement, especially at private moments in the bath or lavatory, cast up his more shameful secrets at him, shout his thoughts aloud. There may be many voices, of men, women, and children, all talking together and perhaps rising and falling in the same rhythm as his pulse. They are so real that the patient answers them ; he may be in doubt about the presence of his tormentors and may shout back insults to see if a blow will follow from the owners of these evasive pursuing voices. Hallucinations of sight and other senses are far less prominent than those of hearing ; cutaneous ones, e.g. of being sprayed with a cold liquid, are not uncommon. Delusions are usually inconspicuous : they are as a rule attempts to account for the hallucinations, and they commonly fade out of the picture or pass into a chronic persecutory disorder. Flight or acts of violence may result from the patient's fear or anger. Usually it is a matter of only 2 or 3 weeks before the hallucinosis clears up, if no further alcohol be drunk ; sometimes, however, a delusional state, more rarely a Korsakoff picture, supervenes in predisposed persons. After recovery, there is little or no amnesia for the events of the hallucinosis. Relapse is to be feared if the drinking goes on.

The *Korsakoff* syndrome is not invariably associated with polyneuritis. Nor, as stated on p. 1825, is it limited to alcoholism ; it can follow other severe chemical and mechanical injuries to the brain. In alcoholics it is commoner in middle life, developing either insidiously in the course of chronic alcoholic demoralisation, or after delirium tremens ; women are especially prone to develop this syndrome after the delirium. The symptoms have already been described. The disorientation, superficial appearance of clarity, incapacity for initial perception and subsequent recall (extending often to most of the material of memory) yet with retention of some capacity for learning by repetition, along with confabulation, dullness of emotion and initiative, and grossly impaired judgment make a striking picture. Complete recovery is on the whole uncommon, occurring in less than a quarter of all cases. The mortality rate is higher in women and older people, in those with acute onset and with a red-cell count below 3,000,000, or with a rise in the protein content of the C.S.F. It does not correlate with the severity of the peripheral neuritis.

Chronic Delusional States have been referred to above ; they are sometimes called alcoholic paranoia, but inappropriately so ; jealousy is the commonest and most dangerous feature. Alcoholic epilepsy has been described. It is no more than a symptomatic epilepsy, often atypical ; some-

times in unstable hysterical patients it may be brought about through over-breathing when intoxicated.

Diagnosis.—The diagnosis of alcoholic psychoses must depend much more on a history of drunkenness in any patient than on his clinical psychiatric features, none of which are limited to alcoholic disorder. Since, however, alcohol is far the commonest cause of most of the toxic abnormalities described, it can be safely presumed in some cases in which the certain history of addiction is unobtainable.

Differential diagnosis, so far as ætiology is concerned, will turn on somatic findings, including the results of chemical and biological tests. If the form of the disorder is in question, the chief diagnostic difficulty arises with acute hallucinosis and the chronic delusional varieties. A hallucinosis of similar type can occur in schizophrenia and in affective disorders, but in the latter is recognisable by the ideas of self-reproach expressed; the differentiation from schizophrenia is difficult, since in many of the cases the progress of the disorder is towards a chronic schizophrenic psychosis, and one may suppose that in these patients the intoxication had activated, as it were, the same mechanisms as those involved in schizophrenia, or had complicated a schizophrenic illness. This applies also to the chronic psychosis with delusions of jealousy. There is no value in differentiating carefully the clinical varieties of alcoholic psychoses, since they overlap.

Treatment.—Prophylaxis is the main thing. The incidence of alcoholic psychoses in London has fallen to one-third of what it was before the War of 1914–1918; and this may be attributed almost entirely to social influences, of which the increased cost of alcoholic drinks is the most effective. Individual prophylaxis is scarcely to be considered, save as a by-product of psychiatric treatment, since a great proportion of unstable persons are potential drunkards, and in any case we cannot yet tell which alcoholics will become mentally ill through their drinking. Social prophylaxis is so immeasurably better in forestalling alcoholism and the psychoses and degradation that sometimes spring from alcoholism, that deliberate individual prevention is here negligible.

When alcoholism is itself to be treated, independently of its ill-effects upon mental health, the problem is that of any drug addiction. Absolute removal of the drug is essential, yet this cannot be done unless the patient, the treatment, or the environment is exceptional. A suitably exceptional environment can be provided by getting the patient into a hospital or home where he cannot obtain the alcohol he desires, but the other requirements are more difficult to meet. The exceptional patient who after years of excess can put aside alcohol while it is within his reach is as rare as the treatment that can bring him to this state; and when such a change does occur, a great emotional upheaval, *e.g.* bereavement, religious conversion, fear of death, has usually led to it. For the most part, treatment of alcoholism without restrictions upon access to the drug is a failure; the restrictions must at first be imposed from without, not left to the patient's self-control and judgment. He should remain in the hospital or home for a year at least. Psychotherapy is an essential feature of the treatment in those cases in which inner struggles and neurotic disabilities have been the basis for the addiction; it must, however, be conjoined with social and other measures (see p. 1815). Special chemical methods have little or no value.

The grosser mental disorders due to alcohol need hospital treatment. Delirium tremens should be treated as far as possible without hypnotics, which have little effect upon the excitement and sleeplessness unless employed in dangerous doses; if any, paraldehyde or hyoscine should be used. Circulatory failure and accidental self-injury are most to be guarded against. The continuous bath at body temperature is sometimes beneficial; otherwise the patient should be in bed with a minimum of necessary restraint, the company of an experienced nurse, and ensurance of adequate diet—mainly fluids and glucose and large amounts of vitamin B₁. No alcohol should be given. Occasionally lumbar puncture is helpful.

Especial care must be taken against the early discharge from hospital of alcoholics with delusions of jealousy. If they have been certified, they may add a deep resentment on this score to their other grounds of morbid hatred, and there is grave danger that they may, if they resume drinking, attack their wives murderously.

2. MORPHINISM

Only the effects of this belong among the organic disorders; its causes, and the incapacity of the addict to escape from it, are due to social and intrinsic factors, not to any physical damage. Weak, unstable, unhappy people, *e.g.* many homosexuals, are most likely to become addicts; it is rare to meet an addict who has not shown pronounced psychopathic traits before his addiction began; and few of those who profess to have been seduced into the habit by more or less injudicious administration of morphine for some pain they had, are in that telling the whole truth. Yet it is a wise caution that withholds morphine from all chronic disease that is not hopelessly progressive, and hesitates to prescribe it at all for those whose personality or opportunities make the risk of addiction greater.

Symptoms.—These are not at first noteworthy, unless the patient be seen during the next 2 or 3 hours after he has taken his drug. The symptoms of withdrawal, sometimes severe, are more likely to occur in those whose tolerance has been raised by the habit; they consist of yawning, sneezing, overflow of tears and saliva, fullness in the head, then restless movements, malaise, twitching in the face, tremors, palpitation, indigestion, vomiting, diarrhoea, strangury, sleeplessness, and circulatory upset which may go on to collapse.

It is difficult to judge how far the drug itself is responsible for the demoralisation that is met with in chronic morphine addicts; probably as important in causing it are the psychopathic personality of the addict, and the underhand life he must lead. Laziness and lying are frequent, and the patient may resort to subterfuges, or even crimes, to get his drug. Dementia does not occur; delirium is rare. The physical effects of chronic morphinism are dryness of the skin, hair, and nails, constipation and anorexia, partial impotence, and poor resistance to infection.

Prognosis.—This is poor as regards recovery from the addiction. The more normal the patient's personality, the better the outlook. After apparent cure, however, relapse is frequent, and the outlook is then correspondingly worse unless the patient can be stopped from getting the drug. Many morphine addicts also take alcohol, cocaine, and such other drugs as they can get. Suicide with morphine is not uncommon, for obvious reasons. Death

is sometimes the result of cutaneous infections, especially when the patient is grossly undernourished.

Treatment.—This must be in an appropriate institution; general hospitals seldom have the necessary facilities. Treatment at home is bound to be a failure. It should be impossible for the patient, however skilled in stratagems, to get hold of morphine. He should, if possible, contract to stay for at least 2 months. The withdrawal of the drug should be abrupt and total, except in very debilitated patients; “tapering-off” prolongs the distressing period of withdrawal symptoms and gives opportunity for the patient to develop psychopathic reactions and dodges. If the patient’s condition demand a gradual withdrawal, this need seldom extend over more than a fortnight. When an abrupt end has been put to the taking of morphine, the rigours of the first 4 or 5 days (after which the worst is over) can be alleviated by sedatives in fairly large doses, copious fluids, warm baths, massage and fresh air; gastric lavage and alkalies help, and for circulatory symptoms caffeine may be given, with small doses of morphine also in very severe cases. After this phase is past, sleeplessness may still be intractable: in giving sedatives or hypnotics for this, barbiturates and paraldehyde, with occasional doses of hyoscine, are the safest, but should be used sparingly, with frequent changes and, it need hardly be said, complete refusal to let the patient know what he is having. Psychological treatment is of great importance, but there is no specific technique applicable to this addiction. To be successful, the psychological treatment requires the co-operation of the patient’s family as well as of the patient himself, and it will be wise for him to keep in touch with his physician for years. The great difficulty of getting the drug in this country, because of the vigilance of the Home Office, is an immensely favourable factor after active medical treatment has ceased. It is wise for the patients to eschew alcohol and, of course, all hypnotic drugs.

3. OTHER INTOXICATIONS

Cocainism is very rare in England. The causes and symptoms are similar to those of other addictions, *e.g.* alcohol and morphine. Deliria, hallucinations, Korsakoff syndrome, or demoralisation can occur: in the former conditions microptic and cutaneous hallucinations, *e.g.* of bugs under the skin, are prominent. A paranoid schizophrenic state sometimes comes on, usually clearing up after the drug has been stopped. In treatment what was said of morphinism mostly applies here, though withdrawal symptoms are far less severe.

Bromide intoxication is common, and often unrecognised. It is probably now in England the most frequent cause, after alcohol, of mental disorder due to a drug. All the organic syndromes can occur, usually as complications of a pre-existing disorder for which the bromide has been prescribed. A delirium and a paranoid confusional state or lachrymose amnesic syndrome are the usual forms. In the more long-standing and severe forms cachexia, circulatory failure, and even death may occur. Acne and other physical signs of intoxication or idiosyncrasy may not be evident. Diagnosis rests on the history and the amount of bromide found in the blood, more than 50 mgms. per 100 c.c. being indicative of a considerable intake or retention of

bromide. Treatment consists in complete withdrawal of the drug, promotion of its excretion by giving sodium chloride and fluids in large quantities, and general physical and psychiatric measures.

Barbituric acid derivatives, such as barbitone (veronal) and phenobarbitone (luminal), can in rare cases lead to apparent euphoric dementia, likely to be mistaken for general paralysis because of the ataxia, tremor, articulatory disorder, and other neurological signs. Recovery is the rule when the drug is stopped. Picrotoxin may be needed for acute poisoning. For the addiction itself, essentially the same problems and methods of treatment are in question as with other drug addiction. This applies also to *ether*, *chloral*, and *paraldehyde*.

Mercury and *lead* poisoning may lead to mental disorder (see pp. 376 and 366), *manganese* to a Parkinsonian syndrome with compulsive symptoms (reminiscent of encephalitis lethargica) and a mild paranoid or euphoric dementia; and *benzene* or *carbon disulphide* may cause delirium.

Acute carbon monoxide poisoning in rare instances leaves behind severe mental disorder of the amnesic-aphasic kind, which may not become apparent until several weeks after the recovery of consciousness. More commonly, it results in a clinical picture almost indistinguishable from hysteria; this may take months to clear up, and is in no wise benefited by psychotherapy. Chronic poisoning by small quantities of carbon monoxide causes neurasthenia.

F. INFECTIONS AND EXHAUSTIVE DISORDERS

1. INFECTIOUS TOXÆMIAS

Delirium and a Korsakoff syndrome are the more acute, and *naurasthenia* the milder, signs of mental disorder due to an infectious fever. In many of the cases, however, in which mental disorder is attributed to "sepsis", or other infection, either the mental changes are unconnected with the infectious process or there has not been an infectious process, as is often found when one inquires into an alleged attack of "influenza" and finds it was nothing of the kind. There are three possibilities: the mental changes are mainly due to the infection; they are independent of the infection; they are partly due to the infection and partly to other, usually constitutional, causes. The depression that occurs in and after many infections is usually of the third category mentioned; delirium instances the first possibility; and the second is often exemplified when some non-organic syndrome is put down to "latent sepsis" in the bowel or the tooth socket or some rather inaccessible cavity. It is not that infectious toxæmia is always innocent of doing this sort of psychiatric harm, but that it is far too often charged with the offence when it is blameless.

Wherever a delirium or other mental disturbance of one infection differs from that of another, e.g. the delirium of typhoid from that of pneumonia, the difference lies only in the severity and duration of the physical effects of the intoxication and in the peculiarities of the affected person; no mental symptoms specific to any one infection can be demonstrated. Among the individual peculiarities just mentioned must be included a constitutional

predisposition or readiness to respond with symptomatic psychoses to mainly physical ills.

There are a few infections that hardly ever cause mental disturbance, *e.g.* tetanus and diphtheria; others do so by their local cerebral incidence, *e.g.* malaria or encephalitis lethargica. *Tuberculosis*, from its chronicity and its occasional incidence on the central nervous system; has a special position. Its treatment, moreover, especially in the pulmonary form, necessitates an abnormal, unsatisfying life for a time, and this with the toxæmia seems to be responsible for euphoric or anxious restlessness in which erotic tendencies and irritability are often prominent. *Spes phthisica* is partly attributable to toxic euphoria, in part it is a form of over-compensation for fear.

2. EXHAUSTION AND INANITION

These, especially if conjoined with some shattering experience—an earthquake, an invasion, a bereavement—bring about severe mental disturbance, *e.g.* a twilight-state or a delirium. *Hemorrhage* and cachexia may be responsible for “light-headedness,” as in advanced carcinoma, or after a severe operation.

G. METABOLIC, ENDOCRINE, AND VISCERAL DISEASE

1. METABOLIC DISORDERS

Various metabolic disorders can similarly, *i.e.* non-specifically, affect mental health. *Diabetes*, for example, which is especially frequent in families with a predisposition to affective psychosis, may be accompanied by transient phases of depression, anxiety or excitement which correspond to changes in the blood-sugar level, or a ketosis may be ushered in by mild delirium. A diabetic pseudoparesis, with peripheral neuritis, may cause slight difficulty in diagnosis. In children, insufficient carbohydrates may be responsible for anxiety, naughtiness, and other disturbances of behaviour. Anomalous psychic states may be produced in the rare condition of hyperinsulinism, and be mistaken for hysteria or an anxiety state of the psychogenic sort. *Gout* may occur in people predisposed to affective disorder; often a depressive phase precedes an attack. Alkalosis and anoxæmia may each be the cause of mental disturbance of the organic type. In *pernicious anæmia* there may be symptoms, *e.g.* an acute confusional state, referable to the structural changes in the central nervous system, but more often depression occurs without “organic” features; mania can also occur, and in some cases a chronic paranoid condition. The more “organic” the picture, the poorer the prognosis for a return to mental health. Of deficiency diseases *pellagra* is the one most commonly productive of mental disorder. It must be remembered that a long-standing anorexia, of psychogenic origin, or occurring in the course of a chronic melancholia, may itself lead to a pellagroid condition, so that the symptoms of mental disorder will then be those of the original illness plus those due to the deficiency. The clinical picture is sometimes very like that of hysteria; or the usual organic

syndromes may be produced, especially florid confusion with perhaps hallucinations of fire.

In the metabolic disorders just mentioned the physical phenomena are relatively coarse and obvious. It is in some cases proven and in others highly probable that less obvious metabolic disturbances are either among the primary symptoms of "functional" mental illness, or are its pathological basis. The acid-base equilibrium and the electrolytes of the blood, the metabolism of carbohydrate, fat, and protein, and the chemical regulation of the vegetative activities are all, in such forms of mental illness as schizophrenia and mania, subject to changes which have not as yet been used in the pathology or treatment of these conditions, because the findings are not sufficiently constant or specific; it is also likely that our methods of investigation are not delicate enough.

2. ENDOCRINE DISORDERS

These play a more prominent rôle in the investigations than in the clinical practice of psychiatry. Many endocrine preparations have, it is true, been administered to schizophrenic, sexually perverted, and melancholic patients, either empirically or in accordance with a premature and ill-devised theory, but the good results of all this are negligible. Estrin treatment of menopausal neuro-vegetative symptoms is a rational procedure. The blind use of the endocrine glands in the theory and practice of psychiatry has had its day.

Exophthalmic goitre is more prone to occur in anxious, nervous people, especially after some sudden shock. The usual concomitants—restlessness, tension, irritability, difficulty of concentration, and liability to sudden changes of mood—may be complicated by a definite mania or depression and, if the disease be severe or advanced, delirium and confusion may supervene. Though such organic syndromes mean as a rule a bad prognosis, they sometimes clear up dramatically after operation. The interaction of constitutional and psychogenic factors with the actual thyrogenic intoxication^a makes some treatment of the anxiety by psychological as well as other methods^s desirable in many cases of exophthalmic goitre, either as a preliminary or supplement to partial thyroidectomy.

In adult *myxœdema* the slowing of mental activity may sometimes be accompanied by a chronic paranoid psychosis, or there may be a phase of excitement with hallucinations; the variety of syndromes that can occur is referable to pre-existing constitutional tendencies and to the varying severity and rapidity of development of the thyroid deficiency. An apparently "functional" syndrome may precede the overt myxœdema.

Juvenile and congenital myxœdema is described elsewhere (see p. 495).

Tetany may be signalled by epileptiform seizures, or there may be a proneness to psychogenic fits; thus the patient may spontaneously overbreathe until a sufficient disturbance of calcium, etc., results in an induced convulsion. Hysterics sometimes use hyperventilation in this way to induce a passing tetany. In severe tetany a resistive lethargy or an excited incoherent confusion may occur.

Pituitary diseases are more often accompanied by mental symptoms that are a comprehensible reaction to the physical symptoms than by organic syndromes; the latter when they occur may be due to increased intracranial

tension. In acromegaly, depression, reserve, touchiness, and irritability are not surprising, though some acromegalics remain cheerful as long as their disabilities are moderate, and sometimes there is a blindness to the disease, a lack of insight, even when it is advanced. In dystrophia adiposo-genitalis a rather childish placidity may be met. In adiposis dolorosa depression may be severe, or hysterical symptoms may develop. Simmonds's disease may be accompanied by depression, severe anorexia, reaction to the psychosexual disturbance, and, in the later phases, by organic syndromes due to the cachexia. Similarly, disorders of pituitary function have been found in some cases of "anorexia nervosa." In Cushing's basophil syndrome depression and other mental disturbances can occur: we have seen a severe paraphrenia develop during the course of the illness.

Addison's disease is accompanied by a neurasthenia of which for a time the physical basis may be quite overlooked (as may also occur in myasthenia gravis); in the later stages delirium has been known to occur.

Sexual epochs may in women be associated with mental disorder of the organic type, e.g. some psychoses of pregnancy and the puerperium. During pregnancy plain psychosis is rare, but hysterical symptoms, depression, and anxiety are fairly common, especially if the mother is reluctant to have another baby; a gross psychosis may, however, break out during the latter months of pregnancy. The organic mental syndromes may develop along with polyneuritis, eclampsia, or chorea gravidarum. Termination of the pregnancy is called for on account of the mental condition when there are symptoms of organic psychosis which are likely to get worse, a history of suicidal attempts or infanticide in connection with previous pregnancies and a depression again in this one, or if on other grounds there is a clear risk of suicide or other untoward result of the mental illness, should pregnancy continue. The decision is often a very difficult one, requiring an expert knowledge of psychiatry for the careful appraisal of aetiology and prognosis essential in every case. The question must turn mainly on the therapeutic value of terminating the pregnancy, so far as the mother's mental state is concerned, as well as upon the stage of pregnancy reached.

In the puerperium "functional" psychoses often develop in predisposed women; if there be septicæmia as well, a confusional state or a delirium, followed by a period of neurasthenia, may occur. In many cases the delirious puerperal psychosis clears up in a week or two; the more endogenous varieties have sometimes a less satisfactory outcome than their form and onset suggest. Infanticide may occur in a puerperal psychosis, especially if the mother has, while pregnant, felt resentful at having a baby or been troubled by murderous preoccupations, e.g. obsessions. Psychoses of lactation are rare, and seldom of the organic type. Menstruation is apt to be associated with depression, irritability, and languor in many women, especially during the few days before the period begins; there are no menstrual psychoses, but the liability to suicide and to psychopathic reactions is somewhat higher at this time. Puberty and the climacteric are periods of stress during which schizophrenic and affective disorders may occur. The effects of castration are dependent on the age at which the gonads are removed: intellectual development is unaffected, but the emotional and conative activities of those castrated in adult life may be impaired. Neurasthenic symptoms are frequent, and in women anxiety symptoms may appear.

3. VISCERAL DISEASE

This may be directly responsible for mental disorder of the organic type. Thus *cardiac* disorders predispose to an anxiety, which at night may take the form of mild delirium, with restlessness, terror, disorientation, and auditory and sometimes visual hallucinations. With improvement in the circulation, the mental symptoms disappear, or remain only as a moody unrest. Reference has already been made to arterial hypertension (see p. 1829). The connection between *alimentary* disorders and neurasthenic states is well attested, and is striking in children. Jaundice may be accompanied by severe depression, but seldom leads to delirium, save in the case of acute yellow atrophy. *Uremia* may disturb consciousness greatly, in the form of any of the organic syndromes, from a twilight-state to a euphoric dementia; a Korsakoff condition can occur, but is infrequent.

II. MENTAL DEFICIENCY

As already stated, there is nothing in principle to separate these from other forms of cerebral impairment save that they occur at an earlier stage of life. As with mental disorder, they shade into normality so that no man can say where stupidity ends and feeble-mindedness begins. Again, as with mental disorder, the same clinical picture may be due to a variety of causes ranging from heredity to trauma. They are, moreover, delimited rather by social than by other criteria, and they are not definitely associated with any constant pathological findings. In that they are capable of only limited improvement when well established, and that the intellectual functions are more obviously damaged than any others, their similarity to dementia is easily seen. They are not by any means cases of purely intellectual defect; they represent, it is true, one extreme on the scale which has people of great intellectual ability at its other end; but they are also examples of a general impairment of mind, affecting the emotional and conative functions, and often associated with a more general impairment of the whole organism, which may be seen in its physical structure. Since the milder forms are indistinguishable (except on an arbitrary reckoning) from what may be termed normal stupidity, it is difficult to use rigorously the official definition of mental defect, as a condition of arrested or incomplete development of mind existing before the age of 18 years, whether arising from inherent causes or induced by disease or injury; but the description is serviceable. It should be recognised that, just as "psychosis" differs from "neurosis" only in a rough social sense, turning on the need for special care; and "neurosis" from "normality" only in respect of the limitations the former imposes on one's daily life as a social organism, so does the distinction between normality and feeble-mindedness, and between gross or certifiable deficiency and the lesser forms, turn on the social adaptation of the person in question. To complete the points of similarity there is recognised a "moral defectiveness," which has its parallel in some kinds of "psychopathic personality." The effects of encephalitis lethargica, parenchymatous

syphilis, and thyroid deficiency upon the mental state and development at different ages, or the varying results of amaurotic familial idiocy in the infantile and the delayed juvenile form, illustrate how important is the stage of growth or maturity at which damage is done.

Ætiology.—The common division is into primary and secondary, though an alternative distinction has been proposed between those who represent the lower extreme of normal variation (the "subcultural" group) and those in whom a gross structural pathology is discoverable. The primary or hereditary group is a large one, making up approximately three-quarters of all cases of mental defect. This is an estimate arrived at by independent workers, but likely to be changed as we acquire better methods of determination and subtler views of the interplay between environment and heredity. The grosser the deficiency the less important the hereditary factor, except in some rare well-defined anomalies such as amaurotic idiocy. Familial concentration of a given form of defect is specific for each clinical type. Dominant inheritance is more evident in the families of simpletons than of idiots. An incompletely dominant single factor or a combination of several genetic factors may be responsible. The mode of transmission of amaurotic familial idiocy and of phenylketonuria is recessive; that of epiloia dominant.

The environmental causes are prenatal (*e.g.* mongoloid idiocy), congenital, or infantile (*e.g.* birth injury to the brain, meningo-encephalitis, hydrocephalus, cerebral syphilis). Various poisons and deficiencies may be responsible, as in the well-known instance of cretinism, as well as certain malformations of the cerebral tissue, *e.g.* microgyria and porencephaly, and of the cranium, *e.g.* oxycephaly. Sensory defects, as in a deaf-mute, may greatly impair mental development. It is possible that some cases of schizophrenia beginning in the first few years of life are indistinguishable from mental defect and are diagnosed as such; in more general terms, it may be said that the mechanisms commonly implicated in the adult illness schizophrenia may be those chiefly affected from the beginning in some cases diagnosed as mental defect.

Pathology.—In many cases there are no significant findings; this is particularly the case with high grade defect. It is probably impossible from the histological appearances to infer the extent of hereditary or exogenous causation. Developmental anomalies, such as general hypoplasia and macrogyria may, however, be mingled with evidences of a past lesion, as in porencephaly or hemiatrophy, or with signs of a disease actually present, as in amaurotic idiocy, cerebral lucas, and tuberosc sclerosis.

Symptoms.—The customary classification is into idiots (who are too defective to be able to guard themselves against common physical dangers like falling into the fire), and imbeciles and feeble-minded persons (who need to be looked after because of their incapacity to manage their affairs or to profit by instruction). Imbeciles cannot earn their living; the feeble-minded cannot get on in an ordinary school, but may learn a good deal in a special school and be able to earn a living. The criterion is in each case mainly a social one; the same is true of "moral defect," *i.e.* mental defect coupled with strong vicious or criminal propensities. Although these terms are defined in an Act of Parliament, they are vague and of administrative rather than medical use. An attempt has been made to render

them more precise by psychometric means: the customary tests for mental age are applied, and if the subject's intelligence-quotient ($\frac{\text{mental age}}{\text{actual age}} \times \frac{100}{1}$) be less than 20, he is called an idiot; if it be between 20 and 50, an imbecile; if between 50 and 70 feeble-minded. It must, however, be recognised that though mental defect is mainly a matter of intellectual capacity, it is not solely this, and that intelligence tests, however valuable and trustworthy, cannot give a complete indication of the degree of mental defect. Even the intellectual defect may be uneven, showing much more in some tasks than in others, and it would be an error to suppose that a mentally defective person with a mental age of, say, $9\frac{1}{2}$ years is mentally in the same state as a normal child aged $9\frac{1}{2}$ years.

The *physical* symptoms are chiefly due to lesions of the central nervous system: birth trauma may have led to paralysis, spasticity, athetosis; or there may be evidence of an inflammatory condition of the brain and its membranes, as from syphilis. The whole clinical picture may be greatly coloured by the motor disturbance, *e.g.* continual rocking and twisting movements, grimaces, and abnormal posture. The special senses may be affected, as the result of an independent anomaly, *e.g.* coloboma, misshapen ears; or from a common cause, *e.g.* interstitial keratitis, the retinal changes of amaurotic idiocy. It is dubious whether the "stigmata of degeneration," such as a "Gothic" palate or a Darwinian tubercle, occur any more frequently among defectives than in the rest of the population: at all events, there are none that can be used diagnostically, except in the case of mongoloid idiocy. There are, however, some correlations between somatic anomalies and mental defect. Thus, there are more physical defects among these people than in the average population, and this becomes more evident as one looks lower in the scale of mental defect, in which skeletal and cardiovascular anomalies may fairly often be found, sometimes, but not always, due to thyroid or pituitary disorders. The mongoloid variety is described below.

The *mental* symptoms are lack of intelligence and of the normal exercise and control of primitive tendencies. This may be extreme, as in idiots, who cannot be taught to feed themselves and keep clean or who can only just recognise their companions and make their elementary needs known—they are, indeed, much less intelligent than an animal. Imbeciles are usually incapable of learning and remembering any but very simple matters. They may, however, be able to do automatically what they cannot understand or put to independent purpose: thus, "idiots savants" are especially clever at doing mental arithmetic, recalling dates and other such operations. What imbeciles manage to learn they cannot utilise in any but the most familiar circumstances. Abstract concepts are too hard for them, and their judgment is as poor as their grasp or awareness of what is relevant in any situation. Though in many ways suggestible and accessible to flattery, they may be obstinate and egotistical, and readily fall into antisocial courses, *e.g.* prostitution, vagrancy, crime. Crude sexual offences or murder may be committed as lightly as some minor deception. The personality of imbeciles varies widely: some are docile and kindly, others rough or deceitful and vindictive. It depends much on their upbringing. It has been found that in satisfactory conditions only about 8 per cent. of defectives show antisocial or

troublesome behaviour. But though the deviations of personality may not lead to delinquency, it is common to find in mentally deficient persons defects of temperament and character, as well as of intelligence, which are reflected in social inefficiency. This is most important in the feeble-minded, who have intelligence enough to learn an occupation; whether they can earn their living by it will depend on their character and the way they have been brought up.

Many persons who are high-grade defectives, when measured by formal tests, are not taken to be such because of their social adaptability, their fluency and capacity for keeping their head above water as long as economic and other stresses are light. There are instances of people classed as mentally defective during childhood, because of their backwardness in school and their low score in tests, who later in life amass money by their own efforts, or even hold a responsible position. A majority of high-grade defectives, however, live dependent and often troublesome lives; at most they do simple repetitive work. Many of them are unstable creatures, whose psychopathic personality may be sufficiently antisocial for the term "moral deficiency" to apply to them. Hysterical trends may show themselves in crude phenomena, *e.g.* convulsions, counterfeit insanity or fantastic lying; and religious and artistic pretensions may take in gullible followers and even lead to the founding of ephemeral movements.

Defectives are prone to disturbances of mood, sometimes arising out of awareness of their inferiority and its social consequences. Sudden outbursts of excitement may show similarity to manic or catatonic hyperkinetic states; they may be accompanied by a paranoid hallucinosis, mainly auditory, which clears up with startling rapidity in a day or two. In respect of these psychotic episodes, defectives are like epileptics and juvenile encephalitics, in whom a cerebral impairment has likewise occurred before the attainment of maturity. Some of the morbid phenomena, especially in idiots, are very similar to the disorders of motility seen in schizophrenics, because, it may be assumed, the same bodily mechanisms are implicated.

The *mongoloid* type of idiocy is characterised by striking physical features. Probably the outcome of intra-uterine conditions, it is most frequent in last-born children in a large family, or in children born of elderly mothers; parental syphilis may occasionally be the cause. In many cases the brain-stem and cerebellum have been disproportionately small, and other signs of maldevelopment have been reported. It is likely that hereditary factors of a recessive nature also play a part. The condition is usually present from birth; physical growth is slow, and has stopped by the time the child is fifteen. Defective growth of the skull, leading especially to abnormalities of the base and the orbit, are responsible for the peculiarities of cranial shape. The pituitary gland has been reported as showing an increase in eosinophil cells and deficiency of basophil cells. The appearance of these usually happy idiots and imbeciles is rather suggestive of a Mongol or of a fetus. The skull is small and round, and the junction of occiput and back of neck flat; an epicanthic fold across each inner canthus, narrow tilted eye-slits and lids without lashes, red cheeks, fissured and often protruding tongue, stubby depressed nose with nostrils looking forward, irregular late-appearing teeth, coarse hair on the scalp, small facial bones and occasional neurological anomalies, such as nystagmus, make the head of every mongoloid a disagreeable but ready

index to his disorder. That the disorder is a general one the rest of his body testifies; his limbs are lax and over-mobile at the joints; he has broad, clumsy feet and hands, with short fingers and a crease running straight across the palm, protuberant belly and low stature; and perhaps a congenital cardiac lesion. The similarity in a few respects to juvenile myxœdema, and the occasional concurrence of the two conditions sometimes make differential diagnosis difficult; not all of the signs here mentioned need be present in any one case. On the mental side, there is a liveliness and amiability not often seen with so much intellectual defect: the patients like music and little jokes of a primitive sort; they will imitate gestures, but seldom learn to speak properly with their rough harsh voices.

The forms of deficiency due to *thyroid insufficiency* and *cerebro-macular degeneration* are referred to elsewhere (pp. 1844 and 1624). *Epiloia* is the name given to the rare condition in which tuberoses sclerosis of the brain, adenoma sebaceum and tumours of the kidney and heart may be associated; epilepsy is common, and there are gross mental disturbances. *Gargoylism* is a rare chondrodystrophy, with hepato-splenomegaly and mental deficiency. *Phenylketonuria* may be recognised by the characteristic metabolic disturbance. It is a hereditary disorder, due to a single autosomal recessive gene.

Diagnosis.—Recognition of gross mental deficiency calls for no skill. The degree and kind of impairment, however, and the somatic variety or cause have to be worked out in every case. The latter problem—a minor one, except in the case of juvenile myxœdema and syphilis—is to be settled by careful physical examination and inquiry into the history. The former is a matter of assessing intelligence and social aptitude.

The assessment of intelligence is nowadays a matter of giving the patient tests which have been standardised on average samples of the population. What is average or normal at a given age is therefore known, and the defective child's performance can be compared with this. The most popular and serviceable tests are modifications of those put forward by Binet and Simon in 1908. As these may give a rating that depends unduly on the child's educational opportunities and facility in language, and may not indicate special abilities, *e.g.* in mechanical matters, many other tests have been worked out which supplement or, in certain cases, replace the Binet scale. A child under the age of 5 cannot be satisfactorily dealt with by the Binet tests, which moreover have only limited value for measuring the intelligence of adults. It is difficult to agree about what in a normal child must be regarded as the limiting age at which he becomes of adult intelligence; it is generally taken as 14 or 16 years. In all tests the emotional state of the subject is a factor that influences his performance. The emotional reactions to being tested must be taken, along with responses to more familiar situations, *e.g.* at home or at school, as evidence of the soundness or instability of the child's personality; by such criteria must be judged the social development of the patient, his fitness for living in the community or being put under lasting surveillance and control.

Treatment.—**PROPHYLACTIC.**—Eugenic measures are desirable for the rare hereditary conditions, like amaurotic idiocy, and in the case of those imbeciles and feeble-minded in whom genetic rather than environmental causes have been responsible for their maldevelopment and who are capable

and desirous of procreating—idiots do not procreate. Voluntary sterilisation for eugenic reasons has been recommended by a Departmental Committee, but has not yet been explicitly sanctioned by law. Birth control, therefore, is the eugenic measure to be advised in cases in which defectives seem likely to transmit their defect; unfortunately few such defectives can be relied on to observe contraceptive precautions effectively. Segregation may indirectly serve the same end. Well-managed parturition and treatment of parental syphilis are the only other practicable ways of forestalling defect.

Educational and social.—Much improvement may be attained by the training of defectives: it is work for experts. Where there are special disabilities, *e.g.* of the senses, or of such capacities as reading and writing, attention to these may lift the child out of the class of mental defectives altogether. Whether the child lives at home or in a colony or institution will depend not only on the degree of his intellectual and social deficiency, but also on the adequacy of his home circumstances. There are many kinds of provision for the care of the 300,000 defectives in England and Wales, ranging from special schools and statutory supervision to mental hospitals; 40,000 defectives are in institutions and nearly 35,000 are under statutory supervision. Well-run colonies serve to socialize many defectives hitherto vicious or violent, who can then go out and live more or less usefully in the community. Some, however, prove intractable, especially those who have epileptic fits.

Physical.—The bodily disturbances, *e.g.* contractures and paresis, call for orthopædic treatment, which sometimes indirectly benefits the mental state. The treatment of the forms due to thyroid deficiency or syphilis is described elsewhere.

AFFECTIVE DISORDER

This is of three types:

1. Manic excitement and hypomania.
2. Melancholia and mild or neurasthenic depression.
3. Agitated depression and anxiety state.

There is in each case a major and a minor form. Each is related to a more or less characteristic personality, and for each the cause of occurrence may be chiefly environmental or chiefly hereditary. Combinations are frequent (mixed forms), or there may be successive appearance of the different types, often with an interval between the attacks. A benign outcome or periodic course is the rule for the major forms, but not for the minor, which often tend to become chronic. This is partly because the environment can have more influence, whether for good or bad, on the course of the minor than of the major, more explosive and sweeping, forms. It would be a very convenient thing if endogenous cases could be sharply differentiated from psychogenic ones, as in the Kraepelinian scheme, but it cannot be done.

Ætiology.—**INTRINSIC.**—Hereditv is the most constant single cause. Research has been mainly into the major manic-depressive cases. The genetic factor is weakly dominant. It may be that more than one gene is concerned, but this is hard to tell, because the predisposition to an affective disorder may be latent in persons who have not been subjected to the stresses

that would make it manifest, and consequently the usual Mendelian figures are not obtained. The present state of knowledge is illustrated by studies on manic-depressive twins, among whom 69 per cent. of those monozygotic (*i.e.* with identical heredity) were alike affected with the disorder, while the corresponding figure was only 16 per cent. for the dizygotic pairs (*i.e.* with dissimilar heredity). In the 31 per cent. of monozygotic twin pairs who were not alike in respect of mental illness, the difference must have lain in the environment, thus showing the relative importance of external factors in causing the inherited tendency to become manifest. Although not manifest as illness, the inherited tendency may express itself in bodily and mental constitution.

The bodily habit that is found in a majority (not the overwhelming majority) of those with affective psychoses is called "pyknic." It is best seen in men after the age of 30. It is characterised by large visceral cavities (head, thorax, belly), a tendency to fat on the trunk, slender shoulder girdle and extremities, stocky build, a broad face on a short massive neck, thick receding hair and, later, baldness, venules on the cheeks, and a disposition to arthritis, gout, diabetes and especially arterio-sclerosis. As this John Bull build is so common in mentally healthy people, it cannot be regarded as a precursor of mental illness, but only as an indication that some of the constitutional and genetic causes, or biological requirements, for affective psychoses are present.

The same is true of the mental constitution or personality. Here there are several groups, shading off on the one side, by way of cyclothymia and other intermediate forms of mild disorder, into definite affective psychosis, and on the other into normal and stable personality. There are those with a pervading gloominess, pessimism and feeling of insufficiency that spoils their lives; others who are for ever anxious, keyed-up, wondering whether something has gone wrong or will go wrong, and whether it is their fault—careworn worrying creatures; while a third group is made up of the lively, enterprising, confident, sociable people, whose euphoria is patent. Irritability may be found in any of these groups, especially the second and the last. Contrasted or different features are often found mixed in the same patient. The most striking characteristic of the personality of manic-depressive patients is their ready responsiveness and lability of mood; they fluctuate with their surroundings, and in many instances pass suddenly and with small occasion from one mood into another far removed from it.

The signs of affective illness may appear in childhood, though major outbreaks of mania, depression or agitation are rare before puberty. When these occur, the phases are usually brief and the environmental influences strong. Milder forms are often regarded as normal, since night-terrors and other fears, mischievous gaiety and sulky gloom are all familiar enough in children; it is the degree, occasion and persistence of the affect which must decide whether it is morbid.

The psychological crises of puberty are only occasionally affective—chiefly self-reproachful depression or agitation—but during adolescence the illness becomes more frequent; it seldom, however, calls for mental hospital care. Each menstrual period may be accompanied by depression or restlessness, usually coming on about two days before the period. In the

third decade of life the number of cases steadily rises, and there is another peak in frequency between the ages of 45 and 55. The latter, "involutional," cases show the influence of age strikingly, so much so that they are often considered as separate disorders.

There is little to choose between the curves of age incidence for morbid depression and morbid anxiety of whatever degree; for mania the frequency is highest before the age of 30, as also for affective illnesses with a strong confusional flavour. Pregnancy is frequently accompanied by depression and agitation; psychological factors are mainly responsible. After childbirth, though there be no septicæmia, affective illness can occur, running a typical and often lengthy course.

The female climacteric is a time when anxiety usually mounts, and is accepted as an ineluctable effect of "the change." It may become definite illness, persisting even for two or three years. It is doubtful whether there is a specific connection between the endocrine causes of the menopause and so-called climacteric insanity; the melancholia then coming on is like the melancholia of five or ten years later, or the melancholia of middle aged and elderly men in whom the endocrine changes are not the same. The influence of sex as a whole is obscure. Women have this illness more than men, though the manic form is relatively more frequent in men. The reactivity is often greater and the syndrome less clear-cut in women.

There are geographical differences, sometimes thought to be racial, in the incidence, but the little that is known points to environmental rather than intrinsic causes for this. It has been suggested that affective psychoses are commonly linked up with high intellectual gifts; another says they have affinity with mental defect. The former statement has much better support than the latter, but both probably are fallacies depending on the material selected for study.

EXTRINSIC.—Physical.—Chronic toxæmia and acute infections, especially influenza and pneumonia, can be responsible for the illness. Various drugs help to heighten the anxiety to a morbid degree, *e.g.* alcohol in certain circumstances, insulin, or hyoscine. Cerebral trauma may provoke an attack. The list of physical factors could be much added to, but it must be borne in mind that wherever a distinctive, rather than incidental, physical cause can be found, the condition passes over into the category of organic psychoses. The most difficult cases in practice are those in which there is a question of cerebral arterio-sclerosis or exophthalmic goitre; the affective disorders indisputably due to these two diseases may be quite indistinguishable from others for which there is no such organic basis. The problem here is clinical rather than fundamental; since vascular, cerebral, endocrine and autonomic functions are particularly concerned in the mechanism of emotional change, certain disturbances of the physical apparatus will necessarily be accompanied by many of the psychological phenomena of these emotional changes. The depression of paralysis agitans and the anxiety of coronary disease are of the same order. The notion that coitus interruptus and other sexual practices produce anxiety is unfounded, but they may contribute to it by psychological means.

Psychical.—A recent misfortune may be the cause: of such, there is a great variety, ranging from commonplace to tragic. Any calamity to which human beings are liable may provoke an affective breakdown. Sometimes

it is induced by the insanity of a close relative. However trivial it seems to outsiders, the event that has precipitated an affective attack has been felt as a catastrophe by the patient; there are no records of great and sudden happiness causing an affective psychosis. The nearest approach to a specific connection between the precipitating happening and the type of affective illness is seen in the anxiety disorders which follow a terrifying experience such as exposure to shell fire and bombardment from the air; morbid depression following bereavement, financial setbacks or degradation is an understandable response, it is true, but to ascribe the type of response directly to the nature of the experience is specious, since on another occasion it may be with hilarious mania that the calamity is met.

Moreover, the experiences of a lifetime will have determined what calamities are most felt; they need not be calamities in other people's eyes at all. Experiences, spread over years, are the common extrinsic cause of the more chronic neurotic forms of affective illness: this applies least to chronic hypomania. In these chronic conditions the patient's own behaviour has so much to do with what happens to him, as it were, from outside that to separate extrinsic from intrinsic is very hard.

Pathology.—The *physiological* changes are characteristic only of emotional disturbance, not of morbid emotional disturbance; and therefore they are not of diagnostic value. They consist in lability of blood-pressure and pulse-rate, abnormal motility of plain muscle, especially in the alimentary tract, carbohydrate disturbances, variations in either direction of the rates of salivary and other secretions, and decreased psychogalvanic activity. The changes are variable from patient to patient and are not always discoverable. More significant are changes in basal metabolism, weight, sleep and menstruation; loss of weight is the rule during the illness. Irregularity of menses and then amenorrhœa often occur. Hypercholesterinæmia, rise in the blood iodine content, changes in the K/Ca ratio, and diminished cellular respiration have been alleged, not as yet conclusively.

The *psychological* changes, in spite of great external differences, have the following in common: the morbid phenomena are in accordance with the prevailing mood, though not wholly derivable from it; thought is less purposively directed to impersonal ends than it would normally be, but more purposively to personal ones; there is a small number of topics of pre-occupation in each patient, but his ways of arranging and embellishing them can be many; the whole body (or parts of it) often receives much of the patient's attention, because of more or, it may be, less feeling in it (hypochondria, depersonalisation); misconstructions abound, with consequent ideas of self-reference and persecution as well as misidentification; and there is a feeling of inner tension, unrest and excitement, however apathetic or care-free the patient's demeanour.

The seemingly greater quickness and capacity of manic patients has not been confirmed by psychomotor, intellectual and association tests; hypomanic patients sometimes, however, do better than in their normal state. This can be compared to the effects of increasing doses of alcohol. Patients with affective disorder are more irritable and excitable than is normal. Time appreciation may be grossly disturbed: personal time seems to pass very differently from clock time; time may seem to stand still; no future is conceivable. Perplexity may be conspicuous, and explanations of this

in terms of Gestalt psychology, conditioned reflexes, and toxæmia have been proffered.

The effects of experience in bringing about this illness cannot be explained in terms of a logical and coherent system, unless one accepts the premises of that system and infers what cannot be observed. Consequently, as there are several such psychological systems, there are several explanations. They state the conjectured ways in which instinctual energy or libido may become misdirected because of environmental conditioning, frustration and loss. It is possible to eschew such conjectures and at the same time to see the conditioning, the threats, frustration and loss that have understandably brought the patient to an excess of sadness, excitement or fear.

Symptoms.—**SYMPTOMS OF MANIA.**—There is excitability of mood and movement. The *mood* is mostly one of jollity, rather infectious, but likely to become boring or overbearing; occasionally it turns to anger and resentment. It is labile; tears will flow readily on some trivial occasion, to pass into laughter in a twinkling.

Thinking is apparently rapid. There is flight of ideas, with successive words and phrases loosely connected only by similarities of sound or chance associations. Consequently, the patient wanders from the point; whether he can come back to it depends on the severity of his condition. Jokes, self-praise, flighty comment on his surroundings, and facile optimism make up the tenor of his exuberant conversation. Nevertheless, the number of topics he touches on in the course of the day is often more limited than if he were in normal health: he reverts to a few matters over and over. He may criticise himself, with cynical bitterness or humour, as he criticises others; he may talk a lot about bodily disturbances, e.g. his varicose veins or his sore throat. His mood and expression are consonant with what he says. He is distractable, herein seeming at the mercy of his sensations and of every small detail, whether it be inside himself or, as is more common, connected with things about him. Judgment is impaired.

Delusions are less common than *distortions* and misstatements. People are wilfully called out of their names, events misrepresented, bodily sensations exaggerated, and accusations of ill-treatment or persecution irresponsibly preferred and sometimes long persisted in. The more confused and excited the patient, the more likely to be deluded and even hallucinated. Most of the seeming hallucinations are *façons de parler* or illusions; sometimes the patient is, as it were, pretending or acting the part of a hallucinated person.

Activity is exaggerated, and in severe cases incessant. Its object may change from moment to moment, but sometimes the main end is kept pertinaciously in view. The patient, if tactlessly thwarted, gets angry, sulky or violent. He feels very strong, and seems untiring. He has many schemes, of an optimistic cast, and, in the course of putting them into action, may be extravagant, inconsiderate or interfering. Sexual excesses or drunkenness may occur and bring much harm, especially when the patient is a young woman. Troubles with the police arise through silly pranks or self-confident exploits.

Sleep is brief but deep. In the early and mild stages the patient looks exceptionally well, but after weeks or months of over-activity and little sleep he looks exhausted, with sordes on his lips, hoarse voice, drawn skin and perhaps less total activity but many unfinished little movements. Food is

welcomed in the mild stages ; when the activity is great, the patient does not give himself time to eat, but plays with his food or is continually diverted to something else. Sexual desire is at first heightened but potency less.

The symptoms vary widely in degree. Mild hypomania may be an enviable time of well-directed expansive energy, unencumbered by some habitual restraints ; gross mania may be a delirious, hallucinatory condition, with incoherent talk and little free activity.

SYMPTOMS OF DEPRESSION.—In the early stages or milder forms, the patient finds concentration and recollection difficult, he has less interest and pleasure in life, he feels that this world is unreal and himself changed, he dreads effort or responsibility.

The *mood* is one of grief and misery, looking in every direction for material to feed on. The past supplies peccadilloes or graver lapses ; what is wretched in the present is dwelt on inordinately ; the future is foreseen as hopeless ruin. Anxiety is mixed with it, often in extreme degree. Weeping is less common in the extreme forms. The patient's expression usually conforms to his affect.

Thinking is more difficult. This "retardation" in thinking shows itself as incapacity to deal quickly and purposively with impersonal topics, while brooding on personal matters goes on, with a press of inner activity, a ceaseless roundabout of painful thought. The making of decisions is dodged. Conversation may become meagre, even monosyllabic, though some patients are ever ready to tell their troubles. The content of their thought is sombre—the product of ruthlessly unfair examination of their frailties and misfortunes. Some criticise themselves remorsefully or with cynical detachment ; some bewail their losses ; others abandon themselves to resigned and world-shunning despair. There are many varieties of misery, and melancholia knows them all—as many varieties as can be made from the experiences, character and imagination of a human being. Consequently they reflect the moral, economic or hygienic standards of what is good and bad that are imposed on us by modern society and our particular education.

Delusions occur in proportion to the depth of affect ; they are the extreme form of the doubts or preoccupations just mentioned. Patients often fluctuate between uncertainty and conviction about their troubles even during the same day or the same conversation. Insight may be good and judgment sound, when the affect is not overwhelming. The delusions are the product of the depression, which is primary ; they are not its occasion, though often adduced as that. Most of them concern the future as well as the past ; anxiety is prominent. Wickedness to be visited with damnation ; secular crime to be punished in this world ; loss of property that will mean starvation and beggary for one's family ; mortal or corrupting diseases—these are the common substance of delusions and are often commingled. For example, some patients blame themselves for having caught venereal disease which will expose them to the loss of their job and of their hope of salvation, exclude them from decent society, and do loathsome damage to their bodies ; no evidence, no argument shakes the erroneous belief. The delusions may be grandiose in that the patient affirms himself the chief of sinners, no one has ever been as wretched or wicked as he, he alone has done the unpardonable sin ; or they may be of a minimising sort—nobody cares about him, he is of no account, let him go into a corner to hide, people despise him. This last

belief is often understandably associated with ideas of reference or persecution—people make contemptuous gestures or remarks as he passes, they set detectives to watch him, they tell each other how bad he is. He accepts this almost always as his desert, though occasionally there may be overt resentment. Apart from this resentment, his beliefs derive understandably from his affective state. There are, however, features that betoken underecurrents at variance with the professed attitude or delusions. Thus many depressed patients, professing humility, are importunate in their demands on those around them.

Such hallucinations as occur are in keeping with the patient's affect and are of much the same nature as the delusions, though expressed more in perceptual terms. People are making derisive remarks, his body gives off foul smells, food has a different and disagreeable taste—it is often the mode of expression rather than of subjective experience that decides whether these are hallucinations or delusions. This is notably the case with bodily pre-occupations, when, for example, patients report their food to be stagnating in their belly, their skin dull or fetid, their eyes impaired, their head empty. Much of this depends on depersonalisation, in which the body as a whole feels bereft of life and feeling, and emotional deprivation or emptiness is translated into bodily experience. In mild forms of depression there is no question of delusion or hallucination, and often no recognisable content to the gloom; the patient cannot say why he is sad. In the more chronic forms a settled and partly justified conviction about ill-health, present troubles, and the dark future prevails; the ideas may be obsessional and partly divorced from the prevailing affect.

Activity is limited, thus contributing to the "retardation." The more severe the depression the less does the patient do, unless the concomitant anxiety makes him restless. It is possible, however, for a patient to be depressed without "retardation." In typical cases facial expression is rather fixed and movements delayed, as though done against resistance; more or less complex activities, dressing, say, or writing a letter, take unduly long. The most extreme form is stupor or lack of all spontaneous activity; it is seldom absolute. Patients rarely become wholly indifferent to cleanliness in defecation and micturition.

Suicide is the greatest danger in depression. Whereas manic patients thoughtlessly do themselves harm or get into a fight but do not try to get hurt, depressive patients are often bent upon doing away with themselves. The risk is not proportionate to the degree of depression; many very retarded and melancholy patients make no attempt, while in depersonalised mild cases a fatal outcome is not uncommonly brought about thus. There is consequently much risk during the phase of improvement—often more risk than during the preceding severe "retardation." Deliberate self-mutilation is rare.

Sleep is bad—hard to come by, light and unrefreshing. The *appetite* is bad too: food may be constantly refused for this reason. Commonly also the patient eats too little because of feelings of fullness and other discomfort in the abdomen, or because of delusions about his bowels or his food. Mild constipation is common, but is often given much exaggerated importance by the patient. The *weight* diminishes, chiefly, but not by any means wholly, because of insufficient intake of food. Daily fluctuation in the general condition, with improvement towards evening is common. The skin may be dry

and sallow, and in some severe cases pigmented, as it is in pellagra. Menstruation may lessen or cease ; sexual desire is much less.

Here, too, there are wide variations, between the mild "neurasthenic" and the grossly deluded melancholic who craves death. There is every gradation between the two extremes, and a single case may during its course exhibit them all.

SYMPTOMS OF ANXIETY.—The *mood* ranges from uneasiness to panic-stricken terror. It may be an abiding or a recurrent state. Though chiefly turned to the future, as fear must always be, it rests on past experience, often painful and largely repressed, and it reverts to the past to account for the troubles in store. Herein, as with rationalisation and some other psychological devices, there is evident a strong desire to make things understandable in a causal nexus—a tendency to be found not only in patients but also in those who observe them. The patient's expression varies with the strength of his fear.

Thinking is troubled, the disorder showing itself in speech somewhere between frightened dumbness and the voluble talk that seems designed to cover up embarrassment and disquiet. The patient can seldom follow a train of thought for long without a limited number of preoccupations forcing themselves in. How far this interferes with daily life or set tasks depends on the amount of anxiety, as does also the impairment of judgment and insight. The content of thought is as manifold as in depression, every normal matter of human concern enters into it. Fears centring strictly on a few special topics, *e.g.* the fear of being run over in the street, may be to the fore ; the fear of insanity is particularly common.

Delusions are frequent in the grosser forms, which are most strikingly though not exclusively seen in patients of late middle life. They may say that their bowels are stopped up and their bodies about to rot ; their enemies are waiting to tear them to pieces ; their families will be tortured ; their names abhorred for ever. Hell, they are certain, awaits their souls though their bodies cannot die ; time stands still and no redemption is possible. There are many delusions less extreme than these mainly hypochondriacal and nihilistic ones ; *e.g.* beliefs that employment will be unobtainable, or that the patient will be victimised for having had such an illness. Hallucinations can occur : at the height of fear every sound and sight and smell may be misinterpreted as meaning some pain to come ; but most of this is illusional colouring of actual percepts. Depersonalisation is common with all degrees of anxiety.

Activity is much disturbed. There may be sudden attacks of panic in which the patient rushes blindly out into the open, or aimless wandering, ceaseless agitation, with movements especially at the small joints—wringing of the hands, rubbing the face, picking at sores, pulling out hair. Starting many tasks and finishing none is as characteristic of anxiety as of mania. Anxious people are distractible : their eyes follow a trivial movement—a fly walking on the window-pane—though they only comment on it when some interpretation that chimes with their mood can be fitted ; their ears are sharp for hints of alarm. During an attack of anxiety with strong somatic repercussions (*e.g.* vaso-vagal attack) activity may be completely interrupted—so-called collapse—while the patient, terror-stricken, expects his death ; alternatively he may run for air or help. Very agitated patients may

lie or sit in semi-stupor, with starting eyes and parted lips, incapable of speech unless under some strong stimulus.

Suicide is uncommon in those with episodic, highly somatic attacks of fear, and in those with chronic mild hypochondriacal anxiety, but not infrequent in the grosser forms and in those mingled with depression.

Sleep is bad: in the mild forms the patient may be afraid to fall asleep because of his horrifying dreams and the terror into which he suddenly awakes.

Sudden highly somatic episodes of anxiety, vaso-vagal attacks, are common: the patient feels his heart palpitating, his bowels turning over within him, he sweats, his limbs tremble, his mouth is dry, he feels he will fall or collapse or die; he turns pale, his pulse-rate changes, usually becoming more rapid, his blood-pressure rises, he may want to open his bowels or pass his urine. When anxiety is long-standing and severe, such attacks are rare. It is possible for parts of this general affective disturbance to be isolated, and to occur with little conscious anxiety, as in muco-membranous colitis, effort-syndrome, aerophagy, neurotic indigestion, enuresis, impotence, ejaculatio præcox, psychogenic asthma, hyperidrosis. The factors determining such special emphasis on one or other system are partly physical (some organic defect or innate functional anomaly) and partly psychological; in depression a comparable problem would be weeping or constipation. In anxiety thyroid enlargement can occur; weight falls off; menstruation is irregular or ceases; the deep reflexes are very active.

Diagnosis.—Typical cases are easy to recognise. The common errors of diagnosis lie in: (1) Missing organic disease (*e.g.* general paralysis, cerebral arterio-sclerosis); or the converse (*e.g.* mistaking the more expansive manic patient for a general paralytic). (2) Forgetting how mixed the symptoms of mania, melancholia, and anxiety may be, so giving rise to atypical pictures that may be mistaken for schizophrenia, if too superficial an examination or too static and rigid a diagnostic criterion be used. (3) Forgetting the influences of age, general personality, and milieu on the content of a patient's mind, *e.g.* his having lived among spiritualists may lead to deceptively fantastic statements. (4) Expecting to be able to diagnose solely on presenting symptoms, without regard to previous history and constitution; the reverse is also to be avoided. (5) Expecting diagnosis always to lie between distinct entities which could not possibly be mixed together in the same person, as though hysteria were incompatible with affective psychosis, or both of these with schizophrenia; in fact, they often are mingled. This is not to make light of diagnosis, which gives the psychiatrist much knowledge that he cannot gain from study of the individual case before him.

Nothing in the mental state of a patient with affective disorder enables one to exclude an organic basis such as general paralysis or cerebral arterio-sclerosis. This decision must turn on the physical findings. The problem becomes simpler when signs of dementia supervene. (See p. 1825.)

From schizophrenia, diagnosis depends on a picture of the whole illness, on the presence of characteristic thought-disorder, incongruity of affect and bizarreness of behaviour, as well as on the previous personality and constitution, rather than on any positive features of affective psychosis; the remoteness and unconvincing manner of the schizophrenic, so hard to describe but

almost conclusive when recognised, may help. Later, when complaints have become empty and repetitive to the point of stereotypy, and catatonic symptoms mix with the anxiety, diagnosis is easier. As between schizophrenic and manic excitement, the setting in which the excitement occurs is almost more important than the *prima facie* symptoms. In young people schizophrenic features may often be found without their being of much significance; in the elderly what seem to be catatonic features may rest on an organic cerebral basis. The more easily one can get in touch with the patient, enter into his mood and understand what he says and does, the more is it an affective, not a schizophrenic disorder. The range of benign affective phenomena is wider than a textbook description can convey.

There is no need to try to diagnose affective psychosis from psychogenic depression, cyclothymia, anxiety neurosis, neurasthenia, or involutional melancholia; these are only subdivisions of it, in which the age, reactivity, severity, or chronicity of the condition is being stressed. Periodicity is sometimes made the hallmark of affective psychosis; this historically interesting point of view is hard to apply, because so many patients have only one definite attack in their lifetime, and because periodicity can be striking in other conditions, such as obsessional disorder and schizophrenia.

From obsessional disorder the diagnosis may be difficult when there is localised anxiety or depression with sharp content and good insight; so closely alike are the conditions, that eminent authorities would include obsessional disorder also in the manic-depressive group, thus disposing of the diagnostic problem. It is best, however, to keep them distinct, and to discover in a particular case whether the characteristic subjective rejection of the obsession occurred at its first appearance; often the anxious or depressive patient at the beginning has welcomed the thought which accords with his affect, though later he struggles against it and may disclaim it. Genuine obsessions, however, are common in affective psychoses.

Course and Prognosis.—The varieties of outcome and sequence are many. They depend on the balance between particular intrinsic and extrinsic causal factors in each case, and on the extrinsic factors which are brought to bear on it in the form of treatment. The more typical the illness, the surer the recovery in favourable circumstances.

A history of definite affective psychosis in a parent or grandparent points to recovery from the attack, but it is unsafe to infer the course of the illness from hereditary data alone. A well-adapted personality and a pyknic build, a history of similar illness followed by complete recovery, a fairly sharp and fairly recent onset, and precipitation by external troubles which will not be likely to continue are all of them points to the good. Advancing years make the prognosis poorer, but a first attack of involutional melancholia, if there be no vascular disease, eventually clears up in two-thirds of the cases. Bodily symptoms are often the best indication of coming recovery. Improved appetite and regularity of the bowels, cessation of anxiety attacks, clearing of the complexion, increase of weight and return of menstruation may be noted, even before any increase of activity and long before any admission of feeling better can be got from the patient.

A first attack of excitement or anxiety will seldom be the only one; of depression it may. Periodic depression and anxiety is less likely to cease in middle life than periodic excitement. The occurrence of hallucinations or

delusions is in itself of little consequence prognostically. A transition from anxiety to depression or mania, and from mania to depression, or *vice versa*, is commonly gradual. Only in predominantly reactive attacks can one surmise how long the illness will last, or when another attack is to be feared. After recovery complete insight into what happened during the illness may not be attained, especially by resentful manic patients, melancholics who are sensitive and suspicious, and agitated patients who feared personal harm.

Generalised somatic disturbances, *e.g.* loss of weight, especially if acute and brief, are of good prognostic import, other things being equal. The more the somatic preoccupations or symptoms are diffused over a period of time and localised to one system, the poorer the prognosis; this, however, does not apply so much to children as to adults. Hypochondria and depersonalisation suggest a long illness, as do nihilistic delusions (*e.g.* denying that one's bowels are opened at all), and, to a far less extent, admixture of hysterical or schizophrenic features. The more the psychogenic causes have been obviously operative for a long period, the greater the tendency to chronicity. In the more chronic forms or after a series of attacks, there may be impaired initiative and judgment, irresoluteness, dullness, and social deterioration—none of them conspicuous. Puerperal and pregnancy psychoses have a good outlook. The milder forms of anxiety and depression, if not already chronic, respond well to treatment, especially to psychotherapy.

Death may occur from suicide, insufficient food, and intercurrent disease, especially pneumonia. Sometimes it is inexplicable on such grounds; a wasting disease, reminiscent of pellagra, carries them off.

Treatment.—**PROPHYLACTIC.**—Genetic prophylaxis is occasionally possible, as when two persons with definite affective disorders marry each other and are advised not to have any children. Rules of thumb do not apply in this matter; it is wrong to tell a patient he should marry or not marry, procreate or not, unless one has been able to weigh the dubieties of our genetic knowledge, the pedigree of the patient and all his transmissible qualities with an informed and cautious judgment.

Individual prophylaxis is not practicable until after symptoms have appeared which bring the patient to the doctor; social prophylaxis, mental hygiene, and child guidance have not yet been proved to have permanent value in staving off or mitigating affective illness except in the matter of depressive suicide. In so far as one finds environmental factors (*e.g.* heavy responsibility, unemployment, or sexual frustrations) important in inducing an attack, advice on these matters may be helpful; it may be practicable by psychological and social treatment during the healthy interval to do much good in this way. But some cases, in which intrinsic factors seem all powerful, are proof against such measures, and in any case it is not easy to persuade the patient when he is well again to put himself for a long time in the doctor's hands.

TREATMENT OF THE ACTUAL ILLNESS.—It is convenient to consider separately the acute major forms, and the minor more chronic ones.

For the former, treatment is directed to safeguarding life, relieving distress, and providing the best conditions for the emotional disturbance to subside; the situation is like that in tuberculosis or typhoid fever. Exhortations to "pull yourself together" are as out of place as advice to take a

voyage or an argument about the delusions. If the attack is sufficiently severe to unfit the patient for ordinary duties, treatment at home is probably inadvisable. Although in such attacks all argument is futile and active psychotherapy harmful, yet the loss of relation between current experience and emotion is never absolute; there is virtue in separating the patient from real trouble and distressing associations, reassuring him, giving him firm, kind management. The essential combination of these, and especially the last, is rarely obtainable at home. The patients, however boisterous or suicidal, usually recognise their need of treatment and are willing to enter hospital voluntarily. They should not transact any business if it can be helped; their judgment may be too much disturbed, they lay up trouble for themselves. Continuous narcosis sometimes seems to curtail an attack; ephedrin and acetylcholine have also been reported as doing so. The former treatment, *i.e.* narcosis, demands experience and care, no treatment cuts short an attack regularly or dramatically enough to justify any set conclusion about its efficiency in this regard.

Prolonged baths—for 8 or 10 hours daily at a constant temperature of 96° to 98° F.—have much value in allaying restlessness, whether of the manic or the anxious kind, especially the former. They have the further merit of diminishing angry contact with other people, permitting fairly free movement and lessening dirtiness, besides promoting sleep.

Drugs are indispensable. The fear of habit formation should not prevent hypnotics being given when there is persistent insomnia. Barbiturates, phenobarbitone or paraldehyde often suffice: it is well to ring the changes, to prescribe the barbiturate in divided doses, and in each case to diminish the dose without the patient's knowledge. For severe anxiety bromides, opium (*e.g.* as papaveretum) and hyoscine may be helpful; the risks of the two latter are obvious. As to bromides, the risk is intoxication, which makes the patient worse; estimates of the bromide content in the blood and clinical scrutiny should prevent this. Continuous narcosis is valuable, and in some cases convulsant therapy cuts short an attack. Both these methods must be used with caution and reserve, because of the risks, which may be disproportionate to the advantages. Food must be given in adequate quantity and kind. Artificial feeding, preferably by nasal tube, may be necessary because otherwise the patient would die of starvation. The presence of acetone in the urine and a falling weight curve are strong indications that nutrition must be attended to promptly. A good nurse may sometimes, by unusual patience and sense, get over an obstinate refusal to take enough food and drink, but often nothing prevails against it. Apart from hydrotherapy, rest in bed, fresh air, attention to the bowels, and other measures of general hygiene are desirable.

Suicide is of the first importance. Prevention of it can be better ensured by close knowledge of the patient and his day-to-day condition than by mechanical precautions, but if he is bent upon it, these may be unavoidable. It is possible to make them unobtrusive without nullifying them. Certain it is that excessive use of bolts and bars can defeat its own ends, and excessive supervision aggravate a patient's misery, his fears, or his resentment. Two good rules are: (1) to discredit the maxim that those who talk of suicide never commit it, and (2) to remember that most suicides are surprises. Convalescence from melancholia is a risky time.

Occupational therapy is good, as soon as the patient can be got to co-operate ; but it is not rational treatment to pester a melancholic, to encourage the fretful restlessness of the agitated, or to give the manic patient more things to muddle himself with and destroy. Still, it is often surprising to find how soon, under tactful guidance, these patients will enter into ordered activity of a more or less simple sort, and how helpful it can be to them. During the stage of improvement the same is true of recreations and social activities. Patients should not leave hospital till recovery is assured, unless it is obvious that the hospital surroundings and the absence from home and work are an actual cause of their persistent anxiety or dejection.

To revert to the *milder* forms, which tend more to become chronic. Here manipulation of the conditions in which the patient lives at home and at work may be conjoined with psychological treatment, both depending on an appraisal of the causes of his illness. There is nothing distinctive (though much that takes account of the individual patient's needs) in the psychotherapy and social treatment called for (see pp. 1815-1818) ; danger signals must be recognised as they occur. Zeal must give way to the real needs and resources of the patient, which are often not appropriate to a drastic or very lengthy treatment. Simple measures of inquiry, explanation and reassurance, together with small environmental changes, may have much effect. A fixed regime imposed in detail by the doctor is helpful ; this becomes more and more necessary as the affect dwindles in long-standing cases. Hypomania does not usually respond to causal treatment of any kind ; it seems to run a largely autonomous course. Anxiety may yield very satisfactorily to patient psychotherapy.

SCHIZOPHRENIA

Definition.—The forms of illness under this name are so diverse that many efforts have been made to distribute them, so far in vain. What is common to them all is a detachment from the world without, and a breaking up of normal psychological connections within. The personality is not integrated as in normal people ; thinking, emotion, and conduct are discrepant and morbid, yet there is no impairment of formal intelligence such as is found, for example, in organic dementia. The obsolescent name “*dementia præcox*” is not a synonym for schizophrenia, but a reminder of its recent history. At the end of the last century a large number of patients in mental hospitals were found to have begun their illness before they were 30, and to have passed ultimately into a deteriorated state that looked like dementia ; their illness was closely studied, delimited, and called “*dementia præcox*.” When the same clinical picture, however, came to be found in cases that had not such an outcome or onset, the latter criteria were waived in favour of a descriptive analysis of the actual symptoms, and along with this larger conception came the new word “*schizophrenia*,” which betokened a more psychological approach, and a more elastic and generous notion of what might be included. Theories of causation, psychopathology, and clinical boundaries are implicit in any view of what “*schizophrenia*” really is ; consequently, it is still possible for two experts to disagree about what should properly be included under this name, yet over the diagnosis and prognosis of any particular patient they will attain a measure of agreement and

certainly surprising to those who know the condition only from reading or limited experience.

Ætiology.—**INTRINSIC.**—The intrinsic factors are very important. Studies of the incidence in twins and in the members of a family demonstrate a hereditary factor in a majority of cases. If one of a monozygotic pair of twins be schizophrenic, the other is also in 70 per cent. of cases. The frequency of the illness among various relatives of patients indicates that it is not transmitted as a simple dominant, nor indeed as a simple recessive; probably more than one recessive gene is responsible. It has been suggested that people of definitely schizoid personality are heterozygotes for the genes concerned, actual schizophrenia requiring that the genes be present in homozygotic form; this is as yet conjectural, though some investigators find about as many schizoid psychopaths as schizophrenics among the brothers and sisters of schizophrenic patients.

The constitutional features that betoken an innate predisposition to this illness are more of the psychological than the physical kind. The bodily attributes have been said to be an "asthenic" (weedy and lank), "athletic," or "dysplastic" build; but, since these are found in much the same proportion among healthy people as among schizophrenics, there is little to be said for them here. It is, however, certain that "pyknic" build (see p. 1844) is uncommon among schizophrenics. More significant, however, are the features of personality, commonly called "schizoid"; they are to be found in a large number of cases, though not by any means in all. The patient is reported to have shown slight peculiarities from his earliest years; he has been quiet, shy, and solitary, a "model child," given more to day-dreaming or abstract speculation than to ordinary interests and activity; sometimes he has been unduly submissive and sentimentally affectionate, or touchy, suspicious, obstinate, and resentful of advice and control. A single "typical" schizoid personality is a myth. It is, moreover, to be stressed that a "frozen" description of the schizoid varieties of personality does not do justice to the true state of affairs: characteristic deviations from the conventional norm of behaviour can always be understood better if the patient's way of dealing with his circumstances is viewed historically as a biography of individual tendencies and experiences, rather than described as a bundle of traits. By paying heed to the development of faulty as well as healthy habits of response, the psychiatrist can often see the march of events that led up to the patient's illness, and escape too artificial a sundering of inherent tendencies from the external happenings by which these tendencies have been evoked and given shape and substance.

EXTRINSIC.—The illness sometimes breaks out after childbirth or an acute infection. None of the efforts made to inculcate some specific infection have succeeded, nor does intoxication in general seem to play any considerable part in the causation of schizophrenia. The same is true of cerebral trauma. There are, however, many instances of a chronic schizophrenia supervening on an intoxication, and of schizophrenic symptoms, especially of the catatonic sort, appearing in the course of an organic disorder, such as G.P.I. or encephalitis lethargica. In these, the same structural and functional systems must be supposed to have suffered impairment as in the "endogenous" forms of schizophrenia, and it has been particularly urged that in the chronic paranoid conditions that may follow an acute alcoholic psychosis, it is really

a matter of schizophrenia that happens to be associated with alcoholism, if not partly activated or released by it. It is further to be remembered that at least one intoxication, namely, with mescaline, produces a mental disturbance that is in some respects similar to schizophrenia, and that any chronic hallucinosis comes in time to look very like a long-established schizophrenia, because the possibilities for abnormality of any human mind are few, the deprivation symptoms almost uniform, and our methods of clinical examination imperfect. Endocrine disorders, especially of the gonads, have been held responsible, but satisfactory evidence is lacking, except in a few cases which can scarcely be representative.

Recent mental stress may sometimes be the starting-point of an attack, but in a considerable proportion of these cases the reported overwork, disappointment in love, or other painful experience, is found to have been a product of the already existing illness, or the last of a long series of disturbing events. No recent experience is ever sufficient to account for the illness without regard to intrinsic causes. Nor is any remote experience either. No matter how searchingly the patient's life be resurrected and analysed, it is scarcely ever possible to discover that anything happened to him which would have led to his adopting a schizophrenic way of shunning daily life unless he had been somehow disposed to it from the beginning; although, of course, much may have happened to him that has strengthened and fostered the disposition.

Among contributory factors, age and sex are noteworthy. An onset after the age of 40 is uncommon. In three-quarters of the cases that later exhibit the characteristic chronic syndrome, the illness begins between 15 and 25. The condition may become overt in children before puberty. Men are more often affected than women—in the proportion of 113 to 100, according to the largest available statistic, the matter is dubious, however, because of the different standards of diagnosis used.

Pathology.—**PHYSICAL.**—Histological changes in the brain are not characteristic; it is doubtful if they are even frequent. A cellular loss in the third and fifth layers of the cortex, with lipoid accumulation, has been found, but it occurs in many other conditions. Many claims about cerebral pathology, and the chemical and physiological changes in schizophrenia have now been discredited, so that all findings in this difficult field have come to be matters of suspicion. Variations in the same individual may be wide. Recent investigations have purported to show:—a disturbance at the acid-base equilibrium towards the acid side, with a diminished excitability of the respiratory centre to carbon dioxide; lowered rate of oxygen consumption; polyuria; diminished gastro-intestinal motility; poor response to epinephrin; abnormal heat-regulation; decrease or sluggishness of total blood-volume; and slowing of the arm-to-carotid circulation time. These findings have not so far been controverted; they represent disorders of metabolism and regulation which may be partly a concomitant of the characteristic mental disorder, and partly an effect of it, *i.e.* they may be essential physical disturbances in the illness, or may be secondary to the abnormal, often inert life the patients have led since they became ill. There is no ground for supposing them causal.

Some inferences have been drawn from the similarity of catatonia to the extrapyramidal syndrome that can be produced in animals by bulbo-

caprine; the argument from analogy cannot be pushed further than to say that certain functional systems are available in the brain, which are sometimes involved in schizophrenia, as they also may be in poisoning or in encephalitis lethargica, G.P.I. and other diseases.

Very significant are the well-attested metabolic findings in the rather rare cases of cyclical catatonia. In these the nitrogen balance varies periodically; with alternating phases of retention and over-excretion, corresponding to the mental change from excitement to stupor or *vice versa*. By means of thyroxin a thorough emptying of the patient's nitrogen store can be brought about and subsequent nitrogen retention prevented, thus leading to clinical improvement. The correlation between metabolic happenings and clinical condition in these patients is now established.

PSYCHOLOGICAL.—The large and inconclusive literature on the psychopathology of schizophrenia is of five main kinds, namely:

- (1) Minute description of the phenomena observed, and abstraction from them of general principles of disordered function.
- (2) Experimental study, chiefly quantitative (*e.g.* psycho-galvanic).
- (3) Studies of artificial hallucinatory psychoses (*e.g.* mescaline intoxication) and parallel experiences.
- (4) Comparative study of animals, children, poets, primitive people, etc.
- (5) Intuitive or speculative interpretation.

It will be obvious that these methods overlap and that they differ widely in acceptability and usefulness. The findings of almost all can sound plausible, when stated in general terms; discrepant or abstruse, when stated in detail. Their exposition touches on the most intricate problems of normal and morbid psychology, and therefore is highly technical and unsuitable here. A working hypothesis for clinical purposes is that in schizophrenia there are inherent faulty habits of reaction, whose severity and persistence depends largely on education and other external circumstances. These faulty reactions are characterised by a deficiency in the function of synthesis, so that there is an inco-ordination, "intrapyschic ataxia," as it were, a splitting up of the mental life, which justifies the name "Schizophrenia." Thereby the whole psychic life of the patient, cognitive, emotional, and conative, is changed in a way that is alien to normal understanding. We can observe the change but cannot enter into it or describe it adequately in terms of our own experience, as we mostly can depression, manic excitement, hysteria, or obsessions. It shows itself also as a turning away from the contacts and realities of daily life, a preference for what the mind can supply from its own stores, however morbidly, rather than for the current experience that the outer world affords.

Symptoms.—Schizophrenia may be regarded for clinical purposes as a form of maladaptation in which there are certain characteristic defects of inner harmony and consistency in behaviour, thought, and emotion. These are rarely seen in childhood, but from puberty onwards they may appear in varied combinations (often in persons who for years have been introspective and unsocial). There is discrepancy between mood and utterance, disturbance of conduct (briefly summed up as catatonic or hebephrenic), self-absorption and incapacity for sustained thinking along normal lines. A guarded or artificial demeanour may conceal these essential features, whereas they may be conspicuous in a florid or "deteriorated" case. Hallucinations

and delusions may fill out the picture ; affective or other morbid types of reaction may complicate it.

The onset is not always abrupt. There is often a long history of preliminary symptoms in which it is arbitrary to decide where personality has merged into illness. Complaints of headache, weakness, anxiety attacks, loss of appetite, and dysmenorrhœa may have accompanied slight oddities of behaviour, such as rudeness or apparent absence of mind and indecision. The patient may have felt an alarming change in himself, in his capacity to think and feel normally, and been notably depressed and anxious. Ideas of persecution or of exaltation may occasionally escape him, or he may have become stilted in his talk and shown other affectations and mannerisms. The more gradual the onset—and in many cases it has spread over many years—the more unlikely is it that it will have been recognised as morbid.

The commonest or basic symptoms are: (1) Disorder of thinking. (2) Emotional incongruity. (3) Hallucinations. (4) Disturbed impulses or conduct. From these can be derived most of the other symptoms, such as delusions, feelings of influence, autism, catatonic phenomena, anomalies of speech, negativism, and the rest.

The *disorder of thinking* is a characteristic and central feature. The patient cannot command the whole range of an act of consecutive thought ; he misses the point, fastens on details and brings in irrelevant associations which are correct in themselves, but which divert him from the main end of his original process of thought ; consequently his thinking is incoherent, rambling and jumbled. He brings together the most far-fetched topics, so that the connections are sometimes so superficial as to be empty of meaning, and at another time profoundly influenced by symbolism and highly individual values. The usual logical sequences are ignored : cause and effect are interchanged ; temporal, spatial, verbal, and accidental relationships are unduly turned from abstract to concrete, treated as grounds of identity, played with or flouted. Things linked only by analogy and chance association are taken to be the same. The condensation of several conceptions in one, or transference of a set of attributes to some inappropriate object, may become a matter of course, so that only the closest knowledge of the patient and his surroundings will enable the psychiatrist to follow his meaning. It is not necessary, however, that such extreme incoherence be evident in the patient's talk ; he may not show any at all when speaking, or may suddenly obtrude a startling lapse from normal ways of thought which he then ignores, justifies or explains away. Inconsistent thoughts can be present together in a way impossible for normal people ; and the same object or notion can appear to him in several interchangeable guises, each of which would normally exclude the others. The patient himself is often aware of his disordered thinking, and may describe it : he feels his thoughts are suddenly taken out of his mind, other thoughts, foreign to him, are put into his head, his mind is not his own, his thinking is suddenly interrupted, some external power controls it.

The thought-disorder is illustrated by the following characteristic remarks of patients : " There were bats and bees coming through the window ; of course that was because my brother-in-law kept teasing me. He said I had bees in my bonnet." " If I should return during my absence, keep me here until I come back." " I have a lot of forced thoughts. My thoughts

are all drawn-out words, they ought to be pin-pricks. There is an unnatural stoppage in my thoughts, too. . . . I have heard voices say 'He is conscious of his life.' . . . To get my feeling back to normal I feel like changing motor-cars into battleships, to be superior to them."

This disorder may only be demonstrable when the patient gets on the topic of his delusions; in other matters he may seem quite sensible. It is not essentially different from what normal people experience during states of altered consciousness, *e.g.* in dreams, or when falling asleep; the schizophrenic, however, has it with clear consciousness, so that a listener often feels that the patient is making fun of him in giving such transparently absurd answers with an air of knowing exactly what he is about. Some chronic well-preserved schizophrenics make their living as comedians, the audience much enjoying the allusive, half-comprehended nonsense, with its background of innuendo and symbolism. Autism, *i.e.* immersion in his own fantasies and preoccupations, may account for much of the oddity and detachment the patient shows; it accounts also in part for his "negativism," in that he resents any stimulus that interferes with his day-dreams.

Delusions arise mainly out of the thought-disorder. They are often bizarre; they may occur to the patient with a suddenness of conviction that puts them beyond all argument; and they are egocentric in that they commonly bring indifferent happenings or people into a special relationship with the patient—*e.g.* he suddenly knows that when his cousin yesterday said he had been reading about Napoleon's divorce of Josephine, it was a subtle way of telling the patient that his wife was committing adultery with this cousin, whose name is Joseph. The delusional ideas may not be firm conviction, but fleeting notions, readily given up, and based upon some casual instance of the thought-disorder; sometimes they are schizophrenic ways of saying something commonplace—*e.g.* the patient declares his wife has poisoned him, but when he is further questioned says airily that he means she gives him ill-cooked food which is bad for his digestion.

Fixed delusions are, however, common, and are usually of a paranoid complexion; they may develop out of more or less ephemeral ideas of reference. They are often intermixed with hallucinations. The patient gets into a state of mind in which he feels there is meaning in everything, something is going on behind the scenes, he is perplexed by all this, and mystified, it has to do with him in some uncanny way. Presently, he begins to "see through it all," sometimes he gives it some religious or cosmic significance, especially if he has much anxiety as well—the Last Judgment is at hand, he is to be responsible for the regeneration of the whole world. The delusions are not always enacted on so grand a stage; there may be homely fancies about neighbours who whisper and sneer, or about some vulgar bogey like the Jesuits or the Jews or the C.I.D. Often, the patient complains that people work on his mind, hypnotise him, influence him for his own good, set about to drive him mad or ruin him. Delusions of grandeur may be linked up with these paranoid ones (*e.g.* he is being persecuted because he is the Messiah), and may be likewise pedestrian or lofty, according to the patient's previous education and interests, the severity of his disorder, the copiousness of his fancy, and the amount of normal mental function still in evidence. Here, as elsewhere in psychiatry, the symptoms are a mixed outcome of impaired or perverted function on the one hand, and of normal

function on the other, the latter either reacting to and modifying the disorder, or obtaining freer play through it. If, for example, a patient feels his thoughts being controlled by some external influence, and he has queer tinglings in his body, his conviction that he is being hypnotised, and that some one is playing an electrical instrument on to him, must be regarded as a normal attempt to find the cause of an almost inexplicable happening. The delusions are sometimes about past events, which are falsified retrospectively, *e.g.* the patient relates details of his having been a changeling or a predestined hero. Delusions about bodily transformation or disease are frequent, and may be complicated and bizarre.

Patients often do not act in accordance with their delusional beliefs, especially when these are fleeting or chronic; they may, for example, be friendly towards a nurse whom they believe to be persecuting them cruelly. But this is, on the whole, unusual in the early or acute stages of the illness: a patient will then act on his beliefs violently or in terror; he may go to the police or be driven to suicide.

Constantly the matter of a patient's delusions will be found to be intimately dependent on his experiences, his emotional attachments and sufferings, his struggles and frustrations; it is impossible, however, by any such analysis and derivation of his delusions to account for the fact of their occurrence, *i.e.* for the patient's choice of this way of dealing with the experiences in question. The same is true of the general thought-disorder: *e.g.* interruption or "blocking" of the train of thought may take place only when some emotionally weighted topic, some complex, is touched on. This accounts for the place where "blocking" occurs, but not for the "blocking" itself; that, like the other fundamental disturbances of function in schizophrenia, eludes a wholly psychological explanation.

Intellectual defect does not occur. There is usually no clouding of consciousness. Intellectual laziness or evasion is often conspicuous; the patient may repeat questions in a musing way, or profess ignorance. Orientation and memory are not, as a rule, diffusely impaired, though hallucinations, delusions, and lack of interest may interfere with them, and consciousness may be disturbed in stupor or excitement. Many a patient who has long borne the appearance of gross dementia will suddenly show that his intelligence is still a sharp instrument: drugs, *e.g.* sodium amytal or insulin, and intercurrent disease or shock can thus dramatically reveal how little ground there is for calling this illness a dementia. Schizophrenics often do the unexpected. Amnesias, and deliria, when they occur in schizophrenia, may be hysterical; obsessional and hysterical symptoms, like anxiety and depression, are compatible with schizophrenia, and are often an intimate component of the illness.

The speech and writing of the schizophrenic betray the extent of his thought-disorder. Stiffness, pedantry, fantastic euphuisms, words of his own coining, queer symbols and grammar, stereotyped repetition, and infantile twists like speaking of himself always in the third person may be conspicuous features of the patient's use of language. There may, of course, on the other hand, be little or nothing outwardly amiss in his conversation and writings. In florid or chronic cases the patient may talk in an unnatural voice, or without any modulation. Writing may be set forth as though it were painting, and the converse: in subject and matter the patient's insanity

may be patent, but his treatment of his matter, however odd, is seldom odder than some forms of modern art, and it cannot, therefore, be called typical of the illness. These anomalies of symbolical representation are as open to psychological explanation as are the delusions mentioned above; the neologisms, for example, can be analysed up to a point; and these phenomena have enriched our knowledge of the psychopathology of schizophrenia.

The *emotional incongruity* is the chief, but not the only, sign of disturbed affect. Often the patient himself notices in the beginning of his illness that he is less moved by habitual affection, or even feels hatred towards a parent he has loved. The strongest and rarest of human passions are not infrequent in this illness: ecstasy, mystic communion, despair, horror, agony of death, limitless abandon, apotheosis, salvation, are approximate names for these exceptional states that are probably indescribable in the current language of normal people. Apart from these, and much the commonest of the affective changes, is apparent emotional shallowness: the patient receives moving news without any sign of being touched by it, or his response is perfunctory; he smiles or looks bored when talking of a recent tragedy in his own family. This shallowness and incongruity of affect is, however, not to be taken at face value. What the patient says, and what he means with his words, may be very different; so may what we say be very different from the meaning the patient attaches to it. It is unsafe to assume that the patient's words have reference to what is mainly going on in his mind at the moment, or that his outward expression is a trustworthy index of his emotional state. Violent emotional outbursts—of anxiety, rage, love, misery—can certainly occur in a patient who has lately seemed empty of all affect. The schizophrenic patient is undoubtedly different from normal people in his emotions, but not in so negative a way as his seeming apathy and lack of affective rapport would suggest. His attitude towards the same person may change quickly, in accordance with conflicting or opposite tendencies in himself; this ambivalence is often understandable in the light of his earlier history. Sometimes the illness leads to a blunting of ordinary reserve, a lack of reticence, or a levelling down of the gravest matters, so that frivolous or cynical indifference and imperturbability are signs of the patient's morbid condition.

Hallucinations are not so frequent as superficial examination of patients might suggest; many of the patient's assertions about queer sights and sounds are not the expression of vivid perceptions but of passing fantasies, imagined more plastically than is normal; this is particularly true of many of the so-called visual hallucinations, or of cases where the unreal perceptions occur in several senses together. Hallucinations are nevertheless extremely common and persistent in schizophrenia: auditory ones occur most often, diffuse somatic ones not infrequently, those of smell, taste and sight more rarely.

The "voices" are sometimes so closely linked with the thought-disorder that it is difficult to tell whether the patient is relating what he has heard or what he has thought. He may show the intermediate stages between the two, declaring that people repeat his thoughts or that everything that passes through his mind is spoken aloud inside his head; his actions are described publicly, he cannot go to the lavatory without shameless comments. What the voices say may be abuse or encouragement, trivial repetition or threats and commands; this content can usually be accounted for by the psychiatrist,

when he knows the patient and his history well. The voices may come from strange places, *e.g.* from inside the patient's own chest or abdomen, and are then often accompanied by curious somatic hallucinations, indicative of morbid attitudes, both physiogenic and psychogenic, towards parts of the body. The latter often occur independently. Queer sexual feelings, or distortions and impossible growth of various organs, may be reported. They are usually bound up, as any schizophrenic symptom is likely to be, with delusional and emotional components, which are partly derived from the patient's experiences and psychological development. The visual disturbance, like the gustatory, is more often illusional than hallucinatory, *e.g.* people's faces look fiendish or artificial or transfigured.

The *actions and bearing* of the patient are often characteristic. Abruptness or lack of grace in movement may be seen early; it can be indistinguishable from the fidgety self-conscious hobbledelay stage of adolescence. The patient may pull faces at himself in the mirror, or may be unaware of his grimaces. Asymmetrical movements of expression, twitchings, mannerisms, queer rituals and tic-like gestures are to be met with. The meaning of the patient's movements can usually be worked out, but after they have been present for long their sharpness is rubbed off, as it were, and the empty stereotyped movement at last gives little clue to what was once a significant emblem of experience and feeling. The movements often seem to become automatic, like the "verbigeration" of empty phrases in the patient's speech. Negativism, talking and acting beside the point, and bizarre escapades may be seen at any stage of the illness.

There may be a suspension of movement, or the reverse: akinesia or hyperkinesia. Both may occur in the same patient, who may lie for weeks or months in a catatonic stupor, from which he suddenly emerges into swift action. He may carry out some impulsive action and then promptly return to bed and stupor; or he may become wildly excited and imperil his life by his blind and raving activity. During catatonic stupor, patients may adopt strange postures, *e.g.* holding their head off the pillow all day, pursing their lips. They may be indifferent to cleanliness about faces and urine, or actively dirty in this regard. Waxy flexibility is rare, but many patients are automatically obedient so that they keep up an imposed posture.

The variety of schizophrenic anomalies of conduct is too great to be described here. They must not be assessed absolutely, but always in relation to the setting in which they occur. Then they have meaning in the individual case, and are not merely so many examples of "ambivalence," or "mutism," or "negativism." It is, however, true in this matter also that understanding the content of an anomaly does not make its occurrence likewise understandable. Much of the schizophrenic's conduct is so close to certain disorders of movement in organic disease of the central nervous system, that somatic mechanisms may be assumed to have suffered damage in this condition. There are three main things to be done with any schizophrenic symptom: (1) to search out its psychological origins, and its meaning for the patient in his present situation; (2) to link it up with the other functional disorders that he shows; and (3) to consider its background of physical structure and function. It is not always practicable to attempt all three, nor is it as yet possible to do them well, but none can be ignored without detriment to a full analysis.

Often the most significant yet intangible effect of the illness is upon the patient's *personality*. After florid symptoms have died away, or when there are no definite symptoms at all, a change in the patient's ways is remarked by his intimates. Not only is he outwardly different—more “peculiar,” less understandable and predictable, rather shut-in upon himself, remote, with queer values and impulses—but in many cases he is also aware of this change, and may complain of an inner perversion of himself, a loss of that unity which we take for granted when we say “I,” or “me.” Insight in schizophrenia, in this respect and more generally too, may be penetrating and just, as many self-descriptions attest. There may also be varying degrees of impairment up to gross lack of insight.

None of the *bodily symptoms* are characteristic of this illness, though many occur. Besides the somatic complaints and preoccupations already mentioned, patients, especially if young, show vegetative anomalies. Thus, vasomotor disturbance may take the form of cold bluish extremities, exanthems or oedema. Seborrhoea is common. Abnormal growth of hair occasionally occurs in women. Loss of weight in the acute stages, and fatness in the chronic condition, interruption or irregularity of menstruation, and fluctuations of temperature may also be observed, especially in catatonic cases; of the schizophrenic states, stupor is the richest in demonstrable bodily changes. Fleeting neurological signs, *e.g.* pupillary anomalies, may be found. In states of acute excitement attacks of unconsciousness may occur, but epileptic seizures are very rare.

VARIETIES.—There are three main forms—catatonic (with acute outbursts); hebephrenic and simple (early onset, chronic course); paranoid (fairly late onset, delusional). They are not exclusive categories, and it is usually profitless to try and apportion a doubtful case to one or the other. They do not correlate closely enough with outcome to be of much use clinically.

In *hebephrenia*, the least common variety, delusions and hallucinations are inconsiderable, but abnormal conduct is to the fore: the patient may be silly and mischievous, abruptly eccentric or inert and without initiative. The illness may progress without acute episodes (*dementia simplex*), or be interrupted by phases of excitement or obvious insanity, which subside, leaving the patient worse than before. In *catatonia*, the most favourable variety, the symptoms are plain even to the layman: akinetic or hyperkinetic states may appear and subside quickly, sometimes for good or for several years. There are usually, also, characteristic disorders of thought and emotion, which may clear up when the stupor or the excitement does. In the *paranoid* form, generally rather late and insidious in its development, but less damaging to the personality than the hebephrenic, partial systematisation of the delusions is common in the earlier stages, but may be later swallowed up in the general thought disorder and deterioration (*dementia paranoides*). The more bizarre the delusions, the more likely is affective emptiness to replace gradually the initial resentment and distress, but sometimes the patient passes into a chronic paranoid state, obviously schizophrenic to the psychiatrist, but compatible with ordinary life outside an hospital. Hallucinations and luxuriant delusions may, however, be conspicuous in the paranoid form (*paraphrenia* and *dementia phantastica*).

Diagnosis.—The chronic and advanced cases—“typical *dementia præcox*”—that abound in mental hospitals, are easy to diagnose, but

early or inconspicuous cases often extremely difficult. The chief positive points to look for are : characteristic thought-disorder, a qualitative change of affect, and other evidence of " intrapsychic ataxia," as well as feelings of being under external influence. Catatonic symptoms are of limited diagnostic value, because of their frequency in organic and symptomatic psychoses. More important than any single feature is the impression of the case as a whole, the development away from normal interest and response to the real world, and the establishment, instead, of " autistic " self-satisfactions so that the patient's personality is twisted awry, as it were, and withdrawn from easy contacts.

From organic syndromes—syphilis of the central nervous system, alcoholic psychoses, disease of the cerebral vessels, encephalitis lethargica, etc.—the differentiation turns on the physical findings, more than on the mental state : a schizophrenic syndrome may appear in an organic condition, because the brain, as Kraepelin said, is like an organ whose stops give out the same sound, whoever works them. Often it is not a matter of deciding whether the syndrome is organic or schizophrenic, but whether, being schizophrenic, it has a discoverable somatic basis or not. Alcoholic delusional states are an instance of the complicated relationship that may be found (see p. 1838). If, after consciousness has become clear again, the other phenomena of toxic confusional psychosis persist, then schizophrenia is the more probable diagnosis.

Diagnosis of schizophrenia from an affective syndrome is difficult, because both are often combined in the same patient. Some of the significant points have already been referred to (see p. 1859). Catatonic excitement differs from mania in that the speech and acts of the latter are intelligible as expressing a general affect and are conformable to the situation in some measure ; the onset and cessation are not so abrupt as in catatonic excitement ; and there are usually characteristic features which make the distinction easy. Melancholia becomes suspect when delusions are repeated without the appropriate affect, and there is a readiness to project responsibility for the illness, to complain of external influence. The inertia of the depressive is not so complete as that of catatonic stupor, nor so likely to be abruptly broken through. States of severe agitation are not always easy to distinguish from schizophrenic excitement, but a more frequent problem is that of deciding whether some bodily fear or conviction of disease is schizophrenic or not. Whether in regard to a preoccupation or a delusion, the chief point to consider is the appropriateness of the affect to the alleged hypochondriacal notion ; the more bizarre the bodily change described, the more likely to be schizophrenic. Depersonalisation is sometimes at the bottom of these somatic complaints ; what is significant is not the depersonalisation, but the way it is elaborated and regarded by the patient.

Hysteria can offer great difficulties, largely because hysterical mechanisms are so often operative in schizophrenia. Plain motor or sensory disturbances commonly give less trouble than hysterical dissociation, stupor, and pseudo-dementia. The previous history, the relationship of the outburst to a particular set of happenings, the behaviour in the intervals, the demands upon the attention or response of bystanders must be taken into account. The mistakes and oddities of the hysterical pseudo-dement may be theatrical, in accordance with his ignorant notion of what insanity is like ; the deliriously dissociated

hysteric does not identify correctly the people around him, as the schizophrenic usually does, even when in a dream-like state; the hysteric who is acting some imagined scene does so without discrepancies or gross interpolations, whereas the schizophrenic is seldom so consecutive and persistent. The degree to which the patient is being influenced by his immediate surroundings is, however, the chief guide, apart from definite schizophrenic features.

Obsessional states offer difficulty when the patient is in doubt as to whether his alien thought or impulse comes from within his own mind or is imposed upon him. If he shows indifference as to the occurrence and content of the compulsive ideas, it is suggestive of schizophrenia; but careful examination of the development of the symptom, and the patient's attitude towards it, permits a clear diagnosis in most cases. Complicated rituals, odd obsessions and chronicity make an obsessional illness look very like schizophrenia; as does intoxication of an obsessional patient by bromides. Obsessions may develop into schizophrenic symptoms (see p. 1888).

Prognosis.—Schizophrenia is always a serious condition. Though some recover, the tendency of this morbid change is to do permanent damage to psychic life. In the individual case, however, pessimism is not justified. It is certainly never possible in the early stages of the illness to be certain that recovery is out of the question.

Heredity is a poor guide to the prognosis, except in the rare cases in which an identical twin of the patient has for some years had a schizophrenic illness, or in which one parent is schizophrenic, and the other has schizophrenic relatives; even then it is difficult to prognosticate with assurance regarding the present attack. If one parent has had an affective illness the prospects of recovery are brighter, but this can better be assessed from the patient's own bodily and mental constitution. If he is of pyknic build, the outlook is much better. Similarly, the patient who has for years tended more and more to withdraw from his surroundings, to be careless of social requirements, to lie late and live alone, given up to day-dreaming and eccentricity—such a one should he become overtly schizophrenic, has a poorer chance of doing well than the active, suspicious and impulsive man, or the self-conscious, introspective worrier who similarly falls ill. A narrow and rigid previous personality makes deterioration more likely than if there had been wide interests and possibilities of adaptation.

The more abrupt and stormy the onset, the better the outlook. This is one of the most reliable guides. When the onset has followed upon a recent painful experience, and the content of the patient's talk and his behaviour refer to this, or when a physical damage appears to have provoked the symptoms (*e.g.* influenza or head-injury), the outlook is rather better than when the provoking factors are obscure; but this is by no means always the case. If the attack occurs during puberty or adolescence, prognosis must be cautious, because of the difficulty of distinguishing between the transient upsets of this period of adjustment, and the progressive schizophrenia that may then show itself plainly. The earlier history is of great help.

The nature of the symptoms is not a safe guide. Very severe departures from normality may clear up, yet an outwardly mild condition be of grave omen. Symptoms such as stereotypies of movement and speech, which indicate that the illness has been going on a long time, and that there is a

general narrowing and fixity, are grave; as are also hebephrenia, and a long-drawn-out stupor, with negativism, impulsive violence and vasomotor changes. The more manic or depressive features, the better. Previous attacks, with an interval of normality between them, are prognostically favourable. If the patient first falls ill after 30, he will scarcely go downhill in the tragic way young people sometimes do. He may develop fixed delusions, which are often rigid and encapsulated, so to speak, and therefore he may be able to return to ordinary life, with reservations; or it may be that his morbid beliefs absorb all his mental powers, and compel institutional life. The more the psychiatrist can discover healthy modes of response in the illness itself, as well as in the previous personality, the happier the outlook. Many patients, after an attack, do not return to work, but have narrower interests, and less spontaneity than before; they are more easily tired, and may be hypochondriac, or show other symptoms thought to be "neurotic." Such patients have sometimes made a poorer recovery than others who return to work and can meet most social demands, though careful inquiry reveals definitely schizophrenic sequelæ in their thinking and emotions.

The simplest rule is that an abrupt onset of the illness, an adequate cause for its occurrence, and a well-adapted non-schizoid personality are the criteria of good prognosis. Sensible early treatment may avert disaster.

Treatment.—**PROPHYLACTIC.**—This, whether eugenic or individual, is limited and uncertain. Even if effective, it can reach only a minority at present, and its effectiveness is a matter of faith. Probably child guidance and other measures of mental hygiene do good in averting potential schizophrenia, but no one can be sure of this. Such treatment aims at diverting the child into social activities and keeping him out of situations in which he will be mortified or otherwise troubled emotionally. However wordily or abstrusely the prophylactic treatment be described, it is essentially a matter of trying to make an unusual child into an average one, or making his surroundings unusual to suit him.

TREATMENT OF THE ACTUAL ILLNESS.—There is no one treatment of the disorder that has manifest superiority over any other. Painstaking attempts at readjustment of the patient's outlook and behaviour by means of psychotherapy (not psychoanalysis), occupation, games, etc., are the most systematic and rational way of making a permanent change for the better. The co-operation of the patient is here necessary, and also the help of a social worker desirable, who may do much to modify and arrange the patient's circumstances in the interests of his mental health, *e.g.* getting him suitable occupation, and schooling his relatives in a sensible attitude towards him. Such treatment is not practicable for those acutely ill, but for the mild, the convalescent, or the imperfectly recovered case it is of great value. By means of it many patients can be discharged from hospital before they have settled into apathy, or become unresponsive to the claims of the external world; it is better not to keep a schizophrenic patient in hospital waiting for complete recovery, but to get him back into ordinary life as soon as possible, provided conditions there are not too adverse for him, or he too abnormal to cope with them.

Treatment in a psychiatric clinic or mental hospital is usually necessary at some stage of the disorder, and must be decided chiefly by the severity

and social risks at the time. For the large number who become permanently in need of institutional care, much of the deterioration formerly customary may be averted by the energetic use of occupational therapy and recreation which make the patient's life less sterile.

There are few conceivable ways of altering a human being that have not been tried in this illness. Many of them have been those believed to be efficacious in other illnesses; some have been intended to shock the patient somehow. Of the former may be mentioned endocrine preparations (in large doses), transplantation of gonads, removal of supposed septic foci, induction of fever by malaria, etc., injection of human serum, manganese salts, production of aseptic meningitis and continuous narcosis. Of the latter, *i.e.* shock-methods, many of the procedures of a bygone time are examples; the whirling chair, precipitation from a height, immersion in ice-cold water, and so forth. The most recent methods which entail a profound and alarming disturbance are those which use insulin or a convulsant (see p. 1819). Though the direct emotional effects of such treatment are not negligible, metabolic changes are no doubt chiefly responsible for the clinical effects seen. Insulin has more value than the convulsant method. It should be employed only in hospitals fully equipped and staffed for the purpose. The treatment is most effective in cases which would have a good prognosis for the attack if treated by other methods. It is not a panacea for schizophrenia, but may shorten an attack in suitably chosen cases.

The details of treatment, whether in hospital or at home, must be individual; even in such matters as the allaying of excitement no uniform procedure, *e.g.* continuous baths, or narcosis, can be a routine measure. When excitement is extreme, disturbances in water metabolism and loss of salts may be combated by giving 5 per cent. saline intravenously, 300 c.c. every other day, alternating with forced fluids. During stupor, general measures for ensuring adequate food (in some cases feeding by tube), cleanliness and evacuation of urine and faeces must always receive attention. It has been found that various chemical agents, such as carbon dioxide inhaled in a 30 per cent. mixture with oxygen, and sodium amytal, will temporarily interrupt a catatonic stupor; this finding accords with the chemical metabolic changes reported in the condition, but its therapeutic value is slight.

PARANOIA AND ALLIED STATES

The words "paranoia" and "paranoid" are used loosely by many. Kraepelin gave paranoia its modern meaning, describing it as the endogenous, insidious development of a permanent and unshakable delusional system, with complete preservation of clarity and order in thought, will and action. If the illness cleared up, if it showed symptoms of an organic, affective or schizophrenic syndrome, or if it was provoked by external happenings, it could not be paranoia. Thus delimited, the condition is exceptionally rare; so rare, indeed, that there is no use in having such a category. Moreover, cases that Kraepelin himself called paranoia have since become obviously schizophrenic. There is now no profit in thinking of paranoia, or paranoid states either, as syndromes in their own right, so to speak, and of the same order as schizophrenia or affective disorders. They are on the

same subsidiary level as stupor, hypochondriasis, anxiety and depersonalisation. When met with, they must be distributed according to the accompanying symptoms and the general trend of the illness; and their prognosis and treatment must be assessed accordingly.

Besides the paranoid beliefs and attitude referred to in previous sections, there are a number of instances of this unhealthy relationship between the patient and his surroundings, which are mild in their outward form, easily understandable in the light of the patient's history, and fairly responsive to treatment. Sensitive and shy people are often troubled by doubts and shame as to their physical or moral worth; and, by projection, attribute to others the dislike or contempt they do not acknowledge in themselves. This occurs in youths who masturbate, and suppose others to remark it, and in old maids who believe men to be pursuing them; but there are many varieties of shame and desire, besides the sexual, which lead to such ideas of reference or persecution. The development of paranoid reactions of this sort is usually plain. So is that of the querulous, resentful type of reaction, *e.g.* in the man who believes himself done out of his rights and who becomes a persistent litigant or writer of memorials. Before judging such a man psychopathic, the extent of the injustice he has suffered must be compared with the degree of his resentment and his relevant conduct. Commonly the injustice is found to be fanciful or trifling, and the man's sense of grievance immoderate, so that he comes to believe there is a veritable conspiracy to wrong him, and devotes most of his time to useless appeals or threats. He may persuade his wife or his children of the justice of his complaints, inducing delusions in them, *i.e.* *folie à deux*, etc. Many such patients, however, never become deluded: they are contentious about their wrongs, and waste years, perhaps, in proclaiming them or seeking redress, but they are well aware how other people regard them, and what has actually happened. Many claimants of compensation, "grouzers," "old soldiers" and unstable adherents of more or less cranky movements, are to be placed here. There is no sharp dividing-line between these psychopathic people, and the more or less normal, often socially precious, leaven who detest injustice and are willing to do much to defeat it. Some deaf people become paranoid, misinterpreting what they cannot hear plainly, and construing it into a jeer or an insult.

HYSTERIA

In hysteria, symptoms of illness are represented by the patient for the sake of some advantage, without his being fully conscious of this motive. The form of representation will vary widely according to the circumstances that have provoked the illness, the patient's experience of what the symptoms are that he is trying to represent, and his somatic resources. These factors, presently to be discussed, bear on the hysterical symptoms that simulate physical disease. But it is impossible to restrict hysteria to this physical form. The illness that is represented by the hysteric may be a mental one; moreover, it is not possible to consider hysteria without regard to the mechanisms of its occurrence which manifest themselves in the personality and are mainly psychological. Hysteria is the most psychogenic of all illnesses. Its recognition is therefore a double problem: (1) exclusion of

what may be called "genuine" illness, *i.e.* of a recognised morbid pattern; and (2) discovery of an adequate motivation. To ignore either of these requirements is to court error, since hysteria may occur along with physical or mental disorder, elaborating upon it and mimicking it, and, on the other hand, some physical diseases give rise to symptoms indistinguishable in their form and apparent psychological mechanism from those of hysteria.

Ætiology.—A hereditary factor is probable in many cases. Thus, a group of hysterics who were pathological liars were compared with the average population in respect of the proportion of their brothers and sisters who were in mental hospitals: it was five times as many; and of the parents of the group, a sixth were psychopathic. From these and similar figures it is not possible to tell the mode of transmission or the nature of what is transmitted, but only to infer a hereditary factor. The occurrence of hysterical mechanisms in children, and their frequency in healthy adults, especially after calamities or in unendurable conditions, such as may occur in war, suggest, however, that hysteria is potentially present in most people and that environment is more important here than heredity. The combination of heredity and environment may result, long before actual illness occurs, in a *hysterical personality*. This is not found in all patients who show hysterical symptoms, but nearly all people of hysterical personality show hysterical symptoms. Many of the features of this personality are socially obnoxious, but other features are not, and it is wrong to use "hysterical" as a depreciatory epithet for a set of qualities that one dislikes. These people are unduly responsive to the situation they are in, especially if by their excessive response they can fulfil wishes of which they are hardly aware, or evade what is painful in the situation, instead of meeting it and disposing of it adequately. Unsatisfied with their own capacities, they seek to cut a better figure than their endowment warrants, and are constantly posing and pretending. This, like all their behaviour and aims here described, is not done with full consciousness, but with a more or less sincere ignorance or ambiguity of purpose; it is not a question of deliberate deceit, of studied histrionics or malingering. In thus responding to situations and turning the response to some inadequate end, the hysterical person is characterised by a lack of inner stability and of constant standards of behaviour, and also by a lability of affect and an exuberant fancy. The fantasies normal in childhood are here seen in physically mature adults, who, like children, can temporarily live their fantasies, absorbed in this unreal compound of past experiences and longings, yet not so wholly divorced from their real surroundings as might appear. In an attenuated form, this is evident when they almost unwittingly manufacture some situation, according to their needs—literally "making a scene"—and enter into it emotionally with a rapidity and fervour impossible for more stable people. Egotism and untruthfulness (*pseudologia phantastica*) may be pushed to the point of delinquency. There may be a longing for prestige, sympathy, love, or some other emotional relationship, which leads the hysteric to behave in a way strikingly out of keeping with his demeanour on other occasions; the inappropriateness of his behaviour even at the time may be obvious to a detached onlooker, but is not always so. Many of these people can use illness or well-acted fantasies of illness to satisfy their hardly conscious needs; they may also gain their ends by forgetting what it would be painful

to remember. Here again the onlooker may find it hard to tell how genuine or complete is this forgetfulness, but the question is of little moment compared with discovery of the motive for the hypomnesia. The much-stressed suggestibility of hysterics is a notable aspect of their responsiveness to situations and of their especial responsiveness to a person with whom they develop an emotional relationship, often unrecognised by themselves as such. The emotional attitude of a hysteric towards other people is often influenced by sexual factors. Hysterical personality is believed to be much commoner in women than in men, and may be associated with psychosexual immaturity. Coquetry and frigidity are not uncommonly allied in hysterics; there may be much flirting and sexual excitation, but not actual coitus; it is, however, juster to say that the sexual lives of hysterics show instability and inadequacy than to specify any particular aberration.

Hysterical personality can be recognised before puberty; in younger children, however, it must be extreme to be recognised, because of the great frequency of hysterical mechanisms then (*e.g.* behaving as though fantasies were real, counterfeiting illness, somnambulism). Some of the grossest instances of hysterical behaviour have been recorded in girls not yet adolescent, *cf.* the Salem witches. Much of the work of Child Guidance Clinics is taken up with the treatment of hysterical tendencies, not perhaps taking the form of definite symptoms but plainly evident in the child's personality.

The precipitating factor for the onset of hysterical symptoms is usually a situation, emotionally charged, out of which the patient's symptoms will bring him more or less overt, but unacknowledged, gain. This gain need not be material and obvious, and may run directly counter to such accepted values as health and ability to work. One of the plainest instances of a partial unsubstantial gain is that created by an accident, and the resulting claim for compensation; hysterical symptoms flourish in such a soil, and are usually influenced for the worse by repeated medical examinations. Hysteria occurs among soldiers under active service conditions, and can readily be fostered in them by injudicious measures. It is not infrequently a sequel of the acute panic or stupor which may in some men be the effect of intensive bombardment and exhaustion during active warfare.

Pathology.—This is almost wholly a matter of psychopathology. It is true that disseminated sclerosis and many other organic diseases of the brain may be accompanied by hysterical symptoms, but the association is not a constant one. The psychological changes can usually be traced further back than the happening that provoked the illness; often they are the continuation of normal tendencies of childhood that have been fostered and extended by ill-judged upbringing. The hysterical symptoms that appear as motor or sensory phenomena show the patient's readiness for the translation of experience into bodily symbols; this is a special instance of the universal tendency for somatic representation of experience, converting it into action. It is the facility and exaggeration, not the existence, of this "conversion" mechanism that is characteristic of hysteria. What is thus translated or "converted" into physical terms has been something painful and unacceptable; the partial exclusion of it from consciousness, "repression" of it, is therefore understandable; in its physical, symbolic form it is tolerable and may even be prized. Identification with other people is responsible for the frequent imitation of symptoms and for the epidemics of

hysteria. Suggestibility, with its characteristically quick formation of habits of somatic response, is another way of describing the phenomenon. Clearly the mechanism need not be limited to the production of physical symptoms, though bodily structure and local weaknesses may conduce to this. There can be hysterical phenomena, such as the dissociation seen in fugues and so-called splitting of personality, which are instances of the exclusion of recent and remote painful experience from clear consciousness. The wishes and fears that deviously attain outward expression as hysterical symptoms do not derive solely from the recent past, though much of their strength may come from it. It must be admitted that there are some hysterics in whom this psychopathology cannot be demonstrated, and that such cases are among the most intractable.

Symptoms.—These may be divided into: (1) sensory; (2) motor including fits; and (3) quasi-psychotic.

The symptoms can be like those of any conceivable affection of which the patient has a notion. The cruder his notion, the less will his symptoms be like those of the simulated condition, but after he has been demonstrated to a class or repeatedly examined he may better his notion, and consequently his symptoms come closer to those of organic disease. Or, if he has had opportunity of seeing insanity, his pseudo-insanity may smack less of the stage than it otherwise usually does. The range of hysterical symptoms is so great that to describe them all in detail would take inordinate space, and there is no need to do so.

The *sensory* or, more properly, the *perceptual* symptoms include clonus and globus hystericus, blindness, deafness, and anæsthesia. The two former are so common in all sorts of mental disorder, especially those accompanied by anxiety, that they are of little specific importance in hysteria; inquiry as to their presence will often in these suggestible, rather hypochondriacal patients lead to their occurrence. The difficulty in swallowing reported by hysterical women may be associated with a strong disinclination to eat—*anorexia nervosa*; it should not be confused with depressive *anorexia* or that of pituitary *cachexia*. Any cutaneous disturbance of sensation that the patient has a notion of can be presented, *e.g.*, anæsthesia, either mono- or bi-lateral, or of stocking and glove distribution, and analgesia of any part. The anæsthesia seldom corresponds to any nerve trunk, nerve root, or spinal segment, unless the patient has had special opportunities of knowing. With an anæsthetic hand objects may be identified, and any test which the patient does not recognise as referring to this disability he will perform satisfactorily. Such tests are not a means of “catching the patient out” as though he were a malingerer, but of ascertaining whether the symptoms express only his notion of some illness. The tests for a malingerer, it is true, amount to the same thing, though one assumes the malingerer to be clearly conscious of his purpose; consequently any distinction between hysteria and malingering must depend on the observer's impression as to the patient's honesty and self-knowledge; certainly it cannot be decided by tests. The tests for blindness (*e.g.* using a stereoscope with a supposedly blind eye), deafness (*e.g.* effect on pulse, respiration and psychogalvanic reflex of exciting remarks addressed to the patient), and for other forms of perceptual defect all depend on the physician's greater knowledge of what should or should not accompany the symptoms of which the patient complains; they are not intended to dis-

cover hysterical "stigmata" or characteristic anomalies. The ovarian and other hyperæsthetic spots, the pharyngeal anæsthesia and the concentric limitation of the field of vision formerly used diagnostically, were all products of suggestion or, as in the last instance, phenomena that may occur in normal fatigue, in hypochondria and in certain cerebral lesions.

The *motor* symptoms are paralyses, pareses, spasms, contractures, and tremors. Hysterical paralysis or paresis never affects individual muscles, but always movements. By various devices it can be shown that the patient can still use the affected muscles, as long as he does not know that the movement in question requires their use. The paralyses affect chiefly the left side of the body, are common in the legs (preventing proper walking or standing), and often occur in limbs or other structures that have earlier been the seat of an organic disability, *e.g.* trauma or paresis. If the paralysis be flaccid, no loss of tone or of reflex response is found, and the patient, through his ill-informed notions of what should happen, behaves otherwise than a patient with organic paralysis would—*e.g.* if asked to rise from the supine to the sitting posture without using his hands, he keeps his paralysed leg flat on the bed. If the paretic part be kept stiff, the antagonists will be found to come into action first when the patient is asked to perform the movement he says he cannot; and if the movement has to be made against resistance, sudden removal of the resistance reveals how much of the apparently tremendous effort was going into associated irrelevant or antagonistic movements. Passive movement to overcome the spasticity or subsequent contractures cause the patient to be more upset than could be accounted for by any pain he may complain of. The varieties of abnormal gait are numerous; many of them fantastically elaborate and, from the look of them, exhausting. Not only the musculature of the limbs may be affected but of the trunk (leading to curvatures and odd postures) and indeed any voluntary muscles, *e.g.* of the tongue, larynx, pharynx or eye. In hysterical aphonia the voice may sink to a whisper, or there may, more rarely, be complete mutism; the voice can, however, be used normally for coughing and similar purposes. The aphonia often comes on after some local inflammation that has caused hoarseness, or after a fright. Stammering, usually of the exaggerated kind, may also occur. Spasm of the external ocular muscles, leading to a convergent squint, may accompany a spasm of accommodation. Ptosis and blepharospasm sometimes occur. Many of the tics and spasms that used to be thought hysterical are now recognised to be often physiogenic, *e.g.* residual symptoms of encephalitis lethargica and chorea; spasmodic torticollis, for instance, is far less often psychogenic than used to be thought. When a spasm or paresis has long been maintained, trophic disturbance may follow: blueness and cedema, shiny skin, fibrosis of periarticular structures, and similar effects of rigidity and disuse. Tremor is most often seen in patients with a spastic paralysis, but may occur independently, as in many of the war cases. It is variable in degree and rhythm, and usually disappears when the patient's attention is turned from it; this does not apply, however, to some very long-standing cases.

Hysterical *fits* commonly occur in patients with obviously hysterical personality. They may be little more than a fainting-attack or an outburst of temper, significantly like the tantrums of an ill-behaved child. Often, however, they are more differentiated than this, and diagnosis from an epilep-

tic fit may be difficult. Sometimes the fit grows out of a tremor induced by fright or anxiety, as in many war-hysterics, or it may express some emotional state, such as great pain, anger or erotic excitement. Occasionally the patient shows plainly by her expression and movements that the fit is erotic; it may be a typical orgasm. The "classical" four-phase fits which Charcot described were artefacts of the clinic; they do not happen now.

Sometimes the patient's fit becomes very like an epileptic one after he has spent some time at a neurological clinic, or he may be an epileptic who also has hysterical fits. Some hysterics, by overbreathing, induce an epileptiform convulsion, which can be abruptly terminated if an injection of calcium chloride or gluconate be given. They may pass from one such fit into another, so that the condition suggests a status epilepticus. The unconsciousness that often appears to accompany a hysterical fit is seldom as complete as it looks; neither is the subsequent amnesia. There may, however, be a delirium, corresponding to the emotional upheaval. Patients very rarely hurt themselves seriously in the fit, however violent, or have a fit when alone or asleep. The length of the attack and its degree often depend on the audience; the more the bystanders try to restrain the movements, the wilder do the kicking, struggling, biting, shouting, panting, spitting, etc., become and the longer they go on. There is neither the pallor nor the cyanosis, the regular sequence nor the subsequent headache and sleepiness of epilepsy; urine is not passed nor the bowels opened; reflexes, including the corneal response, are unaffected, and the end of the fit may be abrupt.

The *quasi-psychotic* symptoms are stupor, twilight-state, pseudo-dementia, and fugue. In the stupor, seen typically in harassed weary soldiers under bombardment, the patient lies motionless, taking food like a twelve-months baby, non-resistive, sometimes incontinent of urine or faeces, and without any predominant emotional tone. It is of brief duration if the exciting circumstances cease to prevail. In less acute forms there may be a sullen resistive akinesia, or a condition lasting even for years, with an occasional break; this is a rare form. The confusional or delirious states may accompany a fit or represent an important emotional experience, *e.g.* some sexual episode. They are often histrionic, and represent wishes of a religious or grandiose sort; or the patient may behave as though he were an animal or a child. Sometimes they occur during the night, and in a somnambulist state the patient repeats some past happening, or may do complicated work. This is closely akin to the hysterical fugue, in which there is not so much a clouding as a narrowing of consciousness, a "dissociation." In the fugue the patient says he has forgotten some or all of his life before a certain date, and later he may profess to remember nothing of what has happened during the fugue. There is, in short, a double set of memories, which may alternate, and since the patient's own identity is commonly included in the repressed and temporarily forgotten material, he may be said to have two personalities, and sometimes three or four. Actually there are no cases in which it is strictly correct to speak thus of multiple personalities; it is only a matter of different aspects or fragments of the one personality. In the fugue the patient may live out some fantasy or—as more commonly happens—simply says that he does not know who he is or where he lives. Nearly always a hysterical fugue with gross amnesia turns out to have been a means of evading some predicament, and it is well to keep in mind in such cases that the patient may have

broken the law or otherwise exposed himself to disgrace and punishment. The amnesia is seldom as complete as the patient states. Fugues may occur as a hysterical mechanism in an organic psychosis ; we have seen a man with arrested general paralysis who had been prominently and in detail reported as a case of multiple personality which responded to psychotherapy.

" Pseudo-dementia " covers the large group who behave as though insane. It may occur, as in the so-called Ganser syndrome, in prisoners awaiting trial. Whatever the circumstances, its motive is escape from a disagreeable situation. It is likely, however, that it is mainly those with a predisposition towards severe mental illness, especially schizophrenia, and the high-grade defectives who have recourse to this kind of hysterical behaviour. It sometimes comes on after brain injury. The patients' behaviour corresponds necessarily to their notion of insanity, which is usually far enough removed from anything the psychiatrist knows as such. Occasionally, however, it is very near the buffoon-like conduct of some schizophrenics. The patients say that they do not know their own age, affect not to understand simple remarks, give absurd answers which nevertheless indicate that they know the right answer (*e.g.* by inverting the correct order of the figures in a sum). When asked about some simple matter, they look as though they were making terrific efforts to remember (herein behaving differently from the schizophrenic). The most characteristic thing is the disparity between the patient's alleged deficiencies and his general alertness : he says he does not know anything about his own past, he cannot read or spell or do the simplest arithmetic, and yet he may be behaving quite naturally and adapting himself to the situation in a way which would be inconceivable if he had actually so advanced a dementia.

Some hysterics go to great lengths in their representation of ideas of illness. They will allow themselves to be put among grossly insane people, or submit to repeated operations, such as amputation. Self-inflicted injuries, *e.g.* keeping wounds and sores open, are not uncommon (*cf.* *dermatitis artefacta*). In some such cases masochistic tendencies can be recognised, but by no means in all. Suicidal attempts are not infrequent. They often have as their purpose revenge, the satisfaction of some spite, and the patient may leave behind a lying, fantasy-coloured letter, indicting someone. Frequently the suicidal attempt is in the nature of a theatrical demonstration, done in such circumstances as make it unlikely to be fatal ; and if the patient kill herself, it is more through bad management than intention.

Diagnosis.—It will be plain from what has been said that diagnosis must be both negative and positive—negative, by excluding any organic cause for the symptoms ; positive, by finding motives and relating the symptoms to them. Neither method is alone sufficient, because of the occasional concurrence of structural disease with psychogenic symptoms. As to the former, *i.e.* the negative method, it is unnecessary to enter here into all the differentiating points. Many of them have been mentioned in the foregoing description of symptoms, and all turn on the disparity between what experience tells us would occur if these symptoms were of organic origin, and what the patient knows about such matters. Consequently a doctor who has hysterical symptoms is extraordinarily difficult to diagnose, in this negative sense. The method of arriving at a diagnosis by suddenly taking the patient unawares, and seeing if his symptoms persist, is to be deprecated ; it antagonises him. Likewise undesirable is the procedure of see-

ing whether one can suggest new symptoms to the patient, *e.g.* an *anæsthesia* ; it can be both misleading and harmful. Neither is the hysterical nature of a symptom to be judged solely by whether it can be removed by suggestion ; for some organic symptoms are temporarily got rid of thereby, and many hysterical symptoms are not. An intimate knowledge of the range of symptoms of physical disease is much more useful to the physician than an equipment with special tests and lists of differences between "functional" and "organic." It is not only a problem of neurology but of the whole of medicine, since the hypochondriacal tendencies of many hysterics lead them to complain of visceral symptoms ; usually, in doubtful cases, the symptoms are those which might well occur in the earlier stages of some physical disease. It is, however, in neurology that the most difficult cases of all arise, *e.g.* in disseminated sclerosis, carbon monoxide poisoning, cerebral vascular disease or encephalitis lethargica ; here there is more likelihood of the organic disease being overlooked than of its being wrongly diagnosed. The patient's previous personality, any provocative situation or emotional disturbance, the previous occurrence of organic signs, *e.g.* transient diplopia, and the age of the patient must be considered. Hysterical symptoms appearing for the first time in middle or later life in someone whose personality has been stable, are probably not solely psychogenic. If the symptoms diminish when little or no attention is paid to them, they are more likely to be hysterical.

Course and Prognosis.—This depends mainly on the patient's personality and social setting, and on the treatment employed. A long history of hysterical traits prior to the illness, a continuance of circumstances favourable to the symptoms, and inadequate or excessive treatment are all unfavourable. This is, however, an illness that sometimes confounds prediction, patients recovering when many adverse factors have been operative and the symptoms have been present for years. In children the prognosis is fairly good if treatment can be undertaken promptly ; it is best if the hysteria is monosymptomatic and has come on after a fright. In all cases in which the situation which provoked the illness persists, the outlook is bad ; for example, in the compensation cases, for which no medical treatment is of any avail—for obvious reasons—until the litigation is settled once for all. Similarly, during war, psychotherapeutic successes are often dazzling while the hysterical soldier is under treatment in hospital, but the symptoms come again when he must return to duty. There are many varieties of outcome, chronic invalidism being the commonest. A few patients later become schizophrenic, and a few become involuntional melancholics. The prognosis in respect of the patient's hysterical personality is more important than that of his hysterical illness ; it is, however, no more to be assessed by rules than the general future of any human being's life and personality. Patients do not necessarily tend to become anti-social ; delinquency is certainly a likelihood in some hysterical people, but bravery and self-devotion may be conspicuous in others.

Treatment.—Too much treatment is worse than too little. Injudicious physical or psychological treatment of hysterics often makes their symptoms worse and their illness intractable. Recondite methods should be eschewed by all but experts. Common sense is as important as psychological understanding ; and social usefulness more to be aimed at than removal of symptoms or attainment of self-knowledge. In short, it is not the hysterical illness or

the mechanism of repression and conversion that calls for remedy, but the patient's inadequate way of dealing with difficult situations. Consequently, the whole treatment must aim at the patient's return to ordinary conditions of life as soon as possible, and at a re-education of his ways of meeting difficulties. To this end it is profitable to go over with the patient the situations, emotional disturbances and motives that led up to the illness, and to do this without implying moral judgment or social indifference—certainly without teaching the patient one's psychological theories. It is a matter of general psychotherapy (see p. 1816); and it may entail a far-reaching analysis of the patient's past life, her emotional development and her instinctual tendencies. It is questionable, however, whether anyone without special psychiatric experience is wise to enter lightly upon this way of benefiting the patient. For, on the one hand, he may be misled into a wilderness of fantasy masquerading as once-repressed, now-recalled psychic trauma; and, on the other, he may be at a loss how to deal with the attachment and dependence upon him which the patient will come to show, and which may in fact be the chief influence in bringing about her precarious recovery. A great deal may be achieved—perhaps as much as by more thoroughgoing methods—if the physician, himself mature and with impartial insight into the psychological motivation of the symptoms, leaves aside in his dealings with the patient any very detailed inquiry into the causes of the illness and the purposes it served; and, instead, directs her towards a better social adaptation, by advising her to avoid when possible the situations that, as he sees, favour the production of symptoms, getting her into a disciplined way of living, and stepping in with explanation, support and advice whenever fresh difficulties arise. His success in getting rid of individual symptoms at the beginning may be an important factor in establishing the necessary relationship with the patient. Such a line of treatment is not heroic, it is scarcely even rational, in the sense of being causal, but it avoids some of the commonest blunders and may be strikingly successful. For this, or indeed any treatment, admission to hospital is not essential; but it will help when there are adverse factors in the patient's situation and, of course, will be essential if there be such symptoms as self-injury, suicidal attempts, pseudo-dementia, or gross paralysis. The danger of the patient's picking up new symptoms in hospital should also be weighed. Isolation is usually inadvisable.

Many of the symptoms of hysteria will not wait upon general treatment, but demand energetic intervention. Anorexia, for instance, cannot be allowed to go on to an avoidable inanition, nor a paralysis to the stage of contracture; a mute patient, or one who is deaf or blind or ignorant of his own identity, offers such practical obstacles to almost any kind of treatment that the symptoms must be tackled and disposed of early. For this purpose suggestive measures are valuable and appropriate physical treatment may be called for, e.g. supervision during feeding, or even tube-feeding in anorexia nervosa, physiotherapy for paralysis, voice-exercises. Suggestive measures need not take the form of hypnosis; suggestion in the normal waking state has many advantages over hypnosis, though those expert in the latter are sometimes very successful in their treatment. Suggestion, like almost every form of treatment of hysteria, has pitfalls, and its triumphs, like those of every other method, sometimes prove vain, but in the hands of a physician who is at once confident and cautious, this method may result in a satisfactory

recovery. If, in using suggestion, such physical devices as faradisation can be avoided, it is better to do so. As a means of demonstrating that the illness is not due to local disease, however, such methods sometimes take their place in a detailed plan of treatment. Motor and sensory symptoms can usually be got rid of in one or two sittings if the physician is patient, determined and confident in the use of persuasion and suggestion.

The choice of occupation, the settlement of any social cause of illness (e.g. claims for compensation), and the obtaining of a healthy attitude—neither complaisant, much-enduring nor harsh—on the part of the patient's relatives and friends, are all important factors in treatment. The hysterical reactions to injury call for special mention because of their frequency. Though often of transparent motivation, they are not by any means to be regarded as outright malingering; for the patient's feeling of illness may be sincere, his symptoms distressing, his anxiety typical, and his irritability and insomnia symptoms that he would gladly get rid of. But they are none the less psychogenic. It is often assumed that so far as an illness is psychogenic, it must be treated only by psychotherapy. This is false theory. There are few mental disorders in which psychotherapy alone produces such small benefit as in the hysterical conditions due to the compensation or pension situation that may follow an injury. Putting an end to the situation early and the resumption of ordinary activity as soon as any physical injury has been repaired are the most potent measures in the earlier stages. Even if the symptoms have been present a long time, the ending of disputes about claims and the return to ordered routine and regular occupation achieve more than do frequent medical interviews. Psychotherapy is then an adjunct, not an essential feature, of the treatment. Marriage should never be recommended as treatment for hysteria; the superstition about this has resulted in lamentable troubles, especially for the person the hysteric marries. This is not to say that every hysteric is to be dissuaded from marrying; there are more things than treatment to be considered then. Married hysterics, however, should not be recommended to have a child. Contrary to popular notions, pregnancy and puerperium more often aggravate than benefit hysteria. Moreover, hysterical women are not usually satisfactory parents, and commonly induce psychopathy in their children.

ANXIETY STATE

As already stated, the emotional syndrome so called is part of the group of affective disorder, in which depression and manic excitement are also included. It is there described. It would be indefensible to put into a special category all the forms of mental illness in which anxiety is conspicuous, for it can be severe in the most diverse conditions, ranging from delirium tremens to schizophrenia. The outwardly mild form, tending to chronicity and often largely psychogenic, responds well in the less advanced stages to psychotherapy; it is therefore important that its recognition should not be delayed because of a doubt as to physical disease. Yet often the correct diagnosis is overlooked while the patient is being investigated or treated for some local disorder. This arises partly because of the quasi-physical signs of fear which he may show—dizziness, tremor, nausea and

vomiting, indigestion, diarrhoea, shortness of breath, palpitation, a sense of oppression in the chest, rapid pulse, flushing, sweating, frequent passage of urine, etc. It is still more due to the patient's anxiety turning on his health, especially his physical health, and leading him to ask for more and more medical opinions, X-rays, laboratory investigations, etc., the favourable results of which, however, do not allay his worry. Over-cautious advice as to regime, based on a possibility that there may be some early physical disease, can be harmful to the patient's mental health in that it restricts his normal life, and may constantly recall and reinforce his anxiety. The converse error of mistaking some early symptoms of physical illness for hypochondriacal anxiety is equally to be avoided. Physical investigation of doubtful cases is, in short, indispensable, and should be prompt as well as thorough. When it fails to confirm the presence of a physical disorder the patient should not be treated as though he will still be in danger of the physical illness unless he takes special precautions in diet, exercise, etc. This is well illustrated by such a condition as effort-syndrome, where care taken to avoid any damage to the heart intensifies the illness. The patient should be fully investigated on the psychological side and treated accordingly ; this does not mean that he should be treated only by psychotherapy. The discovery of a possible psychological cause for the symptoms does not prove that there is not also a physical cause for them, but it makes it less likely. The converse is also true. For ætiology, diagnosis, prognosis and treatment see section on Affective Disorders.

OBSESSIONAL DISORDER

Definition.—In this condition the characteristic feature is that, along with some mental happening, there is an experience of subjective compulsion and of resistance to it. Commonly the mental happening (which may be a fear, an impulse, or a preoccupation) is recognised, on quiet reflexion, as senseless ; nevertheless it persists.

Ætiology.—**INTRINSIC.**—The hereditary factor is strong. A third of the parents of obsessional patients, and a fifth of their brothers and sisters, have themselves shown pronounced obsessional traits ; the proportion is in each case higher if all forms of mental abnormality be included, since both schizophrenia and affective illnesses occur with more than average frequency in families of obsessionals. The abnormal personality of the parents is probably also potent as an environmental cause. Very many obsessional patients have for years before they became ill shown a rather characteristic mental constitution : they are excessively cleanly, orderly and conscientious, sticklers for precision ; they have inconclusive ways of thinking and acting ; they are given to needless repetition. Those who have shown such traits since childhood are often morose, obstinate, irritable people ; others are vacillating, uncertain of themselves, and submissive. " Obsessional " traits occur, however, in many people who never become mentally ill, and in many who become mentally ill otherwise than with an obsessional disorder. Consequently these traits cannot be rigidly held to be the forerunners or non-morbid counterpart of obsessional illness.

EXTRINSIC.—The influence of strict, morose, cruel, overconscientious, or obsessional parents has just been mentioned. It is difficult to weigh its

importance ; certainly in some cases it plays no part. There is nothing specific in the situations which supply the content of an obsession : they might equally well have preceded hysterical symptoms, for example, in a person so predisposed. Nevertheless, the fright or pain which once accompanied a particular experience, or a long series of experiences, must not be overlooked in working out the multiple causes of some obsessions psychologically related to this experience.

Encephalitis lethargica and a few other cerebral diseases may produce typical obsessional symptoms in persons previously free from demonstrable tendencies in this direction.

Pathology.—Apart from the difficult instances in which lesions of the brain are accompanied by obsessions, this is at present wholly a matter of psychopathology. Some elements of an obsession are universal human attributes : all little children tend to ritualise and repeat ; all human beings are at times uncertain of the rightness or sense of what they have done ; they try to avert trouble by symbolical acts and other magical devices, whose effectiveness they may question (*e.g.* superstition) ; many normal people, moreover, have mild obsessions that do not bother them (*e.g.* scruples). The manifest struggle going on in the obsessional patient may be restated in terms of hypothetical instinctual tendencies. Such attempted explanation cannot be verified ; and it is more useful to pay heed to the repression, displacement and substitution which lead to symbolic representation of emotionally significant earlier experiences, and to the protective mechanisms by which the patient tries to ward off the painful and overwhelming obsession, with the result that he develops complicated rituals and similar devices which may be mistaken for the essential symptom. The transition from obsessional to schizophrenic is easy to understand psychopathologically, since in both some contents of consciousness are separated from the main stream.

Symptoms.—Obsessions are conveniently classified as : 1. ideas or images ; 2. impulses ; 3. phobias ; and 4. rumination. These overlap constantly.

Among obsessional *ideas and images* are tunes, phrases, mental pictures of a disagreeable sort (*e.g.* of a mutilated corpse), and obscene associations (*e.g.* every cranny reminds the patient of a vulva). Obsessional *impulses* are often of a suicidal or aggressive character : the patient may feel an urge to kick people in the street, to push his friend over a cliff, or to throw himself under a passing train. In many other cases, however, they are less alarming ; *e.g.* impulses to swear loudly in church, or to laugh at a funeral ; or more of an intellectual sort, such as an impulse to count and manipulate numbers senselessly or to avoid typing any word with a given number of letters or beginning with a particular consonant. *Phobias* are closely bound up with the other varieties of obsession : thus, the patient who has an impulse to plunge a knife into his friend's or his own neck has an understandable phobia of knives ; the patient who is troubled by obscene thoughts whenever he looks at a naked statue develops a phobia of museums. Not all phobias can be so accounted for ; they may rest on some forgotten alarm, and take a queer form, such as a phobia of lavatories or of one-legged men. It is loose usage to give the name "phobia" to every case in which an individual develops fear that is excessive or inexplicable ; the essential features of an obsession, already mentioned, should also be present. Fears of dirt or infection are very common phobias : they are symbols of moral, usually sexual, taint, and they

lead to much washing, etc.; thus, a patient who has blamed himself for masturbation may be constantly washing his hands, or following a complicated ritual of touching nothing with his bare hands for fear of contamination. Often the rituals and defensive precautions seem grotesque when compared with their ostensible purpose, as in the case of a patient who is perpetually putting himself to the greatest trouble in order to ensure that he never steps on a worm inadvertently; much of the grotesqueness disappears when it is discovered what the worm symbolises for him. Ludicrous as his behaviour may seem, it is often tragic in the distress, and indeed ruin, it may cause him. Another phobia is that which has fear as its object, *i.e.* the patient is afraid of any situation in which he may feel fear; some such patients do not leave their homes for years, because they fear they may have an attack of agoraphobia once they get outside. Obsessional *rumination* usually takes the form of endless questioning or search. The patient has to ask himself "Why" with pointless insistence about all manner of problems beyond his or anybody's grasp; or he has to keep casting round in his mind after some forgotten name or word which he could easily do without. Religious scruples sometimes fall into this category, as when a penitent is continually running to his confessor with some venial trifle he has come upon in his interminable self-questioning and doubt.

Obsessional patients are in most cases depressed; their illness is a depressing one. Besides this secondary depression, however, there is frequently an association of a more intimate kind, in which depression—or mania—is the essential or the main part of the illness, and the concurrent obsessions seem to be symptoms of this affective disorder. In such cases the obsessional illness is very often cyclical in its course. Anxiety is a common accompaniment of obsessions; in phobias it is most conspicuous. The anxiety is inseparable from the patient's struggle against the subjective compulsion which is so alarming to his feeling of integrity in self and mind, such a shock to his belief that he is a free agent. Schizophrenic symptoms may be in the offing, or actually present, when the obsessional ideas are of the magical kind, *e.g.* the patient feeling that the effect of his obscene thoughts upon others may be averted by some gesture, or when his rituals are carried to bizarre lengths, *e.g.* having to save the last drops of his urine because of some recurring doubt. Depersonalisation may occur in the course of an obsessional illness.

Diagnosis.—If the essential features, *i.e.* feeling of subjective compulsion and immediate resistance to this, be kept in view, it is seldom difficult to distinguish between obsessions, on the one hand, and delusions, hallucinations, ideas of reference or self-reproach, feelings of being influenced and schizophrenic stereotypies, etc., on the other. The only difference between obsessions and many schizophrenic phenomena towards which the patient retains insight and which he regards as alien to him, lies in the nature of the compulsion he experiences: in obsessions it is subjective—he feels that it comes from within his own mind, whereas in the schizophrenic phenomena he feels that it comes from without, it is imposed upon him. It is a difference, however, that may be obliterated, *i.e.* what was once obsessional may become schizophrenic, but this is an uncommon outcome when the obsessional disorder is definite and well-established. In differential diagnosis it must be remembered that obsessions may occur in the course of almost

any mental illness in a person of obsessional tendencies, and that the psychological mechanism for the production of obsessions, like that for hysterical symptoms, is present in almost everybody in varying degree. Consequently, an illness is not to be regarded as obsessional unless obsessions are the chief symptoms.

Course and Prognosis.—The outlook for recovery is worse if obsessional symptoms have been present since childhood, if they now fill up most of the patient's time, and if he is weakly resigned to his illness. The best outlook is when the obsessional illness comes on suddenly in a person who has not had conspicuous obsessional traits or who has had previous benign attacks. A cyclical course is not uncommon. The situation is ominous when the ritual gets more and more systematised and remote from what previously occasioned it. The development along schizophrenic lines, already mentioned, is more to be feared in such cases and in those with bizarre obsessional thoughts; the great majority of gross obsessionals, however, do not become schizophrenic or anything else than obsessional. About half the cases recover from an attack, which may, however, last for a year or even more. Many people are subject to brief attacks, lasting only a few days, and largely due to fatigue or physical illness reducing their mental health. Inter-current happenings influence the course of the illness, *e.g.* some men were free from symptoms during their period of war-service, with its routine and lack of responsibility or need for decision. The content of the obsessions is of little use prognostically. Old age is not in itself an adverse factor, but attacks in childhood suggest a strong constitutional bias and are therefore unfavourable on the whole. Few obsessionals give way to anti-social impulses, *e.g.* to suicide, homicide, delinquency. It is true that obsessionals who are also depressed may kill themselves, and that obsessionals who are irritable and angry may injure others; but obsessionals rarely yield directly to an impulse they have resisted, or need to have "irresistible impulse" urged in extenuation of a crime. Sexual offences and perversions are rarely obsessional.

Treatment.—Patients should be encouraged to continue at their occupation and not to test themselves, or try to overcome their obsession, by repeatedly putting themselves in a situation in which it will occur. So long as their impulses are not likely to get them into trouble, they should be encouraged to give way to them, rather than to "fight." The physician must aim at getting a patient well by putting an end to his anxiety and struggle; if that is not wholly attainable, the patient must be educated to deal with his obsessional tendencies by acknowledging their existence, their psychological origins, and their harmlessness in those very respects in which he thought them most harmful, *e.g.* obscenity. Frank recognition of obsessional tendencies, which everyone has in some degree, is an important step in learning to control them. In some patients the obsessional attack is so cyclical and almost self-limited that a brief rest and general care are all that is needed. In others, whose affection is chronic, recovery is out of the question, but advice about the management of their lives, varying according to their individual circumstances, helps them greatly. These patients, so prone to rumination and endless questioning, often clamour to be psycho-analysed. There is no evidence that psycho-analysis, however prolonged, benefits them more than methods that are not so exigent of time and money.

Obsessional children usually respond well to changes in their human environment, advised after the physician has inquired into the family and school situation; temporary separation from an obsessional parent or treatment of the latter often proves remarkably beneficial for the child. Discussion of his problems with the child (especially if they centre round secret sexual play) is an important adjunct of such treatment, just as it would be with an obsessional adult.

EDWARD MAPOTHER.
AUBREY LEWIS.

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